III. BIOLOGIC EFFECTS OF EXPOSURE

Extent of Exposure

In excess of a million tons of lead are processed yearly. The total usage of lead has remained relatively stable during recent years, but the consumption by various industries has changed. For example, there has been a decrease of lead usage in the manufacture of house paints and a simultaneous increase in the manufacture of lead storage batteries. The particular properties of lead (Table X-1[1]) have made it useful for many applications.

Scrutiny of Table X-2 (from U. S. Bureau of Mines[2]) gives an idea as to the relative proportion of lead usage for various industries. Metal products and miscellaneous categories account for the bulk of lead consumption. The refining and processing necessary to form these products include heating, grinding, and volatilization and therefore produce potentially hazardous industrial atmospheres. The impression should not be left that all workers in these industries are jeopardized, but rather that such uses of lead places them at risk of lead absorption.

Table X-3 (Gafafer[3]) lists specific occupations and trades where lead exposure occurs. The diversity of occupations displayed in these tables shows why a precise measure of the extent of lead exposure is non-existent. The National Academy of Sciences' recently published document on lead[4] agrees, stating, "A reliable definition of the extent of risk of occupational lead exposure is unavailable." Because of the changing usage of lead in industry and the widely varied trades where exposure occurs, the United States has no reporting system whereby the prevalence of occupational lead poisoning can be analyzed.

Consider Table X-4[5] which gives examples of general exposure from industrial operations utilizing lead. Simultaneous examination of these tables should give at least a general overview of the extent of occupational exposure to lead. Specific levels for operations within lead-using industries are presented in Part IV, Environmental Data.

Historical

Lead has been used for thousands of years because of its availability and desirable properties. Its low melting point (327 C), ductility, malleability, and weathering resistance enabled its use without the need for the more complex equipment that, in modern times, has enabled the use of other metals such as steel that have more desirable properties for many applications.

In the 1800's, there was an increasing recognition of hazards to health associated with lead. It was found that lead could be absorbed by inhalation and ingestion, and that lead absorption was responsible for loss of movement in printers' fingers exposed to heated lead type and for "dry grippes" in pottery and glass workers. In 1839, Tanquerel des Planches[6] published a treatise on lead diseases, to which Dana later added notes on the effects of using lead pipes. Progress in recognizing signs of lead absorption was made during the 19th Century also. Burton,[7] described in 1840 the "Burtonian Line", a blue line on the gums, as a sign of lead absorption, and chemical methods for detection of lead in blood or urine were developed.

The prevalence of lead poisoning in ancient times is speculated upon, and it has been suggested that Rome fell because of the prevalence of lead

poisoning (plumbism) in its citizens. It seems likely that, with the ignorance that existed on the hazards of lead and on methods of limiting exposure, there was a significant incidence of plumbism until its recognition in recent times generated preventive procedures.

Effects on Humans

A description of effects of lead absorption can be graphic if based on effects seen in industries earlier in this century. Thus, Mayers[8] can describe effects of lead poisoning, from studies of many years ago, such as loss of appetite, metallic taste in the mouth, constipation and obstipation, anemia, pallor, malaise, weakness, insomnia, headache, nervous irritability, muscle and joint pains, fine tremors, encephalopathy, and colic. In lead colic, there may be severe abdominal pain, such that abdominal surgery has occasionally been performed. In workers, as pointed out by Mayers,[8] who have had repeated attacks of lead colic over many years, there is a tendency towards the occurrence of weakness of extensor muscle groups. This weakness may progress to palsy, often observed as a characteristic "wrist drop" or "foot drop."

The important routes of absorption of lead by man and animals are ingestion and inhalation. Eating of lead-bearing paint by children and drinking of lead-contaminated, illicitly distilled whiskey are important sources of non-industrial poisoning. Other sources include exposure to buring battery casings, drinking of liquids from improperly fired, lead-glazed containers, and high levels of airborne lead. But man absorbs lead in small amounts not normally leading to poisoning from his food and water, and from the air. These sources lead to the "normal" body burden

of lead. Thus, the lead absorbed in the course of occupational exposure is superimposed on lead abosrbed from other means.

