ENVIRONMENTAL HEALTH ABSTRACTS & BIBLIOGRAPHY

August 1980

focus: LEAD POISONING

DEPARTMENT OF HEALTH AND HUMAN SERVICES - PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL, ATLANTA, GEORGIA 30333

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Foreword

Environmental Health Abstracts and Bibliography presents a survey of recently published literature in the field. Effort is made to keep the abstracts as current as possible and sufficiently informative to enable the reader to decide whether the original article would be of interest to him or her. The journals in which articles originally appeared should be checked for reprint addresses. The Center for Disease Control is unable to supply reprints of articles which are cited in this publication.

In compiling these abstracts we utilize the National Library of Medicine's interactive retrieval service, MEDLARS II. Under this system, both foreign and domestic biomedical periodicals are searched for material dealing with or related to environmental health. We also utilize the libraries of Emory University, the Center for Disease Control and other federal agencies. Abbreviations of periodical titles are those used by MEDLARS and listed in the National Library of Medicine's *List of Journals Indexed in Index Medicus.*

Future issues of *Environmental Health Abstracts and Bibliography* will be devoted to various environmental health topics. Individuals desiring to be placed on the mailing key to receive future issues as published should write to the Center for Disease Control, Attention: Environmental Health Services Division, Bureau of State Services, Atlanta, Georgia 30333.

Vernon N. Houk, M.D.
Director
Environmental Health Services Division

Contents

GENERAL AND EPIDEMIOLOGY

Age and Sensitivity to Lead Toxicity: A Review

E.B. McCabe. ENVIRON HEALTH PERSPECT 29:29-33, Apr 79.

Author's abstract: During the past 20 years considerable attention has been focused on the epidemiologic features of childhood lead poisoning in the United States. Large numbers of children with symptomatic intoxication, as well as those with incipient symptoms, were commonplace a decade ago for physicians working in inner-city hospitals. With the recent availability of improved screening techniques, as well as a variety of environmental control measures, the incidence of symptomatic lead poisoning in children has diminished significantly in recent years. With the focus shifting from children with dangerously elevated body lead burdens to those with less significant exposures, increased attention has been directed to the various inherent metabolic and physical characteristics of the young that may influence the toxic effects of lead exposure. A number of differences with respect to lead exposure, absorption and retention, and varying nutritional conditions between children and older individuals are discussed. Experimental studies dealing with age differences of lead-treated animals are examined, and relevant human studies are reviewed.

Nutrient-Toxicant Interactions: Susceptible Populations

K.R. Mahaffey and J.E. Vanderveen. ENVIRON HEALTH PERSPECT 29:81-7, Apr 79.

Authors' abstract: Nutritional status can substantially modify the toxicity of environmental pollutants. Investigations with experimental animals and epidemiological observations on humans have established the role of nutrition in altering susceptibility to a variety of pollutants including pesticides and heavy metals. The degree of nutritional deficiency that alters susceptibility need not be severe. Frequently only biochemical indications of nutritional deficiency can be associated with changes in the dose-response of an animal or person to a toxic compound.

Lead Toxicity and Nutritional Deficiencies

O.A. Levander. ENVIRON HEALTH PERSPECT 29:115-25, Apr 79.

Author's abstract: Under appropriate conditions, deficiencies of certain minerals and vitamins as well as high intakes of dietary fat increase the toxicity of a given dose of lead in experimental animals. The severity of lead poisoning can also be increased by the consumption of either deficient or excessive levels of protein. Mineral deficiencies appear to have some of the most profound effects on lead toxicity, since the consequences of plumbism can be exaggerated by feeding diets low in calcium, phosphorus, iron, zinc, and in some cases, copper. Evidence for an antagonism between lead and nutritional levels of selenium is inconclusive. Vitamin E deficiency and lead poisoning interact to produce an anemia in rats that is more severe than that caused by either treatment alone. Lead apparently exerts a pro-oxidant stress on the red cell, thereby causing its accelerated destruction. One of the biochemical mechanisms of lead poisoning may be the disruption of normal membrane architecture, thereby leading to peroxidative damage. Epidemiological surveys have suggested a negative correlation between the poor nutritional status of children with regard to calcium and the concentration of lead in blood. Other examples of potential interactions of mineral status and lead poisoning in humans include the hypothesized hazards of soft water to public health in areas with lead plumbing and the possible role of mineral deficiencies in the etiology of pica. Experimental studies have shown that in some situations combined nutritional deficiencies can have an additive effect in potentiating lead toxicity.

Diets and Lead Blood Levels of Children Who Practice Pica

N.E. Johnson and K. Tenuta. ENVIRON RES 18(2):369-76, Apr 79

Authors' abstract: Diets of 43 children from 1 to 6 years of age were assessed and nutrient intakes were

compared to blood lead values. Children with low (12-29 μg/dl), moderate (30-49 μg/dl), and high (50-67 μg/dl) blood lead levels had average daily calcium intakes of 615, 593, and 463 mg, respectively. Thirty-three percent of children in the low blood lead group consumed less than 70% of the recommended amounts of calcium compared to 83% of children in the high blood lead group. Blood lead values of individual children were negatively correlated ($r = -0.327$, $p < 0.05$) with the number of servings from the group of foods classified as a milk group. The incidence of pica was higher in children with moderate and high blood lead levels than in those with low blood lead levels. Average zinc intake for all children was 63% of levels recommended (RDA) with a greater percentage of children with high blood lead consuming less than 55% of RDA than children with low and moderate levels.

Behavioral and Neurological Effects of Symptomatic and Asymptomatic Lead Exposure in Children

J.H. Rummo, D.K. Routh, N.J. Rummo, and J.F. Brown. ARCH ENVIRON HEALTH 34(2):120-4, Mar-Apr 79.

Authors' abstract: Forty-five children, 4 to 8 years of age, who had been exposed to environmental lead were studied. The children included an acute encephalopathy group and groups with short- and long-term exposure but without encephalopathy. Control children were matched for age, sex, race, and socioeconomic status with lead-exposed subjects, but lived in post-1945 housing and had negative neurological history and blood tests. The encephalopathy group had a significantly higher incidence of neurological deficits, retarded mental development, and higher hyperactivity than control subjects. Children with short- and long-term exposure short of encephalopathy were somewhat inferior to matched control subjects, but not to a statistically significant extent.

