EPIDEMIOLOGICAL CONSIDERATIONS OF OCCUPATIONAL LEAD EXPOSURE

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A B S T R A C T

Diagnosis of occupational lead poisoning in the adult depends largely upon the presence of overt signs and symptoms and a variety of laboratory tests, some of which have not been clearly related to clinical findings. Cases are presented to illustrate differences in clinical response among lead exposed workers. Some of these cases cast doubt upon the margin of safety provided by the widely recognized blood lead level of 80 µg/100 ml. Generally accepted criteria for the diagnosis of adult lead poisoning are needed as a basis for epidemiological studies that can define the magnitude of today's occupational lead problem.

In the epidemiology of occupational lead exposure, we seek to understand the cause and prevention of lead's detrimental effects on workers. We need to know where in industry these effects occur and with what frequency, what characteristics of the work environment contribute to the disease, and what characteristics of the exposed worker contribute to an adverse response to lead exposure.

Despite the fact that lead poisoning was studied by the ancients and its symptoms and cause described by Hippocrates or the accumulation of a voluminous literature on the subject, our presence here at this conference gives testimony that our knowledge remains imperfect. Further testimony is found in the fact that we cannot come to agreement on the standards for worker protection and is further found in the observation that occupational lead poisoning is still a major problem today.

In some ways, such ignorance today seems more profound that it did during the early decades of this century. Alice Hamilton[1] reported information on the frequency of lead poisoning in various industries. Based upon calculations of her data given in Table I, it appears that frequency per million man hours worked ranged from 45 for printers to 220 for rubber manufacturers. This latter figure is equivalent to one case of lead poisoning for every two workers exposed. Today our information on the incidence of occupational lead poisoning is probably less precise than that given us for the period 1911-1916 by Dr. Hamilton.

In recent years, it has been customary to look to the statistics collected by the California Department of Health for information on the occurrence of occupational disease. The data collection system for occupational disease in California is often pointed to as being unique in

TABLE I

FREQUENCY OF LEAD POISONING
(Period 1911-1916)

Trade	Frequency/10^6 Man Hours
Battery Manufacturing	90
Casting	8.6
Pasting	97.0
Oxide mixing	200.0
Enameling	182
Lead Smelting	118
Pottery Manufacturing	67
Printing	45
Rubber Manufacturing	200

that doctors' reports are required by the State of California and payment by Workmen's Compensation insurance carriers depends upon receipt of the doctors' report. This system, however, appears to have broken down, at least so far as lead poisoning is concerned.

Many cases of lead poisoning are referred directly to a specialist knowledgeable in the diagnosis and treatment of lead poisoning by plant health care personnel rather than to industrial physicians who are accustomed to treating and reporting the usual injuries and ills of industrial patients. Instead of filling out the standard Doctor's First Report of Injury form, which serves as the basis for statistical analysis by the State Health Department, the specialist writes a narrative report to the insurance carrier; thus, California reported only 79 cases in 1970, 82 in 1971, 101 in 1972, and 81 in 1973. Although these totals may be low, if errors in reporting are uniform throughout industry, the percentage distribution of cases among lead using industries may be fairly accurate.

An analysis of the California data shows that 61 percent of cases resulted from battery manufacturing, 7 percent from construction and building wrecking, 5 percent from primary metal industries, 4 percent from machinery manufacturing, 3 percent from the fabrication of metal parts, 3 percent from the wholesale trade, including scrap metal, and 3 percent from workers in state and local governments. During the 4-year period, less than 1 percent of the cases reported were from the ceramic industry and less than 1 percent of the cases were from mining.

Without data about the number of workers at risk, we have no information on incidence to compare with Dr. Hamilton's. Reliable epidemiological information also depends upon uniformity of diagnosis.

Criteria for the diagnosis of lead poisoning were rather clearly set forth in the early 1920's; today's criteria are vague. Newmann *et al*[2] at that time set forth signs and symptoms in two groups, see Table II.