Descriptions of lead poisoning appear in many texts and reviews, for example Airborne Lead in Perspective, a report of the National Academy of Sciences,[4] and The Diseases of Occupations by Hunter.[9] The rest of this section pertains to the occupational aspects of lead poisoning, with a few notes on effects seen only in children.

Lead can interfere with the synthesis of heme, thereby altering the urinary or blood concentration of enzymes and intermediates in heme synthesis or their derivatives. Thus, lead poisoning can lead to accumulation of non-heme iron and protoporphyrin-9 in red blood cells, an increase in delta-aminolevulinic acid (ALA) in blood and urine, an increase in urinary coproporphyrin, uroporphyrin, and porphobilinogen, inhibition of blood ALA-dehydratase (ALA-D), and an increased proportion of immature red cells in the blood (reticulocytes and basophilic stippled cells).

Anemia from lead poisoning is associated with a reduced red cell life span and with reticulocytosis and basophilic stippled cells in peripheral blood. Symptoms of this anemia include irritability, fatigue, pallor, and sallow complexion. Bone marrow preparations show increased numbers of sideroblasts, and this is useful in differential diagnosis of lead poisoning from iron deficiency anemia.

Gastrointestinal sequelae of lead poisoning include intestinal colic, nausea often without vomiting, and constipation (or, occasionally, diarrhea). Headache usually occurs before or about the time of onset of colic.

Peripheral and central nervous system effects occur in severe poisoning. Peripheral neuropathy of lead poisoning involves considerable loss of motor function but little loss of sensory function. Extensor muscles of the hand and feet are often involved; extensor weakness normally precedes wrist drop or palsy.

Encephalopathy may be either acute or chronic. Acute encephalopathy may follow ingestion or inhalation of large amounts of lead, and may develop quickly to seizures, coma, and death from cardiorespiratory arrest. Chronic encephalopathy usually occurs in children after excessive ingestion of lead, and leads to loss of motor skills and of speech, and to development of behavioral disorders. Lead encephalopathy, often involving psychosis, also occurs from absorption of alkyl lead compounds.

Nephropathy is another effect of lead poisoning. There may be a progressive and irreversible loss of kidney function, with progressive azotemia, and occasionally hyperuricemia with or without gout. Children have developed renal dwarfism, hypertension, marked retention of urea, and low urinary concentration; some children with acute encephalopathy have developed a form of Fanconi syndrome, a kidney disease indicative of severe injury of the promixal renal tubules. Nephritis in adults is not common, but ischemic nephritis may occur after prolonged absorption of lead.

Epidemiologic Studies

Lane[10] examined the causes of death of storage battery workers, including retired workers, and compared data from this group with data from all English and Welsh males of similar ages during the same period of time. Among the retirees who had been exposed to lead, there were found to be greater numbers of deaths than would have been expected, for their ages, from data on the population as a whole. Most of this excess in expected mortality was accounted for by vascular lesions in the central nervous system. Lead workers who died during employment also showed an excess of deaths from this cause.

Another study of electric storage battery workers was conducted by the Public Health Service over 30 years ago.[11] In this study, the incidence of various disease states was studied in relation to lead exposure of 766 workers, most of whom (75%) had worked in storage battery plants for more than five years and some of whom (12%) had worked there for twenty years or more. The incidence of disease (other than plumbism) in men exposed at levels of 0.15 mg/m^3 and higher (high exposure group) was compared to the incidence in men exposed below 0.15 mg/m^3 (low exposure group). Special attention was given to cardiovascular disease because of the common belief that chronic plumbism results in arteriosclerosis; however, the data developed by the PHS team did not show that more severe exposure to lead is associated with a significantly higher incidence of vascular disease. The incidence of arteriosclerotic-hypertensive disease was not significantly different in the high and low exposure groups. The responses to a standard

exercise, in terms of return to pre-exercise pulse rates and to systolic and diastolic blood pressure, were also compared, and again the two groups were found not to be significantly different from each other. These lead workers were also found not to be significantly different from other, non-lead, workers in terms of blood pressure. From this, it was concluded that exposure to lead in the storage battery industry does not cause cardiovascular effects.