Biochemistry and Measurement of Environmental Lead Intoxication

J. Eisinger. Q REV BIOPHYS 11(4):439-66, Nov 78.

Lead has been demonstrated to have harmful effects on the nucleic acids and protein syntheses, such as hematopoiesis, but its equally important interference with the central, peripheral, and autonomic nervous systems is not so well understood. As greater amounts of lead enter the environment, determining safe levels of exposure becomes increasingly important. Because so many biochemical interactions of lead are general in nature and can occur in any part of the living system, early symptoms of plumbism may vary from individual to individual and are non-specific. Epidemiological studies are needed to correlate these subtle symptoms with undue exposure to lead, but proper methods of detecting chronic low levels of exposure have been lacking. A new hematofluorometer developed by Bell Labs can measure levels of zinc protoporphyrin ((ZPP), the result of lead's interference with hematopoiesis and indicative of prolonged exposure) in microsamples of blood. Since this machine is portable, it can be used in the field and should be useful for both screening and epidemiological surveys. The relationship between ZPP and anemia has already been established; more studies, using this instrument, may be able to supply new data to link ZPP levels with the toxic effects of lead on the nervous systems.

Skeletal Concentrations of Lead in Ancient Peruvians

J.E. Erickson, H. Shirahata, and C.C. Patterson. N ENGL J MED 300(17):946-51, 26 Apr 79.

Authors' abstract: The level of biologic lead (expressed as the ratio of atomic lead to atomic calcium) in bones of Peruvians buried 1600 years ago was found to be 3×10^{-8}, as compared to 2100 to 3500×10^{-8} in the bones of present-day residents of England and the United States. The ratio of barium to calcium was 2 to 3×10^{-6} in bones of ancient Peruvians and present-day Americans. Barium and lead have similar morphologic distributions in organisms, so this discrepancy for lead must result from overexposure of present-day people to industrial lead and not from natural variations. The magnitude of this discrepancy has been confirmed by two different lines of investigation not reported in this article. This new evidence suggests that natural interactions of lead in human cells have not yet been determined because reagents, nutrients, and controls used in laboratory and field studies have been contaminated with lead far in excess of naturally occurring levels.

Lead Ingestion in History (Letter)

R.T. Steinbock. N ENGL J MED 301(5):277,2 Aug 79.

Man has been using lead since about 3500 B.C., but the Romans used it most intensively of all. Lead was a major part of their water system; it lined their aqueducts and was a basic element of Roman plumbing. [The very word "plumping" derives from the Latin "plumbum," or lead.] Lead, mixed with silver,

coated their cooper cooking utensils; lead vessels were used to make wine, and lead was even added to the finished product as a preservative or a flavoring agent. Wine, prepared as the Romans prepared it, has been analyzed for its lead content and levels varying from 390 mg/l to 781 mg/l have been found. [Compare the U.S. Environmental Protection Agency's standard for drinking water–0.5 mgPb/l.] Lead poisoning has been proposed as a key factor in the fall of the Roman Empire. English port wines bottled between 1770 and 1820 also have been analysed; their lead levels ranged between 320 μg/l and 1900 μg/l. Since increased lead intake can cause saturnine gout, chronic consumption of similar wines may have been responsible for the "epidemic" of gout among the English gentry in the 18th and 19th centuries. In 1724 the Massachusetts Bay Company passed the first public health act in the American colonies; it forbad the use of lead apparatus to distill rum.

Lead Poisoning

D. Pincus and C.V. Saccar. AM FAM PHYSICIAN 19(6):120-4, Jun 79.

Lead poisoning can be easily cured in its earliest stages, but has nonspecific symptoms and is difficult to diagnose. Early lead encephalopathy, characterized by persistent vomiting, drowsiness, and ataxia, may suddenly develop into severe encephalopathy; then treatment is difficult and the prognosis unfavorable. Blood lead levels (PbB) can be determined directly through laboratory analysis; readings >29 μg/dl are considered abnormal. Measurements of elevated erythrocyte protoporphyrin (EP) levels, caused by the interference of lead with heme synthesis, may be a better indication of lead intoxication than PbB; elevated EP levels are also indicative of iron deficiency, sickle cell anemia, and protoporphyria. An EP level ⩾50 μg/dl is symptomatic of undue lead absorption. Plumbism also causes disturbances in cerebrospinal fluid and the renal system. X-rays of the abdomen and long bones may reveal lead flakes and lead lines respectively; these can confirm a diagnosis, but negative results should never eliminate the possibility of lead poisoning. In instances where there is doubt about the need for chelation, the calcium disodium edetate mobilization test should be considered. Chelation is the preferred treatment for lead encephalopathy. Injections of calcium disodium edetate and dimercaprol, usually in conjunction, are recommended for acute encephalopathy, while penicillamine (not yet approved by the FDA) is useful for long-term therapy. The article contains two tables: "Risk Classification for Asymptomatic Children" and "Revised Dosage Schedule for Chelating Agents (January 1978)."

Lead Poisoning in Children

B.A. Alli. J NATL MED ASSOC 69(11):797-8, Nov 77.

The article discusses the various ways the three cities of Baltimore, New York, and Chicago have dealt with the lead poisoning problem. A survey of their experiences suggests that a successful lead poisoning abatement program would consist of the following components: educational campaigns for health professionals and the public; case finding and followup, including home visits to check for flaking paint and other lead hazards; legislation and enforcement; research; and improved housing. Greater attention should be paid to pica throughout this process, particularly in the area of research.

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An Investigation of Elevated Blood Lead Levels in Detroit Children

G.T. Haar and L. Chadzynski. ARCH ENVIRON HEALTH 34(3):145-50, May-Jun 79.

Authors' abstract: The findings of this report are based on data from 1,309 children living near three heavily traveled streets in Detroit. This study was designed to determine the most probable reasons for elevated blood lead levels in young children.

