DIAGNOSIS AND SCREENING

What Happens in Lead Poisoning?

W.R. Lee. J R COLL PHYSICIANS LOND 1981 Jan;15(1):48-54.

From author's discussion: This article deals with some of the features of clinical lead poisoning. The subject is confusing, because much has been written, particularly in the occupational medicine journals, about the effects of increased lead absorption without the development of clinical symptoms. Quite a small intake of lead may produce detectable biochemical or even neurophysiological changes, and it is difficult, if not impossible, to decide when these changes are simply an adaptation to an increased intake of lead and when they indicate early poisoning. When a patient moves from symptomless "increased lead absorption" to lead poisoning, symptoms generally follow in a certain sequence. Lassitude is the first to appear, often followed by aching in joints and in limb muscles and by abdominal colic. Among biochemical changes is an increased urinary excretion of aminolevulinic acid and of coproporphyrin III. Motor nerve conduction velocity is often reduced, even in symptomless lead workers. The most frequently used diagnostic test for lead poisoning is the measurement of blood lead, which is limited by its poor discriminatory value. The most useful hypothesis for interpreting these results is to assume that the lead level in the circulating blood reflects not only the body burden of lead, but also the affinity for lead of the globin chains.

Laboratory Diagnosis of Lead Poisoning

S. Piomelli and J. Graziano. PEDIATR CLIN NORTH AM 1980;27(4):843-53.

From authors' introduction: Because lead poisoning in childhood is almost always the result of chronic exposure to lead, a laboratory diagnosis can usually be made long before overt clinical disease develops. It is important to detect childhood lead poisoning before it becomes symptomatic, since the neurologic syndrome has an insidious onset and then shows a sudden transition to overt and irreversible clinical intoxication. Additionally, the less obvious neuropsychological consequences that occur at lower levels of exposure can be prevented only if excessive lead intake is detected and the child is removed from the source of lead. Careful neurologic examination and the measurement of the blood lead level should allow early diagnosis. But the more subtle neurologic abnormalities are difficult to detect in any individual child, and, when the neurologic examination is clearly abnormal, it is usually too late to prevent permanent damage. Blood lead level is difficult to determine, expensive, and available only in highly specialized laboratories; moreover, both day-to-day variations in analytical results and environmental contamination of samples may diminish the reliability of the method. For these reasons, the diagnosis of lead poisoning rests, at least at the screening level, on the detection of abnormalities of heme synthesis, for which precise and extremely sensitive microchemical tests are available.

Lead Poisoning in Children

A.H. Drummond, Jr. J SCH HEALTH 1981 Jan;51(1):43-7.

The purpose of this article is to acquaint school health personnel with the dimensions of the lead poisoning problem. In a recent screening conducted by the Centers for Disease Control, some 7,950 of 116,668 children tested showed lead toxicity. In addition to these unsuspected cases, from 12,000 to 16,000 children are treated each year nationwide for lead poisoning; about 200 of these children die. Nearly all the tissues of a child's body are affected by lead poisoning, but the greatest effects are on the brain and nervous system. The early symptoms are often overlooked or misinterpreted. A correlation has been reported between blood lead levels and behavior, progress in learning, and level of ac-

tivity in outwardly normal children. Recent research suggests that there may be no safe threshold level of lead in a child's body below which there are no harmful effects. Several methods are recommended to detect children with lead toxicity and to reduce lead in the environment.

Riposte to "Environmental Lead and Young Children" (Editorial)

F.J. Coodin, C. Dawes, G.W. Dean, P.R. Desjardins, and J.B. Sutherland. CAN MED ASSOC J 1980 Sep 20;123(6):469-71.

In most environments it is difficult to determine the route of intake—inhalation or ingestion—of lead. Airborne lead may be inhaled or may settle on food and be ingested; lead in dirt may be ingested or may be resuspended as dust and inhaled. Children are not only more susceptible than adults to the deleterious effects of lead; but also, because of their physical characteristics and their tendency to put various objects in their mouths, children are likely to ingest more extraneous lead. A problem in the quantitation of lead pollution is the difficulty of accurately measuring blood lead levels. Because blood lead concentrations provide an indication of current or recent levels of exposure only, one investigator suggests measuring lead concentrations in bones or teeth to adequately assess the long-term effects of environmental lead exposure.

Impact of Community Screening on Diagnosis, Treatment, and Medical Findings of Lead Poisoning in Children

J. Schneider, B. Aurori, L. Armenti, and D. Soltanoff. PUBLIC HEALTH REP 1981 Mar-Apr; 96(2):143-9.

From authors' conclusion: Review of the medical records of 525 children diagnosed as lead poisoned during 5 years indicates that a citywide lead poisoning prevention program has resulted in a decrease in the number of children with lead-induced seizures and with central nervous system involvement, as well as a decrease in the mean whole blood lead concentrations (PbB's) in symptomatic children. However, the large number of multiple treatment episodes suggests a serious problem of re-poisoning because lead was not eradicated from the children's environments. The high proportion of children with PbB's of 40 μg/dl or greater indicates that the program has had a limited impact on the incidence of elevated blood lead levels. Findings of this study indicate that, although extensive screening and clinical follow up are effective in preventing the more serious aspects of lead poisoning, they do not address the issue of prevention.