A contrary conclusion was reached by Dingwall-Fordyce and Lane[12] in a study of British battery workers. A significant excess of deaths from cerebrovascular accidents was found in pensioners who had had exposure to lead of sufficient degree to have caused mean urinary lead levels of 0.25 mg/liter during many years of lead work. They compared three groups of workers--those with no occupational lead exposure, those with negligible exposure, and those occupationally exposed to lead*--with the general population of English and Welsh males of similar ages. They found a significant excess of death, over that predictable from the population at large, among retirees in the highest exposure group, and this was largely attributable to cerebrovascular accidents. They also examined records of deaths due to cancer in lead workers, both employed and retired, and concluded that there was no association between malignant disease and lead absorption. While they found an excess of deaths from cancer in the negligible exposure group (in the last decade of the 35-year figures only), there was

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*Urinary lead levels in this group averaged between 0.10 and 0.25 mg/liter for a 20-year period.

a slight decrease in deaths, from that expected from statistics on the whole population, among workers absorbing more than negligible amounts of lead, hence their conclusion that malignant disease is not related to lead absorption. As improved working conditions decreased lead exposure, the excess of cerebrovascular deaths diminished.

Malcolm[13] recently conducted similar investigations of past and present employees exposed to lead. Since 1927, airborne lead to which these men had been exposed had been limited to 0.15 mg/m^3, according to Malcolm. He divided the workers into three groups: (A) no exposure, (B) mild exposure, and (C) severe exposure. Average blood lead* in group (C) workers, since 1961, has been 0.065 mg/100 g, from which it may be inferred that the 0.15 mg/m^3 air concentration was sometimes exceeded. Urinary leads in subgroups averaged 0.09 to 0.180 mg/liter, and averaged 0.119 mg/liter for the entire group of workers.

Based on comparison of blood pressures of the two exposed groups (B and C) with the control group (A), it was concluded that there was no occupationally induced hypertension (although there might have been lead-induced hypertension before improved hygienic measures were instituted). There was a non-significant increase in chest disease among older retired workers, attributed to other causes, since most of these pensioners lived in an urban area with a higher rate of death from chest disease than that in the country as a whole.

*Concentrations of lead in blood are expressed as weight units (such as mg) per 100 ml or 100 g of whole blood. European workers more commonly express blood lead as weight units per 100 ml of blood, while American workers more commonly express blood lead as weight units per 100 g of blood. This document will follow the American custom except in referring to studies reporting blood lead in weight units per 100 ml. The difference between the two expressions is small, about 5% or less. Thus, a blood lead concentration of 0.080 mg/100 g would be equivalent to about 0.084 mg/100 ml.

Unlike the findings of earlier investigators (Dingwall-Fordyce and Lane,[12] and Lane[10]) Malcolm found no evidence of increased frequency of cerebrovascular death in his study, which included deaths occurring between 1963 and 1967, while data from the two earlier reports included deaths from 1926 to 1960. Thus, if all three reports are correct in their conclusions, it would seem that improving hygiene has diminished lead-induced cerebrovascular disease.

For years, chronic nephritis was thought to be a consequence of plumbism, and an analysis of death rates in the U.K. in 1921[10] and in 1931[13] shows a considerable excess in plumbers and painters due to nephritis and to cerebrovascular disease. The question of nephropathy from lead has also been raised by Henderson and Inglis,[14] who showed a relationship between chronic nephritis and excessive lead absorption as indicated by elevated lead levels in bone.

