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Gentlemen:

At the suggestion of Dr. Joel Buxbaum, I am sending you the enclosed material. Some other material will follow.

Sincerely yours,

Janet Y. Sonnenthal

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Administrative Coordinator

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Conference on Lead Poisoning in Children
Rockefeller University, May 25-26, 1969

Synopsis of Medical Aspects of Childhood Lead Poisoning

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Childhood lead poisoning is a preventable disease. An effective program of prevention requires two essential ingredients: A clear recognition of the etiologic factors and a clear commitment of responsibility by physician, public health worker, city official and aroused public to take concerted and effective action.

The etiologic pattern which can ultimately lead to 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 (be they emotional, intellectual, informational and/or economic) to cope with her family's needs. The place is a neglected slum housing unit with flaking lead pigment paint within reach of a small child's grasp. The dynamic interaction of these factors will be discussed, with the main emphasis on the environmental factors. In the young child, symptomatic acute lead intoxication is primarily a summertime disease: In the United States, approximately 80 to 85% of all cases occur between May and October. The disease is almost exclusively limited to pre-school children who live in houses built prior to 1940, many of which are still in use and still contain layers of lead pigment paint which have never been removed. The principle sources of lead within such houses are the painted windowsills and door frames. Recent surveys in Baltimore indicate that 50 to 70% of old houses in selected slum areas still contain dangerous quantities of flaking lead paint. A few small chips of such paint may contain 100 mgm or more of lead. (The safe daily intake of lead is less than 0.5 mgm.) The repetitive ingestion of a few small chips of such paint, if permitted to continue for more than three months, can lead to the absorption of a potentially lethal body burden of lead.

Clinical manifestations tend to vary with both the age of the child and the magnitude of abnormal lead ingestion. Acute encephalopathy is more 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 behavior disturbance. Associated iron deficiency anemia is virtually always present. The onset of acute encephalopathy tends to

be fulminant and unpredictable, especially during the summer months. Unexplained vomiting and decreased interest in play are especially ominous premonitory signs which call for thorough and prompt diagnostic evaluation. As the pre-school child grows older, acute toxic episodes tend to be less severe. Thus, the two to five-year-old child with unrecognized plumbism may present with a convulsive disorder simulating idiopathic epilepsy, chronic impulsive, aggressive hyperkinetic behavior disorder or mental retardation. Uncommon syndromes include progressive loss of mental function simulating degenerative cerebral diseases and peripheral neuropathy. Since there are no abnormalities specific for lead intoxication on physical examination and since routine examination of blood and urine is likely to be unrevealing, prompt diagnosis depends upon a high index of suspicion on the part of the physician and the performance of certain specific laboratory tests. The most valuable of these is blood lead determination. For rapid presumptive diagnosis in the symptomatic child, the qualitative urinary coproporphyrin test is most valuable and should be available in the emergency rooms of all hospitals serving high risk populations.

Prior to the advent of chelating agents, the mortality from severe acute encephalopathy was approximately 66%. With the advent of first BAL and later EDTA, this mortality was reduced to approximately 30%. It has recently been reported that the use of BAL and EDTA in combination, together with careful supportive therapy, can apparently reduce the mortality from acute lead encephalopathy to less than 5%. Nevertheless, the incidence of severe permanent brain damage among survivors of encephalopathy continues to be 25% or more. If survivors of an initial attack of acute lead encephalopathy are reexposed to abnormal lead exposure, the incidence of severe permanent brain damage is increased to virtually 100%. For this reason, the cornerstone of our current therapeutic program is prompt termination of environmental exposure to lead: No child with an increased body burden of lead is ever returned to a leaded home. In practical terms, this usually means brief hospitalization for treatment with BAL and EDTA followed by the administration of d-penicillamine to the child in a convalescent home: during this time, suitable new housing is found for the family or the abnormal lead sources are removed from the current home. In addition, upon return home, every effort is made to enroll the child in a nursery school or day care center to provide the stimulation which he needs: such stimulation usually terminates the child's pica. If the incidence of permanent injury to brain and possibly to kidney is to be substantially reduced, increased efforts must be directed toward early diagnosis prior to the onset of symptoms and ultimately to primary prevention of the disease.

Currently, there is wide-spread interest in the application of mass screening techniques to the prevention of childhood plumbism. Three techniques which are comparable in cost are currently available and under investigation. These include the determination of δ-aminolevulinic acid in urine. The major problem in the application of this test is the difficulty in obtaining random urine samples promptly from young children 12 to 24 months of age, who are the patients at greatest risk. Currently, the suitability of the determination of lead in hair is under intensive investigation. The technique recommends itself because of the ease of obtaining adequate samples of hair. Determination of lead in blood is the most reliable. A venous blood sample is required. More sensitive methods for the determination of lead in blood, preferably upon capillary samples of blood, would greatly facilitate this problem. Not only children, but also houses must be screened. Simple techniques are available for this purpose. The cost of such procedures is small when compared with the cost of a single case of lead encephalopathy, which at the least will entail hospitalization for four to six weeks, not to mention the cost of long-range follow-up care, special educational facilities and, in the unfortunate few, permanent institutionalization.

How much more intelligent it would be to spend our effort and substance on the systematic elimination of environmental lead exposure associated with old dwellings. Were this to be done, childhood lead poisoning could be largely eradicated in the United States, as it apparently has been in Australia. For example, 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.