The authors used a linear multivariate regression to evaluate the effects of distance from the highway, age and sex of the child, and housing condition. The authors found that the distance the child lived from the highway did not have a measurable effect on blood lead level. Housing condition played the most important role in determining blood lead level. Poorer housing caused a higher average blood lead level and caused the blood lead distribution to skew, giving a significantly greater number of blood leads over 40 μg/100 ml. Age also was important in determining blood lead level. There was a decrease of about 1 μg/100 ml for each increase of one year in the child's age. Sex also appeared to play a small role. In all three areas of the study, blood lead levels were slightly higher in girls than in boys. This study shows, as many have before, that the primary cause of the lead problem with children can be greatly diminished by improved housing.

Cardiovascular Disease and Trace Metals

A.G. Shaper. PROC R SOC LOND (BIOL) 205 (1158):135-43, 18 Jul 79.

Author's abstract: Cardiovascular disease is a major cause of morbidity and mortality in the United Kingdom and other developed countries. In the United Kingdom, mortality from coronary heart disease has increased progressively over the past 25 years, particularly in males. This paper examines the possible role of trace metals in the development of cardiovascular disease, with particular reference to the effects of cobalt, cadmium, and lead in myocardial disease, atherosclerosis, and hypertension. It is concluded that cobalt is an unimportant factor in community levels of cardiovascular disease, that cadmium has striking effects on blood pressure in animals, and that there is some evidence for an association between environmental lead and high blood pressure.

Lead Absorption by Children of Battery Workers (Letter)

L.A. O'Tuama, J.F. Rogers, and W. Rogan. JAMA 241 (18):1893, 4 May 79.

A 24-year-old female battery worker with intermittent headaches, dizziness, and amnesia was diagnosed as lead poisoned only after her 20-month-old son showed a high blood lead level on routine screening. Three of her children, and children in the families of five other workers, showed evidence of increased lead absorption, which seemed due to contact with the parents' work clothes. In five of the six families, the mother was the employee. None of the children was symptomatic.

Authors conclude: "The present episode illustrates that employed women likewise transmit industrial toxins and the absence of symptoms in their families does not rule out excess exposure. The importance of obtaining occupational histories in female as well as male patients is obvious."

El Paso Revisited. Epidemiologic Followup of an Environmental Lead Problem

D.L. Morse, P.J. Landrigan, B.F. Rosenblum, J.S. Hubert, and J. Housworth. JAMA 242(8):739-41, 24-31 Aug 79.

Authors' abstract: Increased lead absorption was found in 1972 in 56% of 256 children aged 1 to 18 years who lived within 1.6 km of a lead-emitting ore smeltery in El Paso, Texas. Engineering improvements have subsequently reduced emissions from the smeltery, and levels of lead in air, dust, and soil have declined. To evaluate the impact of these environmental improvements on lead absorption, we conducted a followup study in 1977 of blood lead levels in 140 children aged 1 through 18 years who lived

within 1.6 km of the smeltery. Mean blood lead levels were found to have decreased from 41.4 to 17.7 μg/dl in children living within 0.8 km of the plant and from 31.2 to 20.2 μg/dl in children living 0.8 to 1.6 km. These data argue for the feasibility of reducing children's lead absorption near primary lead smelters.

Occupational Lead Exposure in Denmark: Screening with the Hematofluorometer

P. Grandjean. BR J IND MED 36(1):52-8, Feb 79.

Author's abstract: The zinc protoporphyrin/hemoglobin (ZPP/Hb) ratio was measured in the field with a hematofluorometer. A significant increase in ZPP/Hb ratio with advancing age was found in 1,295 men who denied any excess exposure to lead. Ninety-seven percent of the results were below 110 μmol ZPP/mol Hb(Fe) (4.4 μg ZPP/g Hb). The ZPP/Hb ratio was determined in a lead-exposed population of 2,275 men, and in 305 a blood lead analysis was also performed. A blood lead limit of 2.9 μmol/l (60 μg/100 ml) corresponds to about 500 μmol ZPP/mol Hb(Fe) (20 μg/g). This limit was exceeded in workers engaged in secondary lead smelting, storage battery manufacture, car radiator repair, crystal glass manufacture, storage battery repair, ship breaking, metal foundries, the ceramic industry, scrap metal handling, and PVC plastic manufacture. Other occupations caused lower lead exposures with ZPP/Hb ratios between 110 and 500 μmol ZPP/mol Hb(Fe): such ratios were found in men from shooting ranges, in leaded pane manufacturers, gunsmiths, car paint sprayers, type setters, steel rolling mill workers, shipbuilders and welders, car mechanics, lead pigment handlers, and solderers. Increased ZPP/Hb ratios and blood lead levels in 210 workers were associated with a decrease in hemoglobin concentration in the blood. Thus, the hematofluorometer has proved to be very useful for screening purposes. A blood lead determination should be performed if the ZPP/Hb ratio exceeds 300 μmol ZPP/mol Hb(Fe) (12 μg/g).

Occupational Lead Poisoning in the United States: Clinical and Biochemical Findings Related to Blood Lead Levels.

E.L. Baker, Jr., P.J. Landrigan, A.G. Barbour, D.H. Cox, D.S. Folland, R.N. Ligo, and J. Throckmorton. BR J IND MED 36(4):314-22, Nov 79.

Authors' abstract: Dose-response relationships between blood lead levels and toxic effects have been evaluated in 160 lead workers in two smelters and a chemicals plant. Blood lead levels ranged from 0.77 to 13.51 μmol/l (16-280 μg/dl). Clinical evidence of toxic exposure was found in 70 workers (44%), including colic in 33, wrist or ankle extensor muscle weakness in 12, anemia (Hgb less than 8.69 μmol/l (Hb/4) or 14.0 gm/dl) in 27, elevated blood urea nitrogen (≥7.14 mmol/l or 20 mg/dl) in 28, and possible encephalopathy in two. No toxicity was detected at blood lead levels below 1.93 μmol/l (40 μg/dl). However, 13% of workers with blood lead levels of 1.93 to 3.81 μmol/l (40-79 μg/dl) had extensor muscle weakness or gastrointestinal symptoms. Anemia was found in 5% of workers with lead levels of 1.93-2.85 μmol/l (40-59 μg/dl), in 14% with levels of 2.90 to 3.81 μmol/l (60-79 μg/dl), and in 36% with levels ≥3.86 μmol/l (80 μg/dl). Elevated blood urea nitrogen occurred in long-term lead workers. All but three workers with increased blood urea nitrogen had at least four years occupational lead exposure, and nine had received oral chelation; eight of this group had reduced creatinine clearance, and eight had decreased renal concentrating ability. These data support the establishment of a permissible biological limit for blood lead at a level between 1.93 and 2.90 μmol/l (40-60 μg/dl).