TABLE II

SIGNS AND SYMPTOMS FOR LEAD POISONING

SYSTEM	SIGNS AND SYMPTOMS	
	Group A	Group B
General Appearance	Marked Pallor and Profound Anemia	Pallor, Anemia, Emaciation, and Drawn Expression
Digestive System	Colic Obstinate Constipation	Loss of Appetite or Repugnance to Food Breakfast Anorexia Vomiting on Eating Solid Food Sweetish or Metallic Taste Gastric Disturbances Constipation Pain in Abdomen Parotitis
Muscular System	Muscular Incoordination	Loss of Strength Malaise and Tiring Easily
Nervous System	Peripheral Motor Paralysis of Certain Extensor Muscles Wrist & Ankle Drop Atrophy of Most Used Set of Muscles	Headache, Insomnia Mental Lethargy, Tremor Dizziness, Convulsions Mental Affections, Arteritis Encephalopathic Conditions
Vascular System	Blood-Basophilic Degeneration with Diminished Hemoglobin	Arteriosclerosis Hypertension
Special Organs and Findings	Gums: Lead Line Stools and Urine-Lead Miscarriages-Repeated Liebermann's Test-Positive	Eyes-Impairment of Vision Muscular incoordination Joints-Various Pains Blood-Basophilic Degeneration with Diminished Hemoglobin

Adapted from Newmann et al, 1921

The Group A signs are considered major manifestations, and in Group B, more generalized, less specific signs and symptoms.

In the author's opinion, the diagnosis of lead poisoning requires signs from at least two systems of Group A and several symptoms from Group B, in addition to a clear history of exposure; or alternatively, when only one

Group A sign is present, signs and symptoms from at least two separate divisions of Group B must be present. When manifestations from only Group B exist, they must be from three separate divisions in order to make a tentative diagnosis of lead poisoning. Aub[3] stated in 1926 that the development of distinct toxic symptoms after exposure is necessary for definite diagnosis.

The confidence with which physicians were able to make diagnoses of lead poisoning during the first decade of this century was in part facilitated by higher exposures than exist widely today and perhaps in part by the absence of readily available laboratory measures of lead absorption and enzyme inhibition. The fact that such laboratory tests do exist today has in many cases led physicians to confuse increased lead absorption with lead poisoning. An attempt to clarify the confusion between increased absorption and poisoning was made by an international group of 18 physicians with broad experience in the lead industries.[4] Their statement emphasizes the use of clinical findings together with biochemical evidence of increased absorption and, if possible, evidence of unusual exposure. Also, emphasized is the fact that the clinical findings in lead poisoning are symptoms and signs of other conditions and, thus, such other causes must be excluded.

As laboratory tests become more widely available to industry, there is, appropriately, greater attention being focused upon biochemical response and less demand upon the presence of a constellation of signs and symptoms. As a result, diagnoses of lead poisoning are being made at an earlier point in the natural history of the disease. The trend today is to make a positive diagnosis, even if only a few minor symptoms compatible with lead poisoning are present, so long as indicators of increased absorption and indicators of biological response are present. This trend may have an impact on the choice of standards for the protection of lead workers.

As an illustration of this point, the case of V.L., with three years of employment in the lead acid battery industry is reviewed. His blood lead values have always been well below the 80 µg/100 ml level, considered by many to be the lower limit of excessive absorption. As can be seen in Figure 1, hemoglobin levels, except for two episodes, have been above 14 gms. Early in 1973, we added urinary ALA to our surveillance program. Concern for elevated ALA values led us to require respirator protection, even though time weighted exposure to airborne lead was well below 0.2 µg/cu m. In October of 1973 the patient reported to the plant nurse that he was having mild constipation and feeling increasing fatigue. There were no other findings on system review or physical examination, urinary coproporphyrins were 1355 µg/liter and urinary ALA was 5.2 mg/100ml. This case was diagnosed as being one of early lead poisoning. Because respirator protection had been unsuccessful, he was transferred to work which did not involve exposure to lead with a resultant gradual drop in blood lead levels and a marked decrease in his excretion of ALA and a

disappearance of symptoms. Returned to his regular work on the paste line in February, 1974, has not resulted in an increase in blood lead but his excretion of ALA tells us that he may be headed for trouble again.

