Lead Absorption in Children

Management, Clinical, and Environmental Aspects

J. Julian Chisolm, Jr.
David M. O'Hara

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J. Julian Chisolm, Jr.
Project Director
Lead Poisoning Clinic
The John F. Kennedy Institute
Baltimore, Maryland

David M. O'Hara
Acting Director, Social Work
The John F. Kennedy Institute
Baltimore, Maryland

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Contributors

Paul Burgan, M.D., Ph.D., F.A.A.P.
Mount Washington Pediatric Hospital
Baltimore, Maryland

Michael F. Cataldo, Ph.D.
The John F. Kennedy Institute
Baltimore, Maryland

Lawrence Chadzynski, R.S.
Detroit Department of Health
Herman Kiefer Health Complex
Detroit, Michigan

Evan Charney, M.D.
Sinai Hospital of Baltimore, Inc.
Baltimore, Maryland

J. Julian Chisolm, Jr., M.D.
Lead Poisoning Clinic
The John F. Kennedy Institute
Baltimore, Maryland

Jack W. Finney, M.A.
Pediatric Research Institute
University of Kansas School of Medicine
Kansas City, Kansas

Alan Goldberg, Ph.D.
Department of Environmental Health Sciences
Division of Toxicology
Johns Hopkins University School of Hygiene
Baltimore, Maryland

Robert A. Goyer, M.D.
National Institute of Environmental Health Sciences
Research Triangle Park, North Carolina

John W. Graef, M.D.
The Children's Medical Center
Boston, Massachusetts

Paul B. Hammond, D.V.M., Ph.D.
Institute of Environmental Health
Kettering Laboratory
University of Cincinnati Medical Center
Cincinnati, Ohio

Vernon N. Houk, M.D.
Bureau of State Services
Center for Disease Control
Atlanta, Georgia

Jane S. Lin-Fu, M.D.
Office for Maternal & Child Health
Bureau of Community Health Services
Rockville, Maryland

Nancy A. Madden, Ph.D.
Department of Psychiatry
University of Maryland School of Medicine
Baltimore, Maryland

Kathryn R. Mahaffey, Ph.D.
Division of Nutrition, FDA
Cincinnati, Ohio

Christopher Milar, Ph.D.
Division for Disorders of Development and Learning
University of North Carolina School of Medicine
Chapel Hill, North Carolina

Douglas G. Mitchell, Ph.D.
New York State Department of Health
Division of Laboratories and Research
Albany, New York

Hugo W. Moser, M.D.
The John F. Kennedy Institute
Baltimore, Maryland

Paul Mushak, Ph.D.
Division of Environmental Pathology
University of North Carolina School of Medicine
Chapel Hill, North Carolina

David M. O'Hara, D.A.S.S.
The John F. Kennedy Institute
Baltimore, Maryland

Lawrence Reiter, Ph.D.
Health Effects Research Laboratory
U.S. Environmental Protection Agency
Research Triangle Park, North Carolina

Walter J. Rogan, M.D.
National Institute of Environmental Health Sciences
Research Triangle Park, North Carolina

Dennis C. Russo, Ph.D.
Children's Hospital Medical Center and Harvard Medical School
Boston, Massachusetts

Estelle Siker, M.D., M.P.H.
Community Health Division
Connecticut State Department of Health Services
Hartford, Connecticut

Walter J. Sobolesky, M.S.
Accident Control Section
Department of Public Health
Community Health Services
Philadelphia, Pennsylvania

Preface

Clinical management of the asymptomatic child with increased lead absorption—to be rational—demands an understanding of the many factors which influence the course and outcome of this disorder. The contributions of several disciplines need to be coordinated for effective management. These include: heavy metal metabolism and toxicology; neurotoxicology; nutrition; environmental, behavioral and social sciences; public health; analytical chemistry; environmental hygiene and pediatrics. This volume represents the proceedings of a conference organized under the auspices and support of the Office of Maternal and Child Health, Bureau of Community Health Services, for the purpose of amplifying the clinical dimensions of their various scientific perspectives. Formal papers and discussion focus on the contribution of each discipline to the management of the child with increased lead absorption and are grouped into four sections as follows.