Lead Poisoning from a Gunshot Wound. Report of a Case and Review of the Literature

R.O. Dillman, C.K. Crumb, and M.J. Lidsky. AM J MED 66(3):509-14, Mar 79.

Authors' abstract: A man was hospitalized on three occasions for symptoms of lead intoxication 20 to 25 years after a gunshot wound that resulted in retention of a lead bullet in his hip joint. The potential for lead toxicity as a complication of a lead missile injury appears to be related to (1) the surface area of lead exposed for dissolution, (2) the location of the lead projectile, and (3) the length of time during which body tissues are exposed to absorbable lead. Cases of lead poisoning of immediate onset resulting from lead shot have been reported in Europe, but all documented cases of ammunition-related plumbism reported in the United States have involved synovial fluid dissolution of a single lead bullet over many years. The solvent characteristics of synovial fluid and associated local arthritis are apparently important factors in the dissolution and absorption of lead from projectiles located in joints. Awareness that lead intoxication can be a complication of retained lead projectiles should allow rapid institution of appropriate diagnostic and therapeutic modalities when such a clinical situation arises.

Exposure of Children to Lead in Drinking Water

D.L. Morse, W.N. Watson, J. Housworth, L.E. Witherell, and P.J. Landrigan. AM J PUBLIC HEALTH 69(7):711-2, Jul 79.

In an investigation of children of workers in a lead battery plant in Bennington, Vermont (population 15,000), several children in the control group were observed to have elevated blood lead levels also. Later it was determined that the lead content in the tap water in the home of one child exceeded the standards set by the Environmental Protection Agency (EPA); further investigation revealed that approximately one-third of the homes in Bennington failed to meet these requirements. The Vermont Department of Health asked the Center for Disease Control to participate in a joint study to assess the possible harmful effects of these water lead levels; no significant correlation between water lead levels and blood lead levels in the exposed children could be determined. The threshold of daily lead intake for the bioaccumulation of lead is estimated to be 100 μgPb/day for infants and 300 μgPb/day for children; it is theorized that the amount of lead in Bennington's water supply, even combined with other sources of environmental lead, does not exceed these amounts. However, Bennington is located in a semi-rural setting and does not have the same pollution problems that other larger, more crowded cities have. Excess water lead levels are more serious in urban areas where background levels in dust and air are much greater than they are in rural areas; in the city, these same water lead levels could be high enough so that the threshold of lead bioaccumulation could be passed and the danger of lead intoxication increased.

The Contribution of Drinking Water Lead to Maternal Blood Level Concentrations

M.R. Moore, A. Goldberg, P.A. Meredith, R. Lees, R.A. Low, and S.J. Pocock. CLIN CHIM ACTA 95(1):129-33, 2 Jul 79.

Authors' abstract: The association between domestic water lead concentrations and blood lead concentrations has been examined in 232 mothers at delivery. The blood level was found to vary significantly with the cube root of the water lead. This association was stronger for first flush water lead rather than for running water lead. This study emphasizes the danger to mothers and to their children of environmental lead over-exposure in areas of soft acid plumbosolvent water.

Environmental Intoxicants and Their Fundamental Interactions

J. Eisinger and W.E. Blumberg. Q REV BIOPHYS 11(4):429-37, Nov 78.

This article defines environmental intoxicants as materials, man-made or natural, in the environment which are dangerous to human health. The health problems caused by them can be divided into two groups: those with a low incidence of disease and environmental disasters. The first group is associated with widely distributed toxicants; the diseases' origins are notoriously difficult to trace and often their connection with environmental intoxicants remains unrecognized. Levels of inorganic lead [known to cause disorders with a wide variety of non-specific and difficult-to-diagnose symptoms] in the atmosphere are some orders of magnitude greater now than in the pre-industrial period. Lead is also associated with environmental disasters. Lead contaminated slag from a battery plant was used as a road fill and to surface a schoolyard in Thailand. Exposed children suffered severely. Zinc protoporphyrin levels, an indication of lead's inhibition of heme synthesis, were a hundred times higher in one Thai village than the average for a rural population in the United States.

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DIAGNOSIS AND SCREENING

Recognition and Management of Children with Increased Lead Absorption

J.J. Chisolm, Jr., and D. Barltrop. ARCH DIS CHILD 54(4):249-62, Apr 79.

From authors' conclusion: There is no reasonable doubt that acute lead encephalopathy can result in severe residual central nervous system (CNS) injury, though clinical studies of asymptomatic or mildly symptomatic plumbism have yielded conflicting results. There is some evidence that a sustained blood lead level (PbB) >50-60 μg/dl during early childhood carries a significant risk of subtle neurobehavioral impairment that will not become evident until later in childhood. Concurrent nutritional deficiencies, especially of iron, calcium, and zinc, as well as the habitual overuse of fat, may be important cofactors in determining the final clinical outcome. Identification of the sources of contamination, and the prompt separation of the child from them, is essential and is the cornerstone of any therapeutic plan. Although the use of combined chelation in acute lead encephalopathy can be life-saving, treatment started after the onset of severe symptoms will not prevent serious CNS sequelae in the survivors. Modern screening tests permit the detection of plumbism before the onset of symptoms, but the indications for the use of chelation agents in asymptomatic cases are uncertain. Pica, the response to assimilated lead, and the pattern of neurodevelopment during the first three to four years of life make the young child more vulnerable to lead overexposure than older children and adults.