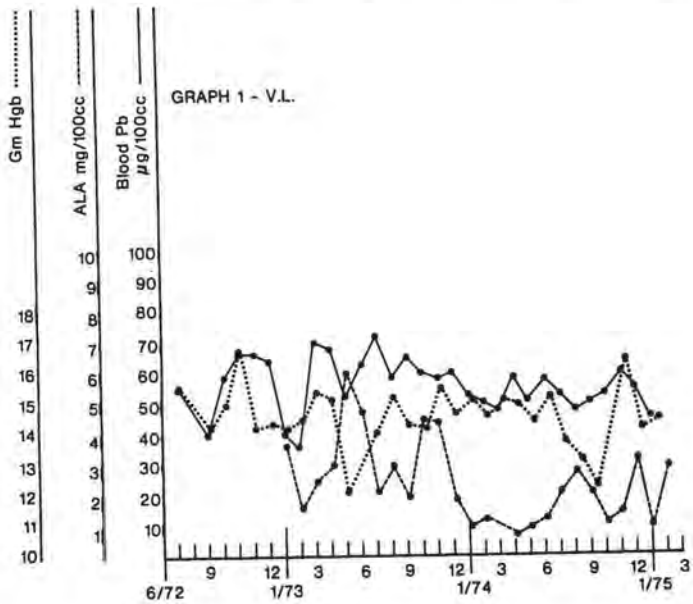

Figure 1 Graph of Laboratory Results for Worker V.L.

A somewhat different response in a worker exposed to lead is seen in the case of P.M. See Figure 2.

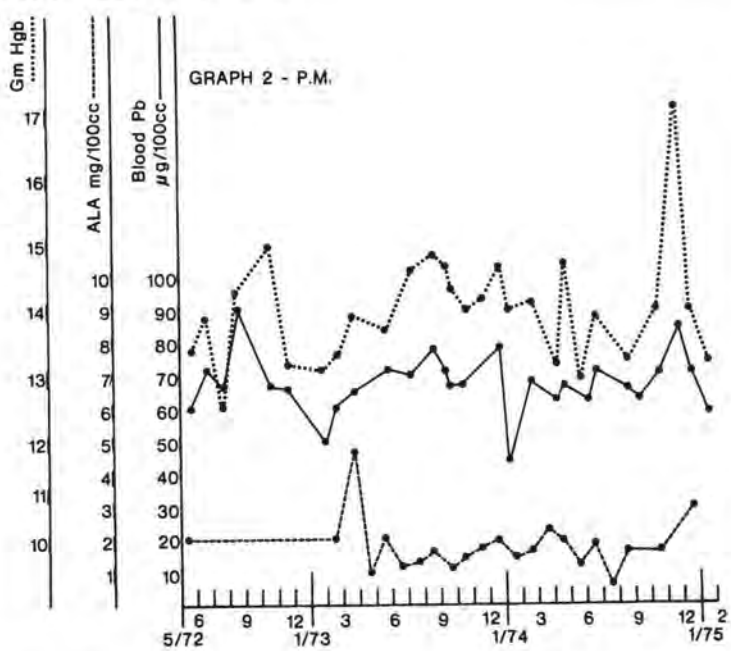

Figure 2 Graph of Laboratory Results for Worker P.M.

His blood lead levels hovered around the 70 g/100 ml level with some peaking above 80 g. In contrast to the consistently higher blood lead seen in the preceding case, excretion of ALA in this worker has generally been much lower. At no time during our period of observation have we been able to elicit symptoms or signs compatible with lead poisoning.

The case of F. L. provides support to the impression that monitoring of blood lead provides little insight into the biological activity of lead. Fig 3 shows pre-exposure base-line blood lead levels of 17 g/100 ml. With the onset of employment involving lead exposure, blood lead rose over a 5-month period to 53 g/ml. This is not a precipitious rise for lead workers and from this point, there is a gradual decrease in blood lead levels to 40 g/100 ml. The reason for this decrease was rigid enforcement of respiratory protection and personal hygiene prompted by evidence that this case responded markedly to absorbed lead. This response can be seen in part in the plot of hemoglobin which shows a precipitous drop from pre-exposure baseline levels of 15 gms to 10.3 gms in the initial 5-months of exposure. The gradual climb to near baseline levels may in part be the result of better control of exposure and in part, possibly, by reduced biological response.