Lane[15] described 9 deaths from renal failure in lead workers, men who had been exposed for long periods at lead concentrations around 0.5 mg/m^3. Terminally, they all had evidence of chronic azotemic nephritis. These men, all of whom worked in storage battery industries for over 20 years, died between the ages of 42 and 52 (average age at death was 48.4). Other than two episodes of colic, there had been no previous history of lead intoxication.

In the United States, there have been few reports of renal disease in lead workers,[13] though the PHS survey of storage battery workers discovered an increased incidence of albuminuria in affected workers.

Animal Toxicity

Unlike toxicologic studies of many industrial substances, experimental animal studies of either inorganic or organic lead have contributed far less to an understanding of the toxicology of lead and its compounds than studies on man, and hence have directly contributed very little to the criteria for the standard for lead. The reason is that until recently, much of the investigative effort was directed to the effects of lead on the red blood cell, its urinary intermediates and lead content of blood and urine, all readily investigated in man. Moreover, many of the studies in man or animals relate to detecting changes in biologic constituents of the blood and urine, and hence are relevant more to criteria for biologic standards than to air standards. Thus, the experimental studies discussed herein will be confined to those that confirm or extend the findings in man in these areas and which are related, even if only indirectly, to the criteria for the air standard.

In recent years, research investigations have broadened to include biologic systems other than the erythropoietic, and in this way may ultimately provide new criteria for standards. Lead intoxication has been studied for its effects on the rat thyroid, comparative changes in kidneys of rat and man, and the effect of certain trace metal deficiencies on the toxicity of lead. But only a beginning has been made in our understanding of the action of lead on the nervous system; behavioral effects have been studied in rats following exposure to tetraethyl lead after the finding of marked metabolic changes in the brain from its administration.

a. Experimental Animal Toxicology. The USPHS-sponsored conference on environmental lead[16] in 1965, although oriented towards the community

environment, marked a turning point in experimental animal investigations on lead. Up to this time, animal studies relating to standards criteria used hematologic disturbances for the most part as a focal point of investigations because of their practical usefulness as criteria for judging harmful exposures to lead.

b. Biosynthesis of Heme. Following the first evidence by Rimington[17,18] that lead interfered with the incorporation of iron into the protoporphyrin molecule, and the subsequent demonstration by Eriksen[19] and others that lead also interfered with an early step in heme synthesis catalyzed by delta-amino-levulinic acid dehydratase (ALA-D), Kreimer-Birnbaum and Grinstein[20] confirmed in rabbits poisoned by lead the earlier findings of Eriksen and others. As determination of ALA-D in the red blood cell became recognized as the most sensitive criterion of response to lead exposure yet discovered, it was applied to the control of lead exposures among industrial workers. It was soon suspected, however, when red cell ALA-D was markedly inhibited in the absence of subjective symptoms of lead poisoning and at blood levels within currently accepted normal limits[21-23] that, as a criterion for overexposure of lead workers, ALA-D was of less value than had been anticipated. Studies in dogs[23] confirmed this suspicion; dogs that had been given lead acetate for a period (46 weeks) until their red cell ALA-D was nearly or completely inhibited and were bled to a reduction of from 30 to 40% in hemoglobin, red cell count and hematocrit ratio, recovered to normal hematologic values as well as did controls not treated with lead. Thus, animal studies resolved the important issue of the relative usefulness of the measure, reduction in red cell ALA-D, as an indicator of response to lead exposure, and hence as a criterion for a lead standard, albeit a criterion only indirectly related to an air standard; measurement of changes in ALA-D is too sensitive

to be usefully applied to workers exposed to lead at this stage of knowledge.*

c. Other Animal Studies on Hematologic Effects of Lead. In addition to the inhibitory effects of lead on the biosynthesis of heme, animal studies have included 1) the stimulation of erythropoietic activity[24]; 2) increased rate of basophilic stippling[15]; 3) reticulocytosis[25]; 4) concentration of coproporphyrins in urine and certain tissues[26]; and 5) the effect of lead on iron metabolism in hemoglobin formation.[27]

d. Serum Protein Changes. Changes in the patterns of the proteins in human blood serum, consisting of a decrease in albumin-globulin ratio with marked increases in the alpha- and beta-globulins, have been confirmed in animals.[28]

Similar confirmation has been made in animals of the findings in man of reduced quantities of mucoid and sialic acid, prosthetic groups of conjugated proteins,[29] reductions of which were used as a warning of impending lead poisoning in industry. Unfortunately, other common conditions such as inflammation also cause changes in the amounts of these blood constituents.