The first part of the book lays the background in metabolism and overall toxicity of lead. As with many environmental toxins, lead has a very slow turnover rate in the body and affects many different organ systems, especially the hematopoietic, nervous and renal systems. Experimental studies have vastly expanded our knowledge of the neurotoxicological effects of lead and reveal that low levels of lead can not only induce maturational delay but also morphological changes that are likely to be permanent. Clinical studies, despite their individual faults, strongly suggest that increased lead absorption without symptoms during early life can lead to subtle disturbances in learning. A lively discussion, including the presentation of some provocative new clinical data, concludes this section.

The second part focuses on environmental, nutritional, behavioral and social factors that can modify the absorption and toxicity of lead in children. The implications of nutritional data suggest that adequate nutrition may play a much greater role in prevention than in treatment. Recent evidence clearly establishes the critical interplay of lead-bearing dust with the normal hand-to-mouth activity of young children as the major route of lead into the body. Not only are dust lead levels high in the home environment of the affected children, but limited data

presented at the conference indicate that lead dust is extremely difficult to remove. Clearly, more research in this area is needed. Without the appropriate environmental management, other behavioral and social interventions are of limited efficacy.

Later, the need for close coordination of the health agency, the analytical laboratory, environmental hygiene and clinical disciplines is emphasized. Traditionally, lead has been removed from painted surfaces of dwellings by burning and sanding, which leaves a residue of fine particulate lead in the home. Newer abatement approaches using heat guns and avoidance of sanding give promise of less hazardous abatement procedures in the future. In clinical management, there are clearly similarities between occupational exposure and exposure during childhood. The "dusty trades" have long been recognized as the more hazardous types of occupational exposure to lead. Only recently has the link between dust and increased lead absorption in childhood been appreciated. Treatment of the child, however, is far more difficult than that of the worker. The workman can be removed from his primary source of exposure, the work place, while the child's principal source of exposure is his primary residence, from which he cannot be easily removed. This complicates and limits the effectiveness of treatment in children. Examples are cited of recurrences where exposure in the home persists and, by contrast, improvement often occurs spontaneously, when a new residence is found.

Finally, future clinical directions and research needs are summarized. Walter Rogan provides a comprehensive overview and yet another illustration of idiosyncratic toxicity on the Green Parrot Goat Farm.

The conference and this volume would not have been possible without Dr. Jane S. Lin-Fu, whose longstanding concern for this problem was borne out by her constant support and excellent summation of the history of lead poisoning in children. Thanks must also be given to the participants for their careful attention to the clinical implications of their contributions. Not least is our appreciation for the encouragement of the John F. Kennedy Institute, Hugo W. Moser, M.D., Director.

J. Julian Chisolm, Jr.
David M. O'Hara

Frequently Used Abbreviations

Pb. . . . . .lead

PbA . . . . . .air lead (μg Pb/m^3 of air)

PbD . . . . . .dust lead ppm or μg/unit of exposed surface

PbH . . . . . .hand lead (μg Pb/hand) as washed off

PbB . . . . . .blood lead (μg Pb/dl whole blood)

PbU . . . . . .urine lead (μg Pb/L or μg Pb/24 hr as defined)

PbU-EDTA .chelatable lead, usually expressed as μg Pb excreted/mg CaEDTA administered/24 hr

CaEDTA . . .calcium disodium ethylenediaminetetraacetate

ALAU. . . . . δ-aminolevulinic acid in urine

ALAD. . . . . δ-aminolevulinate dehydratase

FEP . . . . . ''free'' erythrocyte protoporphyrin (μg protoporphyrin/dlerythrocytes)

EP . . . . . .erythrocyte protoporphyrin (μg protoporphyrin/dl whole blood)

ZnPP . . . . . .zinc protoporphyrin

Contents

1. The Evolution of Childhood Lead Poisoning as a Public Health Problem

Jane S. Lin-Fu

Introduction

This paper reviews briefly the evolution of childhood lead poisoning as a public health problem and the metamorphosis in our concept of this illness.

Lead is an extremely useful metal; it has almost become an intrinsic part of our modern way of life. In solder for food cans and electronic equipment, in gasoline, in car and other storage batteries, in craft materials, art works and newsprints, in brasswares, in dinnerwares, crystals and plastics, in caulking and sound-proofing material for buildings, ships and jet planes, in cable covering of intercontinental communication systems, in ammunition, in curtain weights and sinkers for fishing, this metal has found almost endless application since its discovery by man more than 5000 years ago. Following the Industrial Revolution of the 18th century, and particularly since the early 1940's, the use of lead has increased rapidly. Between 1940 and 1977, the consumption of lead in the U.S. almost doubled from 782,000 tons to an estimated 1,505,000 tons (Lead Industries Association, 1978). Between 1935 and 1977, the amount of lead used as a gasoline additive in the U.S. increased six-fold from 37,000 tons to 233,000 tons per year (Lead Industries Association, 1978; National Academy of Science, 1972). It is important to recognize, however, that even though the comfort and convenience of our life style may depend on lead, our life itself does not. Lead plays no physiologic role in the human body.