Congenital Lead Intoxication

A.E. Timpo, J.S. Amin, M.B. Casalino, and A.M. Yuceoglu. J PEDIATR 94(5):765-7, May 79.

An 8 months' pregnant adolescent's complaint of poorly defined pain in her lower extremities was diagnosed as lead poisoning. Examination had revealed a history of pica (eating paint chips off a wall), a faint lead line on the lower gum, stippling of numerous red blood cells, and a blood lead (PbB) level of 86 μg/dl. This woman's dangerously high PbB level demanded immediate chelation, even though chelation has been demonstrated to have teratogenic effects on the fetuses of experimental animals. On the second day of admission, amniocentesis showed a lead level of 90 μg/dl in the amniotic fluid, coproporphyrin 3+, and negative aminolevulinic acid. The woman later had a normal delivery of a 2.665 kg baby girl; the child exhibited no apparent physical or neurological abnormalities. Cord PbB level was 60 μg/dl with a free erythrocyte protoporphyrin (FEP) level of 350 μg/dl; radiographs showed metaphysitis, submetaphyseal lucencies, and shaft sclerosis of the long bones. Two weeks after birth PbB levels were still high, so the infant was chelated; PbB levels fell to 40 μg/dl. When the baby was 5 months old PbB and FEP levels were still high and another course of chelation was instituted. Afterwards, PbB dropped to 21 μg/dl. When the child was a year-and-a-half old all the apparent ill effects of the plumbism and the chelation had disappeared. However, it must be stressed that the prolonged exposure to lead in utero on optimal brain development and function can be determined only by long-term followup.

Pb Encephalopathy Mimicking Reye Syndrome (Letter)

P.D. Magnus, R.J. Powers, and A. Leong. J PEDIATR 95(3):495, Sep 79.

A 21-month-old black girl was admitted to Children's Hospital of Los Angeles with a 4-day history of vomiting, anorexia, and lethargy. She was anemic (Hgb 7.8 gm/dl; MCV 62[3]) and mildly dehydrated. CSF pressure was normal (5 leukocytes/mm[3]), as were CSF and blood glucose concentrations; however, CSF protein concentration was 173 mg/dl. The initial impressions were iron deficiency anemia with Reye syndrome, toxic encephalopathy, acute gastroenteritis, gastrointestinal obstruction, or hepatitis. Approximately 12 hours after admission, after being rehydrated, the patient had a grand mal seizure lasting 10 minutes and eventually needed prolonged ventilatory assistance. Several hours after the medical management of her seizures and cerebral edema, however, "lead flakes" were noted on the

admission flat plate and long bone films revealed "lead lines." The patient made a successful recovery after several successive chelations. Abnormal liver enzymes were confusing initially, but the CSF protein concentration is usually normal in Reye syndrome and was elevated in this lead-burdened child.

Peripheral Neuropathy in Lead-Intoxicated Sickle Cell Patients

C.E. Imbus, J. Warner, E. Smith, C.H. Pegelow, J.P. Allen, and D.R. Powars. MUSCLE NERVE 1(2): 168-71, Mar-Apr 78.

Authors' abstract: Peripheral neuropathy and hypertension caused by lead intoxication are reported in two children with sickle cell anemia. One child had generalized weakness in the initial occurrence and distal paralysis during a relapse 2 years later. The second child had foot and wrist drop. Both had slow peripheral nerve conduction velocities during the episodes. Chelation therapy was successful and resulted in a return of strength (over a period of several months) and a normalization of the blood pressures. Children with sickle cell anemia who are subjected to lead intoxication appear to be predisposed to peripheral nerve damage.

Selective Screening for Lead Poisoning in an Urban Teaching Practice

J. Froom, V. Boisseau, and A. Sherman. J FAM PRACT 9(1):65-70, Jul 79.

In 1975 the Rochester Family Medicine Program launched a pilot project aimed at selective screening of high-risk children for lead poisoning. Since one of the main causes of plumbism is the ingestion of substances contaminated with lead dust, as might come from old flaking paint, attention was focused on children from poorer urban neighborhoods, ones the census tracts label as socioeconomically "low to middle." Followup of children with elevated blood lead levels is a problem, particularly since they generally come from a section of the population that tends to be very mobile; therefore, a lead screening coordinator was appointed with the important task of keeping track of these children for followup. Other responsibilities included: liaison between physicians, parents, local health officials, and regulatory boards; the initiation and implementation of various outreach programs; and the maintenance of detailed records on all children screened. It was found that request by physician and outreach home visit were the most effective in leading to screening that revealed danger of possible lead intoxication. Selective screening in this instance proved to be more cost efficient than indiscriminate testing. As an added advantage, doctors-in-training became aware of the benefits, and the frustrations, of a screening program by actual participation in such a program.

Bilirubin Sensitivity of Zinc Protoporphyrin Hematofluorometers (Letter)

A.A. Lamola, J. Eisinger, and W.E. Blumberg. J LAB CLIN MED 93(2):345-8, Feb 79.

From authors' introduction: In an article which recently appeared in this Journal [Buhrman et al., *Journal of Laboratory and Clinical Medicine* 91(4): 710-6, April 1978], it was pointed out that the ZZP hematofluorometers used for screening at-risk populations for lead intoxication and for iron deficiency respond to the serum bilirubin as well as to the erythrocyte zinc protoporphyrin levels in blood. It was suggested that this interference taken with normally encountered variable serum bilirubin levels constitutes an important source of error in ZPP assays.

We show that the sensitivity of a ZPP hematofluorometer to bilirubin depends primarily on the choice of the filters used in the excitation and emission optics and may vary from one manufacturer to another. We present data showing that in the Bell Laboratories prototype ZZP hematofluorometer, as well as in one commercial version of this instrument, the relative sensitivity to serum bilirubin is only one-third that in the instrument discussed by Buhrman et al. and that it leads to a negligible error in the determination of the ZZP level of individuals with normally encountered bilirubin levels.

Micromethod for Zinc Protoporphyrin in Erythrocytes: Including New Data on the Absorptivity of Zinc Protoporphyrin and New Observations in Neonates and Sickle Cell Disease

J.J. Chisolm, Jr., and D.H. Brown. BIOCHEM MED 22(2):214-37, Oct 79.