A distinct relationship has been found between lead poisoning and the metabolism of nicotinic acid[30]; animals poisoned by lead showed a marked decrease in the nicotinic acid content of blood (and urine), indicating an increased utilization of this constituent by lead, and suggesting that lead exerts serious effects on the pyridine nucleotides, either by blocking

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*This is not to detract from the major recommendation of the PHS conference on lead[16] to search for ever-more sensitive indicators of response, because much of value on the mechanism of lead in the biosynthesis of heme has resulted, but it does clearly point out 1) that ultra-sensitive methods may not always have practical utility in estimating and controlling workers exposure, and 2) that, inasmuch as highly sensitive methods are used as the criteria for many, if not most, of the air standards in the U.S.S.R., these standards must be carefully reexamined in the light of their appropriateness and suitability.

their synthesis or by accelerating the degradation of nicotinic acid. These changes have been suggested as a means of assessing the severity of lead poisoning.

In line with the general opinion that toxic substances adversely affect the body's resistance to disease by interfering with natural immunologic processes, Fonzi et al.[31] showed that lead-treated and actively immunized animals developed lesser amounts of gamma globulin than did immunized controls. Similarly, lysozyme, another part of the defense mechanisms of the body, was progressively reduced in the blood serum of dogs administered lead salts for a prolonged period.[32]

Although shifts in the body's inorganic elements (copper, calcium phosphorus, sodium and potassium[33]) from lead poisoning have been reported,[34] their significance in over-all body metabolism is yet to be clearly demonstrated.

e. Endocrine Changes. The effects of lead exposure on some aspects of endocrine function have been studied in animals, as well as in man. The excretion of steroids was studied in the urine under different conditions of lead exposure in the hope of finding some evidence of their relation to lead absorption. Adrenal steroids were reported at first to decrease, then to increase considerably during advanced stages of lead intoxication.[35] Vitamin C content of the adrenal gland was decreased in the guinea pig following exposure to lead.[36]

Relatively little use has been made of animals in the study of other endocrine functions, these functions being readily studied in man. Sandstead[37] has, however, reported that lead, like other heavy metals, impairs the uptake of iodine by the thyroid, and that the conversion of iodine to protein-bound iodine is retarded; females were more affected than males.

f. Renal Changes. Goyer[38] has recently reviewed the current state of knowledge of the effects of lead on the kidney; his review is based in large part on his investigations and those of his associates. Prominent among their findings of acute lead poisoning in animals were 1) formation of intranuclear inclusion bodies, 2) mitochondrial swelling with impairment of oxidative and phosphorylative processes, and 3) aminoaciduria (apart from the long-recognized delta-aminolevulinic aciduria); the intranuclear inclusion bodies were a lead-protein complex that may have adaptive function in excessive lead exposure. The acute renal changes progress to a diffuse nephropathy with tubular atrophy and dilation. Rats developed hyperuricemia and in chronic lead poisoning, renal adenocarcinoma. In all but the last, the findings made in rats paralleled those seen in man.

g. Trace Metal Interactions. In recognition that lead poisoning is often associated with an iron-deficiency anemia, the interaction of lead on iron deficiency was studied in the rat.[39] An enhancement of lead retention and toxicity was found in the iron-deficient animals as measured by elevated ALA excretion.

h. Effects on the Nervous System. Despite the fact that the nervous system can be affected by lead, comparatively little experimental attention has been directed to gaining an understanding of the manner in which lead acts on this system. Behavioral response studies in animals, predominantly by Soviet scientists, comprise most of the research effort, although of late, Xintaras and associates have initiated investigations in behavioral toxicology.