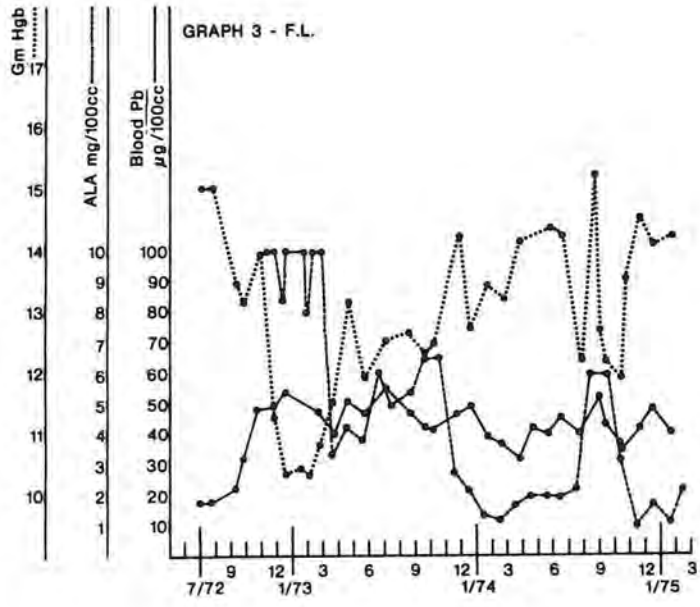

Figure 3 Graph of Laboratory Results for Worker F.L.

A further indication of marked biological response in the face of only moderate blood lead levels can be seen in the urinary delta amino-levulinic acid levels. On this graph initial values are shown with a maximum of 10 mg/100 ml. In reality these initial values may have been considerably in excess of this number but the analytical method used without an extra dilution step is unreliable above 10 mg. When looking at the ALA values for this case, it should be kept in mind that acceptable levels for lead workers have a maximum of 2 mg/100ml and values above this are considered excessive.[4] With few exceptions, the values shown here are well in excess of 2 mg, probably indicating high reactivity to lead absorption. Other causes of anemia and high ALA excretion were sought in this case and excluded. At no time have we been able to elicit clinical signs or symptoms of lead poisoning in this worker.

An evaluation by Malcolm[5] of epidemiological data of workers in the storage battery industry poses the question: are the standards of human safety now employed in the lead industries capable of eliminating the effects of absorption of small amounts of lead on the general health, the susceptibility to other disease, andthe length of lives of exposed workmen? He states that in this study of workers who, since 1930, have generally not been exposed to air lead concentrations in excess of 0.15 mg/cu m, he is unable to answer the question with assurance.

Today, as we observe enzyme responses to lead at levels consistent with exposure below 0.15 mg/cu m, we need to undertake prospective epidemiological studies using methods available to us to probe for lead related damage in order to answer Malcolm's questions. As indicated in this morning's session, it is unlikely that the enzymes of heme synthesis are the only enzymes in man which are affected by lead exposure.

We are, thus, justified in making diagnoses of lead poisoning on the basis of mild, vague symptoms when accompanied by laboratory evidence of biological response to lead. Further, it is in the best interest of both management and labor that early manifestations be recognized. The feelings of fatigue, vague physical discomfort, anxiety and fear of the unknown can lead to accidents and greatly reduce productivity. These are sufficient reasons, even if chronic pathologic changes do not occur at low levels of lead exposure.

Thus, I recommend the establishment of criteria for the diagnosis of lead poisoning at the earliest point in the natural history of lead poisoning. Widely accepted criteria will allow the epidemiologist to define for us the magnitude of today's lead problem.

REFERENCES

1. Hamilton, A., Section in Lead Poisoning, Medicine IV, by Aub, J.C., Williams and Wilkins, Baltimore, 1926

2. Newmann, B. J., *et al*, Lead Poisoning in the Pottery Trades, Public Health Bulletin No. 116, Govt. Prngt. Office, Washington, D.C., p223, 1921

3. Aub, J. C., Fairhall, L. T., Minot, A. S., and Reznikoff, P., Lead Poisoning, Medicine, IV, Baltimore, Md., 1926.

4. Diagnosis of inorganic lead poisoning, A statement, British Medical Journal, 23:501, Nov, 1968

5. Malcolm, D., Prevention of long term sequelae following the absorption of lead, Arch Environ Hlth, 23:292-298, 1971

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HEALTH EFFECTS OF *OCCUPATIONAL LEAD and ARSENIC EXPOSURE* A SYMPOSIUM

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A SYMPOSIUM

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