A quantitative fluorometric micromethod specific for zinc protoporphyrin (ZnP) in blood is reported. This method gives results equivalent to a free erythrocyte protoporphyrin (FEP) method when ZnP is the only species of protoporphyrin present. If protoporphyrin IX is also present, the FEP method, which measures both, gives a high result. The fraction of protoporphyrin IX may be calculated from the differences between the FEP and the ZnP. Millimolar absorptivity of ZnP in pyridine (221) and in acetone

containing 8.2% of 0.5 mole aqueous acetic acid per liter (211) has been determined on a sample characterized by chromatography, fluorescence spectroscopy, nuclear magnetic resonance spectroscopy, and elemental analysis. These values are substantially higher than previously reported values. Observations in patients show that protoporphyria is not the only condition in which the concentration of protoporphyrin IX in erythrocytes is elevated. Increase in protoporphyrin IX has been found in sickle cell disease, cord blood from newborns, and in anemic, iron deficient children with, and without, increased lead absorption. Density gradient studies in human cord blood show that protoporphyrin IX is found in the reticulocyte-rich fraction, as has been reported by others in studies of patients with iron deficiency anemia.

Direct Determination of Lead and Cadmium in Blood and Urine by Flameless Atomic Absorption Spectrophotometry

V. Lagesson and L. Andrasko. CLIN CHEM 25(11):1948-53, Nov 79.

Authors' abstract: We describe procedures for direct determination of lead and cadmium in blood and urine by flameless atomic absorption spectrophotometry. Before analysis, the samples are preashed in microboats in an ordinary laboratory oven. In this way, many samples can be prepared and pre-ashed simultaneously. We find the procedures presented in this work to be rapid, accurate, and precise.

Spectro-Fluorometric Determination of Erythrocyte Protoporphyrin by Using Rhodamine-B as Standard

K. Harada, S. Ohmori, and H. Miura. SANGYO IGAKU 21(1):74-9, Jan 79.

In a two-stage operation the erythrocyte protoporphyrin (EP) in 50 μl of whole blood (using ethyl acetate-acetic acid and hydrochloric acid) are reduced to their free base (FEP). An aliquot of the final mixture is read on a spectrofluorometer; rhodamine B-ethylene glycol is used as the standard fluorescence. Since the maximum fluorescence intensity of the standard (F_R: at 577 nm) is equivalent to that of the 2.0×10^{-3} μg/ml protoporphyrin in the blank solution (F_S: at 605 nm), the concentration of FEP is calculated from the formula: EP μg/dl of blood$=(F_S/F_R) \times 0.002 \times (V/0.05) \times 100$, in which 0.002 is the conversion constant. The EP and FEP levels of 48 lead workers were determined by both the present and the classical methods. A close correlation (r=0.96, n=48) was found between the values determined by both methods.

Interlaboratory Comparison of Blood Lead Determinations

C.C. Maher, D.M. Roettgers, and H.J. Conlon. AM IND HYG ASSOC J 40(3):230-7, Mar 79.

Authors' abstract: Results are presented for an interlaboratory proficiency study of blood lead determinations. Samples were pooled from individuals occupationally exposed to lead. The performance of individual laboratories is compared over a 2-year period. With increased emphasis in recent years on proficiency studies of this type, agreement between laboratories has been somewhat improved, but blood remains a difficult matrix in which to measure lead concentration.

Erythrocyte Delta-Aminolevulinic Acid Dehydratase Activity and Changes in Delta-Aminolevulinic Acid Concentration in Various Forms of Anemia

B.C. Campbell, P.A. Meredith, M.R. Moore, and A. Goldberg. BR J HAEMATOL 40(3):397-400, Nov 78.

Authors' abstract: Whole blood delta-aminolevulinic acid (ALA) concentrations and erythrocyte ALA dehydratase activity have been measured in patients with iron deficiency anemia, megaloblastic anemia and secondary anemia, and in normal subjects. ALA concentration was found to be significantly increased in all types of anemia compared with normal. Erythrocyte ALA dehydratase activity was significantly increased in iron deficiency and megaloblastic anemia but not in secondary anemia.

Nutritional Anemias of Childhood

L.C. Wolfe and S.E. Lux. PEDIATR ANN 8(7):435-43, Jul 79.

Three categories of nutritional anemias are discussed: hypochromatic-microcytic, macrocytic, and normocytic. Hypochromatic-microcytic anemia is most often associated with iron deficiency, but also can be caused by lead poisoning, α-thallasemia trait, or β-thallasemia trait. A free erythrocyte protoporphyrin test will be elevated in lead poisoning (>200 μg/100cc RBC) and moderately high in iron deficiency (30-200 μg/100 cc RBC); it is not significant in either thallasemia trait.

Assessing Iron Status of a Population

J.D. Cook and C.A. Finch. AM J CLIN NUTR 32(10):2115-9, Oct 79.

Authors' abstract: Reliable methods for assessing the iron status of a population are essential for developing effective public health measures to combat iron deficiency. The hemoglobin concentration, transferrin saturation, free erythrocyte protoporphyrin, and serum ferritin are all useful, but they vary widely in their specificity and sensitivity for detecting iron deficiency. In applying these laboratory parameters, the usual approach in nutritional surveys is to determine the percentage of values outside the normal range. As an alternative, a model is presented here that uses these measurements to estimate the distribution of iron stores in a population. This approach may be particularly useful for evaluating the effectiveness of iron supplementation and fortification programs.

Biochemical and Behavioral Aspects of Sideropenia

R. Leibel, D. Greenfield, and E. Pollitt. BR J HAEMATOL 41(2):145-50, Feb 79.

This article reviews the literature on the biochemical and behavioral aspects of iron deficiency. Although a "lesion" in almost any of the iron-dependent metabolic pathways could provide a basis for the behavior changes attributed to iron deficiency, recent animal and human research has focused on oxidative and neurotransmitter metabolism.