Using a range of atmospheric concentrations of lead oxide dust, Gusev[40] found that at a level of 11 $\mu g/m^3$ disturbed reflexes began to occur at 1.5

to 2 months of exposure, whereas no impairment of reflexes was seen at levels averaging about 1 $\mu g/m^3$; base-line conditioned reflex activity returned 10 to 23 days after cessation of exposure. Although no changes in the formed elements of the blood were seen, despite a lead content in rat bone of 10-fold higher than that of animals on the lower dose, histopathologic changes in the central nervous system were seen in both rats and rabbits at the 11 $\mu g/m^3$ level.

Shalamberidze[41] reported disturbed conditioned reflexes in rats exposed to lead sulfide ore dust at a level averaging 48 μg Pb/m^3, 6 hours daily for 6 months. However, because of the insolubility of the sulfide supported by health experience with lead sulfide, responses to lead sulfide at that level are unlikely.

Xintaras studied the applicability of evoked response in the rat's cortex in air pollution toxicology[42,43]; in rats intoxicated with lead acetate he found electroencephalographic changes similar to changes in man.[43] From studies of alterations in rapid eye movements during sleep, he concluded that lead may cause impaired neural control in rats.[43]

i. Developmental Effects. Although mice nursing on dams fed diets containing high levels (1% or 4%) of lead carbonate showed evidence of faulty growth and various neurologic changes,[44] recent evidence reveals a low degree of teratogenic effects in rats and mice.[45]

Correlation of Exposure and Effect

Tsuchiya and Harashima[46] studied storage battery workers and compared airborne lead with urinary lead, urinary coproporphyrin, basophilic stippling of erythrocytes, and specific gravity of blood as indications of anemia.

To control urinary coproporphyrin to normal levels (below 50 μg/liter), they recommended a TLV of about 0.12 mg/m^3 for daily 8- to 10-hour exposures. However, the workers studied by these investigators worked 48 to 60 hours a week. With the increased lead absorption from these working hours, a lower standard than that suitable for a 40-hour week would be indicated. If other criteria were chosen on which to base an air limit, other limits would have been selected; 0.10 mg/m^3 would have been recommended to keep urinary lead levels below 0.15 mg/liter, 0.14 mg/m^3 to keep basophilic stippling at 0.3 per thousand, and 0.14 to 0.15 mg/m^3 to prevent anemia. They did not use blood lead as a criterion of effect.

The study of Williams, King, and Walford[47] was based on observations of storage battery workers who worked a 40-hour week, and were stable in their exposure. They had worked without job change for a year, there was no recent absence for sickness or vacation, and no change in overtime or productivity for 6 months.

Workers in the plastics department were exposed to airborne lead levels of about 0.01 mg/m^3, while workers in lead handling departments were exposed to higher levels, up to about 0.3 mg/m^3. Specific gravities of urine samples averaged 1.020 in the morning and 1.022 at lunch time. They concluded that air levels of 0.20 or 0.15 would result in the blood and urinary lead levels given in Table X-5 (urinary lead levels were corrected for a specific gravity of 1.024; it should be noted that a urinary level of 0.20 mg/liter corrected to a specific gravity of 1.024 would be 0.133 mg/liter corrected to 1.016).

These investigators also showed a very low correlation ($r = 0.09$) between airborne lead and blood hemoglobin levels.

Selander and Cramer[48] compared blood lead, urinary lead, and urinary ALA in lead workers. They found several workers with high urinary lead and ALA values in relation to blood lead and attributed this to a metabolic influence of lead; ALA excretion in these workers had seldom fallen to normal values. They recommended that workers removed from lead overexposure not be allowed to return until ALA excretion was normal.

