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AUTHOR Steinfeld, Jesse L.
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ABSTRACT

This document is a policy statement written by the Bureau of Community Environmental Management and approved by the Surgeon General of the U.S. Public Health Service. Its purpose is to assist in the development and implementation of programs for the control of lead poisoning in children. Information included covers the medical aspects of detection, treatment and follow-up. Suggestions are given for the removal of lead hazards and for the reporting of lead adsorption. A listing of the addresses and telephone numbers of the 10 regional representatives of the Bureau of Community Environmental Management is provided for obtaining consultation and guidance in developing community programs. (AJ)

THE SURGEON GENERAL'S

POLICY STATEMENT

on

MEDICAL ASPECTS OF CHILDHOOD LEAD POISONING

Jesse L. Steinfeld, M.D., Surgeon General of the U.S. Public Health Service has approved the attached Policy Statement and has designated the Bureau of Community Environmental Management as the activity within the Department of Health, Education, and Welfare to assist in the development and implementation of programs for the control of lead poisoning in children.

August 1971

DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service

MEDICAL ASPECTS OF CHILDHOOD LEAD POISONING

The U.S. Public Health Service recommends that screening programs for the prevention and treatment of lead poisoning (plumbism) in children include all those who are 1 to 6 years of age and living in old, poorly maintained houses. Children exposed to other special local conditions involving lead hazards also should be screened.

Lead-based paint was commonly used for interior purposes until the 1940's when it was largely replaced by titanium-based paint; therefore, children living in dilapidated or obviously deteriorating houses built prior to that time are to be given particular attention. Children who frequently visit such neighborhoods--homes of baby sitters, relatives, and playmates--also should be included in screening programs.[1] Today lead-based paint is still used to some extent for the exteriors of dwellings, and this potential source of exposure to lead should not be overlooked. Children at risk should be screened periodically during the years 1 to 6, and longer if indicated.

The prime goal of screening programs is the prevention of lead poisoning. The prevention of plumbism can be achieved through the early detection of children with undue absorption of lead, followed immediately by remedial action before the state of overt poisoning is reached. Consequently, screening programs should not be limited

to the detection and treatment of children with lead poisoning. To be effective, such programs must also include adequate plans for medical follow-up of those children screened and found to have high levels of lead absorption, as well as those diagnosed as having lead poisoning. For all of these children, the program must provide for adequate and speedy removal of lead hazards from their homes.

In children, the determination of blood lead, even with its pitfalls, is generally considered the most reliable of the many biological tests or indices of lead exposure and absorption. The U.S. Public Health Service, therefore, recommends that blood lead determinations be used in screening children for the detection of lead poisoning and excessive absorption of lead.

Various studies have reported that the median concentration of blood lead in both adults and children in the urban population--without undue exposure to lead--ranges from 16 to 27 micrograms per 100 milliliters of whole blood.[2,3,4,5,6,7] The normal range of blood lead is stated to be 15 to 40 micrograms per 100 milliliters of whole blood.[3,4,8]

Until future studies indicate otherwise, it is recommended that a blood lead concentration of 40 micrograms or more per 100 milliliters of whole blood (as validated by the dithizone technique), determined on two separate occasions, be considered evidence suggestive of undue absorption of lead, either past or present. It is essential that the current degree of exposure be determined in children who present such evidence. Since 90 percent or more of the measured lead in blood is

attached to the red blood cells, seemingly low blood levels in children with anemia may be misleading.

In some cities, undue exposure to and absorption of lead among children may be so prevalent that an overwhelming number of those screened are found to have blood lead values of 40 micrograms or more per 100 milliliters of whole blood. Under such circumstances, local resources may not permit immediate evaluation of all such children, and a schedule of priorities will have to be adopted. Programs with inadequate facilities or inadequate financial support may, in their initial phase of operation, give priority to children with blood lead values of 50 micrograms or more per 100 milliliters of whole blood. Among children with blood lead values of 40 to 49 micrograms per 100 milliliters of whole blood, the 1- to 3-year-olds should be given priority. This age group comprises approximately 85 percent of the reported cases of plumbism, and it also has the highest mortality rate from this disease.