Behavioral studies on iron-deficient humans have examined the role of pica in affective disturbances and the relationship between iron deficiency and mental development and function, but results have generally been inconclusive. There have also been a number of studies on the relationship of iron deficiency and brain biochemistry. The authors conclude: "If iron status does influence metabolism of the brain, it is likely that the impact of iron deficiency would vary with the developmental status of the brain. Thus, in the older child or adult whose brain growth is complete, one might expect to see neurological or psychological signs consistent with altered neurotransmitter function of oxidative metabolism, whereas in the infant (or older patient who was iron-deficient as an infant) one might expect to see, in addition, signs related to subtle structural alterations in the brain."

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RESEARCH AND EVALUATION

Determination of Carbonic Anhydrase C and Beta 2-Microglobulin by Radioimmunoassay in Urine of Heavy-Metal-Exposed Subjects and Patients with Renal Tubular Acidosis

N. Taniguchi, M. Tanaka, C. Kishihara, H. Ohno, T. Kondo, I. Matsuda, T. Fujino, and M. Harada. ENVIRON RES 20 (1): 154-61, Oct 79.

In a study to determine the relationship between urinary carbonic anhydrase C(CA-C) and β_2-microglobulin (BMG) levels and renal tubular disorders, 12 lead exposed workers from a ceramic factory (mean blood level of 30 ± 4.2 μg/dl and mean urine lead level of 13 ± 3.5 μg/1) were included. When compared with the control group, it was found that 58% of the lead exposed workers had BMG less than 300 μg/g creatinine and a C(Ca-C) less than 40 μg/g creatine. All of the control group (44) fell within this same range.

Assessment of the PKU Card as a Retrospective Index of Neonatal Blood Lead Status

M.E. Morgan, M.A. Hughes, and P.A. Meredith. TOXICOLOGY 12 (3): 307-12, Mar-Apr 79.

Authors' abstract: PKU cards were obtained for 50 one-year-old children whose cord blood lead, maternal blood lead, and household water lead were known. These three values are known to correlate well. Lead values were measured from the blood on the cards and correlated significantly with the other values. However, predictive validity was poor due to card contamination and it is felt that , for the individual patient, this is not a reliable retrospective index of blood lead at the time of birth.

Inhibition of Delta-Aminolevulinic Acid Dehydratase in Human Red Blood Cells by Lead and Activation by Zinc or Cysteine

Y. Mauras and P. Allain. ENZYME 24 (3): 181-7 1979.

Authors' abstract: Inhibition of blood delta-aminolevulinic acid dehydratase (ALA-D) activity by lead was studied in vivo and in vitro. In vivo, a negative linear correlation (r = -0.85) was found between the logarithmic values of ALA-D activity and blood lead levels. In vitro the inhibitory effect of lead on blood ALA-D activity increased both with contact time and contact temperature of lead with blood before ALA-D assay. Maximum enzyme inhibition occurred after 14 hours of contact at 25° C. Inhibition of ALA-D activity by lead, in vivo as well as in vitro, is suppressed by the addition of zinc or cysteine. The logarithmic values of the activity ratios increase linearly with blood lead concentrations. The increase in ALA-D activity brought about by the addition of zinc or cysteine can be used to identify cases of low enzyme activity with no lead intoxication envolved. The same technique can also detect cases in which ALA-D inhibition may be concealed by a presumably high initial enzyme activity as observed in some patients.

The Carcinogenicity of Lead

M.R. Moore and P.A. Meredith. ARCH TOXICOL 42 (2): 87-94, 8 Jun 79.

Authors' abstract: The potential for lead to cause neoplasm in animals and man is reviewed. A multitude of studies indicate that principally renal tumors may be produced by various forms of inorganic lead in small rodents. No human study either of an epidemiological form or of a case report in industrial, agricultural, or community medicine has proven that lead may cause cancer in man.

The Effect of Carbon Monoxide upon Erythrocyte Delta-Aminolevulinic Acid Dehydratase Activity

M.R. Moore and P.A. Meredith. ARCH ENVIRON HEALTH 34 (3): 158-61, May-Jun 79.

Authors' abstract: The activity of the delta-aminolevulinic acid dehydratase enzyme in the second reaction of the heme biosynthetic pathway has been determined in human blood in the presence of varying concentrations of carboxyhemoglobin. In vivo and in vitro carbon monoxide exposure causes a consistent, but small, significant diminution of ac-

tivity. At concentrations of carboxyhemoglobin likely to be found in vivo it is unlikely to significantly influence the use of this enzyme as a measure either of lead exposure or of ethanol consumption.

An X-Ray Fluorescence Technique for in Vivo Determination of Lead Concentration in a Bone Matrix

L. Ahlgren and S. Mattsson. PHYS MED BIOL 24 (1): 136-45, Jan 79.

Authors' abstract: We have previously reported the in vivo detection of lead in the skeleton of man by means of X-ray fluorescence analysis using a 740 MBq ^{57}Co source for excitation and a 1 cm^3 Ge(Li) detector for registration of the Pb K_α and K_β radiation. The varying geometry, density, and atomic composition of the tissues of interest (mainly fingers) introduce several problems in the estimation of the true concentration of a given element. A two-component cylindrical finger phantom was therefore constructed from silica paraffin wax and animal bone ash.

The diameter of the finger bone was estimated from X-ray examinations in two orthogonal projections. The bone mineral concentration was then estimated from the quotient of the number of coherent and Compton scattered primary photons. The lead concentration in the finger bones was then derived from a measurement of a finger phantom made of silica paraffin wax and bone ash with the same size and bone mineral concentration as the real bone. The minimum detectable lead concentration in a finger bone was 14 $\mu g\ g^{-1}$ for 15 minutes measuring time.

The lead concentration measured in workers from a metal industry was found to be in the range of 40-100 $\mu g\ g^{-1}$.

Another Role for Lead?