A statement by a group of experts (R.E. Lane, D. Hunter, D. Malcolm, M.K. Williams, T.G.F. Hudson, R.C. Browne, R.I. McCallum, A.R. Thompson, A.J. deKretser, R.L. Zielhuis, K. Cramer, P.S.I. Barry, A. Goldberg, T. Beritic, E.C. Vigliani, R. Truhaut, R.A. Kehoe, and E. King)[49] on diagnosis of inorganic lead poisoning suggests ranges of indices of lead absorption for occupationally acceptable exposures with the following upper limits:

Blood lead: 0.08 mg/100 ml
Urinary lead: 0.15 mg/liter
Urinary coproporphyrin: 0.50 mg/liter
Urinary ALA: 20 mg/liter

They point out that these values may not be applicable when there are low hemoglobin levels or where chelating agents have been used.

Stankovic[50] reported on blood and urine lead concentrations, urinary coproporphyrin, and urinary ALA in workmen exposed to lead at various concentrations of lead in air. In workmen exposed to 0.15 mg/m^3 and below, the highest individual blood lead found was 0.06 mg/100 g, the highest urine lead 0.12 mg/liter, the highest urinary coproporphyrin 0.186 mg/liter, and the highest urinary ALA 11.85 mg/liter. There were 48 workers exposed to air lead levels of 0.025 to 0.15 mg/m^3, whose mean blood lead level was 0.05 mg/100 g (range of 0.03 to 0.06). However, the number of workers exposed to or near 0.15 mg/m was not stated.

Zielhuis[51] has reviewed and analyzed the data of several other investigators of human absorption of lead, in terms of the relationships between blood lead, ALA, and coproporphyrin. He concluded from analysis of these data that a combination of blood lead greater than 0.08 mg/100 g with values of urinary lead greater than 0.15 mg/liter or urinary ALA greater than 20 mg/liter or urinary coproporphyrin greater than 0.80 mg/liter is evidence of an unacceptable degree of occupational exposure to lead. He did not review the relationships between airborne lead and the several indices of biological effect of absorbed lead.

The selection of 0.08 mg Pb/100 g of whole blood has been described by Kehoe[52] as the critical concentration of lead in blood below which no case of even mild poisoning has been induced by lead. The higher the concentration of blood lead above 0.08, the greater the likelihood of lead poisoning, though higher concentrations did not mean lead poisoning in all individuals. The scientific consensus supports the view of Kehoe as it applies to adults.

However, even in the hands of the best analyst, there may be a 10% error in a specific lead determination. Thus, an analysis showing a blood level of 0.08 mg/100 g may have a true value of almost 0.09. This may account for the recommendation of some authorities[48] that blood lead levels be kept below 0.07 mg/100 g.

IV. ENVIRONMENTAL DATA

Information presented in this section was selected to satisfy two purposes: (1) link measured environmental and biological levels to specific lead using industries, and (2) to link exposure levels to clinical lead intoxication. Table X-4 (from Elkins[5]) gives an overview of in-plant lead levels from various industries. Specific data for industries and a discussion of the exposures therein follow. The principal plant types covered are printing, storage battery manufacturing, and welding operations. Note that the general concentrations of lead in in-plant air range from negligible to those indicative of imminent danger. Scrutiny of specific plant operations is necessary to determine where the hazards exist and how priorities for control should be developed.

(a) Printing

The necessary characteristics of type metal prescribe the use of lead alloys. Examinations of Table X-6 (from Brandt[53]) shows that many areas could presently comply with a 0.15 mg/m^3 standard. Others such as the remelt room and stereotype room will require additional control measures.

Table X-7 (from Ruf[54]) associates exposure levels to significant functions performed by workers in the printing industry. They are obviously not 8-hour TWA levels but are nevertheless indicative of conditions. Most of the higher exposures occur while either some mechanical action is applied either to the metal (such as drossing and filing) or near the melting pots. In the former, large amounts of dust are generated, and in the latter the lead fumes present the problem.

Table X-8 shows data of Belknap [55] on calculated exposures in printing industries. The calculations were based on time spent by printers at various tasks and used data of Ruf [54] summarized in Table X-7. Calculated air exposures and urinary lead levels are shown for various operations. These air concentrations (or urinary levels) may be erroneous, because much less urinary lead would be expected at the listed air concentrations.