This schedule of priorities is permissible only in the initial phase of operation of programs with limited resources. All programs that adopt such a schedule should plan to expand their operation systematically so that 2 years after the programs have come into existence all children with undue absorption of lead or with lead poisoning will be given adequate care.

CHILDREN WITH BLOOD LEAD VALUES OF 80 MICROGRAMS OR MORE PER 100 MILLILITERS OF WHOLE BLOOD

The U.S. Public Health Service recommends that all children found to have a blood lead concentration of 80 micrograms or more per 100 milliliters of whole blood, regardless of the presence or absence of clinical symptoms or of other laboratory findings, be considered as unequivocal cases of lead poisoning and that they be handled as medical emergencies. They should be hospitalized immediately for chelation therapy. This emphatic recommendation is made because the risk of acute lead encephalopathy in this group is great, the onset of the disease is unpredictable, and its course is fulminant. If encephalopathy develops, at least 40 percent of these children will sustain severe and permanent brain damage.[9] Treatment prior to the onset of encephalopathy may improve this grim prognosis.

CHILDREN WITH BLOOD LEAD VALUES OF 50 TO 79 MICROGRAMS PER 100 MILLILITERS OF WHOLE BLOOD

All children who in screening programs are found to have blood lead values of 50 to 79 micrograms per 100 milliliters of whole blood should be referred immediately for evaluation as possible cases of lead poisoning. Physicians in charge of such evaluation have the responsibility for making a diagnosis of lead poisoning in these children. Symptoms of lead poisoning--such as abdominal pain, anorexia, constipation, and those of central-nervous-system (CNS)

origin--are frequently absent in this group of patients. If any of these symptoms are present and cannot be explained otherwise, the diagnosis of lead poisoning should be considered.

In the absence of clinical symptoms, the following tests are helpful in suggesting a diagnosis of lead poisoning. The U.S. Public Health Service recommends that those whose blood lead values are in the range of 50 to 79 micrograms per 100 milliliters of whole blood on two successive tests be considered suggestive cases of lead poisoning if they have any of the following conditions:

1. Urinary excretion in 24 hours of more than 1.0 micrograms of lead per milligram of Ca-EDTA administered intramuscularly at a dose of 50 milligrams per kilogram of body weight--the total dose not to exceed 1 gram of Ca-EDTA;[10, 11]
2. Serum delta-aminolevulinic acid (ALA) level of greater than 20 micrograms per 100 milliliters of whole blood using the Haeger-Aronson method;[12,13]
3. Urinary output of coproporphyrin greater than 150 micrograms per 24 hours;[14]
4. Urinary output of delta-aminolevulinic acid greater than 5 milligrams per 24 hours;[15]
5. The presence of basophilic stippling of red blood cells, "lead lines" in long bone x-rays, or a strongly positive

urine spot test for coproporphyrin[15] may be considered indicative of lead poisoning when laboratory facilities for making the tests designated above are not available.

It is emphasized that only positive findings are significant; negative findings do not rule out the possibility of lead poisoning. Knowledge and technology related to lead and its effects on human beings is rapidly advancing and the future is sure to hold more accurate and simpler biological indices of increased lead exposure and toxicity. The U.S. Public Health Service stands ready to modify the above recommendations when future research so indicates.

Children whose blood lead values fall in the range of 50 to 79 micrograms per 100 milliliters of whole blood and who are not diagnosed as suffering from lead poisoning should be closely followed and supervised. Determination of blood lead values at monthly intervals at the very least is recommended, particularly in the summer. Sources of hazardous lead exposure should be identified and promptly brought to the attention of the appropriate local government agency for corrective action.

CHILDREN WITH BLOOD LEAD VALUES OF 40 TO 49 MICROGRAMS PER 100 MILLILITERS OF WHOLE BLOOD

Where resources permit, all children who in screening programs are found to have blood lead values of 40 to 49 micrograms per 100 milliliters of whole blood should be recalled immediately for evaluation. This evaluation should include another determination of

blood lead and inquiry concerning pica and the child's current exposure to lead in his home and in the homes that he frequently visits. Exposure may be significant even in the absence of a history of pica since parents may be unaware of such ingestion, or unwilling to admit it. X-ray of the abdomen is useful in confirming current or recent ingestion of lead.