P. Grasso. FOOD COSMET TOXICOL 17 (1): 86, Feb 79.

Besides its other toxic effects on the central nervous system, lead has been suggested as a possible cause of motor neuron disease. This rare degenerative disorder affects the cranial nerves, motor cells of the spinal cord, and the pyramidal tracts (nerves connecting the brain with spinal cord cells), and causes a progressive weakness and atrophy of the voluntary muscles. Increased lead levels have been reported in the cerebrospinal fluid and the plasma of those struck with this disease; 16 patients had significantly higher plasma-lead levels compared with 18 control subjects, although both were within generally accepted "normal" limits. It is theorized that increased lead in the plasma passes in a retrograde manner from the endplate along the axon to the body of the cell. Such a movement has been demonstrated for horseradish peroxidase, but, because of the difficulty of detecting lead in nerve cells, will be harder to prove with lead.

Metals in Spinal Cord Tissues of Patients Dying of Motor Neuron Disease

H.M. Kurlander and B.M. Patten. ANN NEUROL 6 (1): 21-4, Jul 79.

Authors' abstract: To evaluate the role of toxic metals in causing motor neuron disease (MND), we used a photon-excited, energy-dispersive x-ray analytical system to measure the metal content of spinal ventral horn tissue. Specimens were taken from the cervical and lumbar enlargements of 7 patients who died of MND and the results compared with those found in 12 control patients. Anterior horn lead levels were elevated in MND patients compared to controls (mean, 40.7 $\mu g/gm$ versus 14.6 $\mu g/gm$; $p < 0.05$), and lead levels correlated with the duration of illness ($r = +0.84$, $p < 0.05$). Only 2 MND patients had detectable manganese levels (72.3 and 132.2 $\mu g/gm$) whereas 1 control had detectable manganese (14.3 $\mu g/gm$). One MND patient had 244 $\mu g/gm$ selenium, but 3 controls had levels of 180, 58, and 62. Patients with the histories of greatest environmental exposure to metals during life exhibited the highest tissue levels of metals after death; despite chelation therapy for about a year, high lead levels remained in their tissue.

Erythrocyte Delta-Aminolevulinic Acid Dehydratase Activity and Blcod Protoporphyrin Concentrations as Indices of Lead Exposure and Altered Heme Biosynthesis

P.A. Meredith, M.R. Moore, and A. Goldberg. CLIN SCI MOL MED 56 (1):61-9, Feb 79.

From authors' summary: The activity of erythrocyte delta-aminolevulinic acid (ALA) dehydratase and blood protoporphyrin concentrations have been measured in patients with various anemias, a group of subjects with known lead exposure, and a group of control subjects. Leucocyte ALA synthase was measured in subjects from the last two groups. Erythrocyte ALA dehydratase activity was significantly depressed in the group of lead-exposed subjects and showed a highly negative exponential

relationship with blood lead concentration. Blood protoporphyrin concentrations were significantly elevated in the group of lead-exposed subjects and patients with iron-deficiency anemia and showed a significant positive exponential relationship with blood lead concentration. Comparison of the least-squares regression analysis of these relationships and incidence of false positive and false negative results indicates that erythrocyte ALA dehydratase activity is a more accurate measure of environmental and moderate industrial lead exposure than blood protoporphyrin concentrations. The correlations of erythrocyte ALA dehydratase and leucocyte ALA synthase activity, and of blood protoporphyrin concentrations and leucocyte ALA synthase activity, suggest that blood protoporphyrin more accurately reflects heme synthesis than does erythrocyte ALA dehydratase activity.

Erythrocyte Protoporphyrin Levels in Patients with Freidreich's and Other Ataxias

R.O. Morgan, G. Maglie, D.F. Horrobin, and A. Barbeau. CAN J NEUROL SCI 6 (2):227-32, May 79.

Authors' abstract: Of 13 patients with Friedreich's ataxia (Type Ia) and 17 with type IIa recessive ataxias, all were found to have levels of free erythrocyte protoporphyrin (FEP) above the normal range. The rise in FEP in Friedreich's ataxia correlated well with the age of the individual and thus appears to be related to the course of the disease. Subjects with olivo-ponto-cerebellar atrophy, Charlevoix syndrome, Duchenne muscular dystrophy, and Parkinson's discease were also found to have significantly elevated FEP, although the distribution overlapped with the normal range. The finding of elevated FEP may indicate a relative heme deficiency in ataxia due to inhibition of ferrochelatase leading to a state of ineffective, persistent erythropoiesis. The possibility of a prostaglandin abnormality being related to this defect and to the pathogenesis of ataxia is considered.

Apparent Effect of Ascorbic Acid Medication on Semen Metal Levels

W.A. Harris, T.E. Harden, and E.B. Dawson. FERTIL STERIL 32 (4): 455-9, Oct 79.

Authors' abstract: The apparent effect of ascorbic acid therapy for nonspecific spermagglutination on semen levels of ascorbic acid as well as macro- and micrometals was determined in 20 men (ages 25 to 38). Pretreatment diagnosis was based on infertility and relatively low ratings in sperm density, and motility, motility index, and semen volume and were associated with large numbers of abnormal sperm, sperm precursors, and leukocytes. The pretreatment levels of ascorbic acid, sodium, iron, potassium, zinc, manganese, lead, magnesium, and copper were measured in each patient's semen and compared with levels following 60 days of dietary vitamin C supplementation (1.0 gm/day). Analysis of the vitamin C preparation prescribed revealed that each subject was given an impure ascorbic acid medication to supplement a normal diet. Therefore, the significant increses in levels of ascorbic acid and metals in semen following therapy could not be attributed to ascorbic acid alone, nor, similarly, the improved physical parameters of each subject's semen following therapy; no apparent spermagglutination and restored fertility may be due to the interaction of ascorbic acid with cations found in semen.

Free Erythrocyte Protoporphyrin and Cobalt Excretion Test in Regular Blood Donors

U. Torelli, A. Donelli, P. Zaniol, and O. Bertani HAEMATOLOGICA 63(5): 505-11, Oct 78

Authors' abstract: Free erythrocyte protoporphyrin and cobalt excretion were evaluated in two groups of male and female regular blood donors and in two groups of control subjects. A significant difference in the two parameters was observed between control men and women. No significant change in the free erythrocyte protoporphyrin was induced, either in men or in women, by the donations. On the contrary, a significant increase in the cobalt excretion, suggesting a decrease of iron stores, was observed in donor women.

RESEARCH AND EVALUATION

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*Abstracted

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