(b) Storage Battery Manufacture

Tables X-9 and X-10 furnish data on levels found in plants where storage batteries are produced. The percentages of workers exposed to air-lead levels greater than 0.15 mg/m^3 is important. Table X-9 directs attention to the operations where the serious hazards occur. The levels shown are serious in that they are above the recommended concentration, but also appear to be in a range that are responsive to conventional industrial hygiene control techniques.

(c) Welding and Cutting of Steel

Welding or cutting of lead bearing steels results in the generation of lead fume in significant concentrations. This is also the case when these operations are performed on steels which are either galvanized, zinc-silicate coated, or painted with lead pigmented paints. Elkins [5] observed that at 507 C the vapor pressure of lead (VP = 0.000016 mm Hg) is high enough to produce a concentration after oxidation of 0.18 mg/m^3 of lead fume. During welding or cutting temperatures may reach 1000 to 3000 C.

Table X-11 contrasts lead fume exposures when welding galvanized steel and zinc-silicate coated steel. The worst exposures occurred when welding the zinc-silicate coated steel. Electric arc welding produced an average concentration of 5.63 mg/m^3 and oxy-acetylene produced 1.96 mg/m^3 of lead. The information presented in Table X-11 developed by Pegues.[56] Samples are well identified, providing a clear picture of lead exposure in these welding operations. Note that with good ventilation breathing zone samples can be controlled to within the recommended standard. Note also that room air samples downwind from the welder can rise to levels which jeopardize the health of other workers. In Table X-12 (from Tabershaw[57]), limited data are presented to illustrate the exposures of those workers who perform cutting operations on painted structural steels. The urinary lead data indicate that sufficient protection from lead fume is not given through the use of the indicated respirators, and further controls are needed.

(d) Workers Whose Occupational Exposure is Out-of-Doors

Policemen, firemen, taxi drivers, vehicle tunnel attendants, garage mechanics, and service station attendants are examples of occupational groups who work out-of-doors, but are nonetheless exposed to lead. The primary source of this exposure is the lead salts emitted from internal combustion engines which burn leaded gasoline. Tables X-13 and X-14 were taken from a U.S. Public Health Service survey[58] of lead in the atmosphere and describe lead levels in blood and urine. This same survey shows that these workers are placed in atmospheres containing various amounts of lead for their 8-hour workday. Few of the samples indicate levels which even approach the biologic standard; however, the distribution of the samples does demonstrate the need for monitoring these individuals for lead exposure. There are many levels shown in these tables which are in excess

of normal (not occupationally exposed) levels, and this fact shows that there is absorption of lead on the job.

(e) Miscellaneous

Limited data for lead exposures in many other industries prevent a detailed analysis here. Nonferrous foundries often utilize lead alloys. Berg and Zenz[59] reported on one such foundry and stated that atmospheric lead concentrations have risen in the past twenty years. They stated that from 108 samples collected between 1943 and 1947, there were average concentrations as follows: 0.14 mg/m^3 in the melting room and 0.18 mg/m^3 in the pouring floor area. The results from 40 samples of 1953-1954 produced the following increases: 0.28 mg/m^3 in the melting room and 0.29 mg/m^3 in the pouring floor area. Extensive modification and increased ventilation reduced the concentration from 0.28 mg/m^3 to 0.03 mg/m^3. Attention to the processes and analysis of what operations produced the high concentrations facilitated the control of the lead hazard.

Leaded steel production sometimes generates hazardous occupational exposures to lead. Ruhf[60] reported that the highest atmospheric lead concentrations prevailed during the steel pouring operation in which the lead is added. Other elevated exposures were measured in processes such as the rolling mills. However, because of the intermittent nature of the operations the time weighted average exposure was below the <u>then</u> current limit of 0.20 mg/m^3. Ruhf further described control measures and manufacturing techniques whereby lead exposure can be minimized.