Industrialization, Reproductive Failures and Congenital Lead Poisoning

Our knowledge of lead toxicity dates back at least 2000 years. The problem in children probably first drew attention as congenital lead poisoning in offspring of lead workers following industrialization. In the 18th and 19th centuries, sterility, abortion, stillbirth and premature delivery were common not only in female lead workers, but also among wives of men who worked in lead industries. Infant mortality was extremely high in their offspring, among whom congenital lead poisoning was manifested as low birth weight, convulsions, failure to thrive and mental retardation (Hamilton and Hardy, 1949; Oliver, 1911). Although a high frequency of reproductive failure also occurred in wives of lead workers, removal of women from lead industries in the late 19th century was viewed as some sort of solution to occupational lead poisoning. This is analogous to the removal of children from homes with peeling lead paint without correcting the lead hazard, a practice some still use as a solution to the lead paint poisoning problem today.

With gradual improvement in industrial hygienic standards, congenital lead poisoning became a rarity in the literature. But as late as the period from 1931 to 1940, 853 deaths from lead poisoning in adults were reported in the U.S. (McDonald and Kaplan, 1942). These reported fatality figures tell little of the actual magnitude of the problem of occupational lead poisoning and its byproduct, congenital lead poisoning. The silence of the literature on congenital lead poisoning after the early part of the 20th century should therefore not be equated with the non-existence of the problem.

Discovery of Lead Paint Poisoning in Children

Congenital lead poisoning results from indirect in utero exposure. The prevalence of lead poisoning in children through direct exposure was first observed in Queensland, Australia, in the 1890's (Gibson et al., 1892). The source of lead remained a mystery until 1904, when Gibson traced it to the paint used on railings of verandas and walls in the homes of the children (Gibson, 1904). In the U.S., physicians viewed the Australian experience with some skepticism. In 1914, Thomas and Blackfan of Johns Hopkins published what was perhaps the first case report of lead paint poisoning in the U.S. in a child who chewed the paint off his crib. These authors noted that childhood lead poisoning was uncommon in the U.S. and commented that Australian children "seem peculiar-

ly liable to lead poisoning'' (Thomas and Blackfan, 1914). It was three years later, when Blackfan (1917) reported four other children with convulsions due to lead, that he acknowledged the importance of the Australian studies and noted that lead as a cause of convulsions in children had been largely ignored by U.S. physicians.

A handful of reports of lead meningitis in infants and young children followed Blackfan's paper, but the prevalence of lead poisoning among U.S. children did not gain recognition until 1924, when Ruddock made the important observation that children lived in a ''lead world'' and that pica, or a perverted appetite for non-food items, was important in introducing this toxic element into children's bodies (Ruddock, 1924). In lead paint on houses and furniture, toys, food coloring, food receptacles, cosmetics and even medicinal ointment, lead finds its way into children's mouths. In 1926, McKhann of Boston published the first study of a large series of children with lead poisoning in the U.S. Ingestion of lead paint on cribs and furniture was the cause in most cases (McKhann, 1926).

Lead Poisoning from Burning Battery Casings

In Baltimore, two fatal cases of lead paint poisoning in children attracted the attention of the Health Department in 1931 (Chronology of Lead Poisoning Control, Baltimore, 1931–1971, 1971). This was followed in 1932 by a mass outbreak of lead poisoning primarily involving children which was due to burning of battery casings for fuel in the home (Williams et al., 1933). Similar episodes occurred in Philadelphia, Chicago, Long Island and Detroit (Levinson and Harris, 1936). These outbreaks focused some attention on the problem of lead poisoning in children and its predilection for the poor. In 1935, the Baltimore Health Department's Division of Chemistry began to provide free blood lead tests to physicians and hospitals. This resulted in an increase in the number of diagnosed cases of lead poisoning in children (Kaplan and McDonald, 1942).