Children whose blood lead values in repeated tests fall in the range of 40 to 49 micrograms per 100 milliliters of whole blood, and who--according to the evidence of their histories--are no longer exposed to lead hazards, do not need to be followed for continued evaluation. For example, those who live in new housing projects, and who do not frequently visit homes with lead hazards, are presumed to be no longer exposed.

Those who continue to be exposed to lead hazards in their homes, or elsewhere, should be closely followed, with determination of the level of blood lead at least every 6 to 8 weeks. Closer supervision in the summer months is advisable, particularly among children under 3 years of age. Where possible, determination of blood lead should be made at 4-week intervals in the summer. Dwellings identified in screening programs as potential sources of lead hazards should be brought promptly to the attention of the local government agency responsible for enforcing housing codes and regulations so that proper corrective action may be taken.

TREATMENT AND FOLLOW UP OF CHILDREN WITH LEAD POISONING

All children who are diagnosed as having lead poisoning should be:

1. Treated immediately;
2. Removed from the source of lead exposure at home or in any other environment--until proper corrective action has been taken to eliminate the hazards;
3. Carefully followed until 6 years of age or longer, if indicated, in order to prevent repeated lead exposure and poisoning;
4. Given adequate neurological and psychological assessment at the time of diagnosis and in ensuing years to detect at an early stage any neurological or behavioral deviation, including minimal brain damage, so that proper therapy and school placement can be instituted; and
5. Given additional clinical and laboratory evaluation, when indicated, to assess other sequelae of lead poisoning, such as renal, myocardial, and metabolic disorders.

The treatment and follow-up recommended can best be accomplished through development of centers designed specifically for the treatment and evaluation of children with lead poisoning, and integrated into planned or existing community facilities for comprehensive care. Careful medical follow-up throughout the preschool years is necessary because of the ubiquity of lead paint in old, deteriorating housing and the persistence of pica in many young children once the habit has been established.

REMOVAL OF LEAD HAZARDS FROM THE CHILDREN'S ENVIRONMENT

Sources of lead must be removed from the environment of children who have lead poisoning or who have absorbed hazardous amounts of the poison into their blood. Immediate follow-up of these children should be initiated with the appropriate local government agencies--with reports on their dwelling units and on other suspect environments or sources of lead hazards--so that proper corrective action may be taken. In fact, effective medical care of children with plumbism is almost totally dependent upon prompt and thorough environmental hygiene to prevent a continuing buildup of lead in their bodies.

REPORTING LEAD POISONING AND LEAD ABSORPTION

1. Lead poisoning should be considered a disease that must be immediately reported to the local health department, when suspected or discovered.

2. All laboratories performing blood lead determinations should be required to report to the local health department the results of tests of blood samples having 40 micrograms or more per 100 milliliters of whole blood.

3. A uniform reporting form should be used to record information collected in screening programs in order to provide comparable data and meaningful statistics on the problem of excessive lead absorption and lead poisoning in children.

Approved by:

Jesse L. Steinfeld, M.D.
Surgeon General
Public Health Service

For consultation and guidance in developing community programs to control childhood lead poisoning, contact the Bureau of Community Environmental Management representative in the appropriate regional office:

Region I Conn., Maine, Mass., N.H., R.I., Vermont	BCEM Representative Public Health Service, DHEW J.F. Kennedy Federal Bldg. Boston, Mass. 02203	(617) 223-6668
Region II N.Y., N.J., Puerto Rico, Virgin Islands	BCEM Representative Public Health Service, DHEW Federal Building 26 Federal Plaza New York, N. Y. 10007	(212) 264-2522 2528
Region III D.C., Delaware, Md., Penn., Va., W. Va.	BCEM Representative Public Health Service, DHEW 401 N. Broad Street P.O. Box 12900 Philadelphia, Pa. 19108	(215) 597-9133
Region IV Ala., Fla., Ga., Ky., Miss., S.C., N.C., Tenn.	BCEM Representative Public Health Service, DHEW 50 Seventh Street, N.E., Room 404 Atlanta, Ga. 30323	(404) 526-5031
Region V Ill., Ind., Mich., Minn., Ohio, Wisconsin	BCEM Representative Public Health Service, DHEW 433 West Van Buren Street, Room 810 Chicago, Ill. 60607	(312) 353-5278
Region VI Ark., La., N.Mex., Okla., Texas	BCEM Representative Public Health Service, DHEW 1114 Commerce Street, Room 1607 Dallas, Texas 75202	(214) 749-2261
Region VII Iowa, Kansas, Missouri, Nebraska	BCEM Representative Public Health Service, DHEW 601 E. 12th Street, Room 528 Kansas City, Mo. 64106	(816) 374-5491
Region VIII Colo., Mont., N.D., S.D., Utah, Wyoming	BCEM Representative Public Health Service, DHEW 19th & Stout Streets, Room 9024 Denver, Colorado 80202	(303) 837-3641
Region IX Am. Samoa, Ariz., Calif., Hawaii, Guam, Nev., Trust Ter. of Pacific I., Wake I.	BCEM Representative Public Health Service, DHEW 50 Fulton Street San Francisco, California 94102	(415) 556-3687
Region X Alaska, Idaho, Oregon, Washington	BCEM Representative Public Health Service, DHEW Second Avenue Seattle, Washington 98101	(206) 442-0378

REFERENCES

1. Lin-Fu, J.S. Lead poisoning in children. Public Health Service Publication No. 2108, 1970. 25 pp.

2. Blanksma, L.A., H.K. Sachs, E.F. Murray, and M.J. O'Connell. Incidence of high blood lead levels in Chicago children. Pediatrics, 44:No 5, Part I, 661-667, November 1969.

3. Chisolm, J. Chronic lead intoxication in children. Develop. Med. Child, Neurol., 7:529-536, October 1965.

4. Goldwater, L.J., and A.W. Hoover. An international study of "normal" levels of lead in blood and urine. Arch. Environ. Health, 15:60-63. July 1967.

5. Moncrieff, A.A., O.P. Koumides, B.E. Clayton, A.D. Pattrick, A.G.C. Renwick, and G.E. Roberts. Lead poisoning in children. Arch. Dis. Child., 39:1-13, February 1964.

6. Robinson, M.J., F.E. Karpinski, and H. Brieger. The concentration of lead in plasma whole blood and erythrocytes of infants and children. Pediatrics, 21:793-796, May 1958.

7. U.S. Public Health Service. Survey of lead in atmosphere of three urban communities. Publication No. 999-AP-12. Raleigh, N.C., U.S. Dept. Health, Education, and Welfare, National Air Pollution Control Administration, April 1970.

8. American Academy of Pediatrics Subcommittee on Accidental Poisoning. Prevention, diagnosis, and treatment of lead poisoning in children. Pediatrics, 44:No.2, 291-298, August 1969.

9. Perlstein, M.A., and R. Attala. Neurologic sequelae of plumbism in children. Clin. Pediat. 5:292-298, May 1966.

10. Chisolm, J., and H. Harrison. Quantitative urinary coproporphyrin excretion and its relationship to EDTA. J. Clin. Invest. 35:1131-38, October 1956.

11. Whitaker, J., W. Austin, and J. Nelson. American Journal of Diseases of Children, Ethylenediamine calcium disodium diagnostic test for early lead poisoning. 102:779-80, Nov. 1961.

12. Haeger-Aronsen, B. Studies in urinary excretion of 5-aminolaevulic acid and other haem precursors in lead workers and lead-intoxicated rabbits. Scan J. Clin. Lab. Invest., 12(Suppl 47):1-128, 1960.

13. Feldman, F., H.C. Lichtman, S. Oransky, E. Sta Ana, and L. Riser. Serum delta-aminolevulinic acid in plumbism. J. Pediat. 74:No. 6, 917-923, June 1969.

14. Hsia, D.Y. and M. Page. Coproporphyrin studies in children. I. Urinary coproporphyrin excretion in normal children, Published in Proc. Soc. Exper. Biol. Med., 85:86-88, 1954.

15. Barltrop, D. The excretion of delta-aminolevulinic acid by children. Acta. Pediatrics Scandinavica, 56:265-68, May 1967.

16. Benson, F., and J. Chisolm. A reliable qualitative urine coproporphyrin test for lead intoxication in young children. J. Pediat., 56:No. 6, 759-767, June 1960.