Pica Patterns, Toxocariasis, and Elevated Blood Lead in Children

L.T. Glickman, I.U. Chaudry, J. Costantino, F.B. Clack, R.H. Cypess, and L. Winslow. AM J TROP MED HYG 1981 Jan;30(1):77-80.

Authors' abstract: Blood samples were obtained during a lead screening program from 100 children aged 1-6 years in Allegheny County, Pennsylvania, to determine whether there was any association between specific forms of pica and infection with *Toxocara canis*, the principal cause of visceral larva migrans in the United States, or elevated blood lead levels. Significant associations were found between: (1) feces, soil, or grass pica and *Toxocara* infection; (2) paint or plaster pica and elevated blood lead; and (3) dog ownership and *Toxocara* infection. These findings suggest that an accurate pica history may be useful in identifying potential health problems in children.

The Relationship between Zinc Protoporphyrin (ZPP) and "Free" Erythrocyte Protoporphyrin (FEP) in Lead-Exposed Individuals

V. Karacic, D. Prpic-Majic, and S. Telisman. INT ARCH OCCUP ENVIRON HEALTH 1980; 47(2):165-77.

Authors' abstract: The relationship between zinc protoporphyrin (ZPP) and total erythrocyte protoporphyrin, measured as "free" erythrocyte protoporphyrin (FEP), was determined in 194 adult subjects with different occupational and non-occupational lead exposures. Furthermore, the ZPP-FEP comparison was considered with respect to the dose-effect relationship of ZPP and FEP with blood lead (PbB) for males and females, respectively. Bilirubin (Bil.) interferences in ZPP analysis were taken into account. A very close and highly significant relationship ($r=0.962$, $p < 0.001$) was established between ZPP and FEP values. A significant correlation ($p < 0.001$) between log ZPP or log FEP and PbB (males, $r = 0.767$ and 0.718; females, $r = 0.525$ and 0.405) was also found. It was established, by both in vitro and in vivo studies, that Bil. interferes with the ZPP fluorescence readings; the relationship between "false" positive ZPP concentrations and Bil. concentrations (in vitro, $r = 0.987$; in vivo, $r = 0.903$) was highly significant ($p < 0.001$). A small but highly significant ($r = 0.948$, $p < 0.001$) influence of increased carboxyhemoglobin (COHb) concentrations on the decrease in hematofluorometer ZPP readings, due to inadequate oxygenation of the blood, was found. The results obtained confirm the usefulness of ZPP determinations using hematofluorometers for surveillance of in-

employed. The standard addition method was used to minimize the matrix effects of whole blood. The ASV results correlated well with those obtained by flameless atomic absorption analysis. The methods are simple, reliable, and suitable for applications in the clinical field. The procedure using the Charge Transfer Analyzer is recommended because of its sensitivity and rapidity.

Screening for Lead Absorption

C.R. Corum, L. Garcia, Jr., and J.D. Repko. OCCUP HEALTH SAF 1981 Mar;50(3):8-12.

Recent attention in occupational medicine to the neurological consequences of industrial lead exposure has led to the development of a method of partial antidromic blocking to determine the conduction velocity of the slower nerve fibers of the ulnar nerve. Since the slower conducting fibers of peripheral motor nerves are thought to be susceptible first to damage in neuropathies induced by absorption of lead, this technique appears to be appropriate for detecting subclinical nerve damage in individuals without clinical neurological symptoms. The described technique and equipment for neurological surveillance of workers could provide results conclusive enough to warrant removing an employee from a job.

University Conducts Lead Exposure Study

Anonymous. OCCUP HEALTH SAF 1981 Feb; 50(2):36-7.

The nervous system is particularly vulnerable to the toxic effects of certain heavy metals. In some industries, workers are unwittingly exposed for long periods of time before clinical manifestations are noted. Chronic low-level exposures may go unrecognized. The Department of Neurology at Boston University School of Medicine has detected subclinical damage among industrial workers and schoolchildren exposed to lead and other neurotoxins by using sensitive neurophysiological techniques that measure the speed at which an impulse travels along a nerve. A research team is conducting a case-control study among 112 workers at a lead foundry and a valve manufacturing company, which are located on the same grounds. Previous studies have found adverse physiologic, neurological, and psychological reactions among children and adults who had been exposed to even low doses of lead over a period of time in their homes or industrial environments. For example, behavioral disturbances were eight times more frequent in children exposed to lead than in children in a control group. These studies will contribute to the development of a protocol that could have broad application in the evaluation of occupational exposure to all neurotoxins.

Subclinical Effects of Chronic Increased Lead Absorption — A Prospective Study. III. Neurologic Findings at Followup Examination

G.H. Spivey, R.W. Baloh, C.P. Brown, B.L. Brody, D.S. Campion, J.L. Valentine, D.E. Morgan, and B.D. Culver. JOM 1980 Sep;22(9):607-12.

Authors' abstract: Neurologic examination, nerve conduction testing, and electro-oculographic testing have been performed at a baseline examination and a followup examination in a group of lead workers with blood lead levels predominantly between 60 and 80 μg/dl and in a group of control workers. A statistically significant decreased saccade accuracy measurement in the lead workers compared with the controls was found at both examinations. No other simple test or pattern of findings differentiated between the lead workers and the controls, and the biological significance of the lower saccade accuracy is not clear. Nerve conduction measurements do not appear to be a satisfactory method of detecting subclinical neurologic effects of lead exposure.

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*Abstracted