Limited Case Findings, 1950's and 1960's

Although the Baltimore Health Department became interested in childhood lead poisoning in the early 1930's, elsewhere health officials gave the problem virtually no attention until the early 1950's. Then a few cities such as New York, Chicago and Philadelphia exerted some effort at case finding and public educa-

tion. Wherever health workers made such an effort, the number of reported cases invariably increased and the severity of diagnosed cases and fatality rate decreased. But even into the mid-1960's, encephalopathy had often set in before lead poisoning in children was diagnosed. Between 1959 and 1963, physicians at Chicago's Cook County Hospital treated 182 children for lead encephalopathy, of whom 51, or 28%, died (Greengard et al., 1965). Among survivors, sequelae such as convulsions, mental retardation, blindness, cerebral palsy, behavior disorders and learning disabilities were common (Perlstein and Attala, 1966).

That undue exposure of young children to lead reached epidemic proportions is best illustrated by the report of Bradley et al. (1956) of Baltimore. Among 333 children 7 to 60 mos of age from low income areas seen at well-baby clinics and the pediatric outpatient department of the University of Maryland between August 1, 1953, and September 1, 1954, 299 or 90% had blood lead levels of $\geq$30 μg/100 ml; 86 or 26% had levels of $\geq$60 μg/100 ml; 197 or 59% had a positive urinary coproporphyrin test; 77 or 23% had positive x-ray evidence of dense metaphyseal lines (so-called "lead lines"). Bradley et al. observed that a blood lead level of $\geq$50 μg/100 ml was associated with an increase in other findings compatible with the diagnosis of lead poisoning and suggested that this be used as the upper limit of normal. This suggestion went unheeded, and three years later the USPHS recommended that blood lead levels of 60 to 80 μg/100 ml be considered evidence of abnormal lead absorption (National Clearinghouse for Poison Control, 1959).

Epidemiology of Childhood Lead Poisoning, 1950's and 1960's

The epidemiology reports of childhood lead poisoning from different cities from the early 1950's to the middle 1960's yielded a surprisingly uniform pattern (Lin-Fu, 1967).

> Childhood lead poisoning was inextricably related to dilapidated housing where peeling lead paint and broken painted plaster were readily available; the high risk areas or "lead belts" were practically synonymous with large inner city slums. Pica was an important contributing factor. Children 1 to 6 yr, and particularly those 1 to 3 yr, were at greatest risk. Blacks had a higher incidence than whites. Siblings were often affected together. The disease affects children year-round, but symptoms and lead encephalopathy occurred more frequently in the summer. Not unexpectedly, recurrence was the rule unless the lead paint hazard was corrected and, with each recurrence, the prognosis became worse.

Little Action Despite Formidable Data

The formidable data on childhood lead poisoning published in the 1950's and early 1960's troubled surprisingly few health officials, and the public was tragically unaware of their existence. Several factors accounted for this. First, many had mistakenly thought that replacement of the lead pigment by titanium oxide in the early 1940's had solved the lead problem. They failed to realize that millions of houses with layers of old lead paint remained occupied. Others who were aware of the problem considered it an illness inevitable to slum dwelling for which little could be done. The lack of adequate housing codes and the failure to enforce existing ones for financial and other reasons further frustrated interested health workers who knew the return of children to uncorrected housing virtually guaranteed a recurrence. The non-specificity of the symptoms of lead poisoning also confounded the problem, not only for parents, but also for uninformed physicians, since routine physical examination and laboratory studies will not provide an unsuspecting physician with the correct diagnosis (Lin-Fu, 1979).

Acknowledgment of the Problem, Mid-1960's

The turmoil and awakening of social conscience of the mid-1960's brought with it a sudden acknowledgment of the magnitude of childhood lead poisoning. In 1970, the U.S. Surgeon General issued a statement on the disease which shifted the focus from case finding to prevention. He advocated mass screening and early identification of children with evidence of undue lead absorption, defined as a confirmed blood lead level of 40 μg/100 ml or more (Department of Health, Education and Welfare, 1971). That progress cannot be made easily is illustrated by the backward step taken a year later when the American Academy of Pediatrics recommended that the environment of children be investigated only after demonstration of two blood lead levels of ⩾50 μg/100 ml (American Academy of Pediatrics, 1971).

Mass Screening and Epidemiology, 1970's

The 1971 Lead-Based Paint Poisoning Prevention Act provided, among other things, for Federal assistance through DHEW to help communities carry out screening and treatment programs. Mass screening under the act began in mid-