V. DEVELOPMENT OF STANDARD

Basis for Previous Standards

The American Conference of Governmental Industrial Hygienists (ACGIH)[61] has reviewed previous standards for lead in the work environment, and has commented that there are few meaningful data relating to the threshold limit value, probably because most authorities rely primarily on other tests for estimating lead hazards, such as urinary and blood leads, urinary coproporphyrin and ALA, as well as examination of the blood for stippled cells.

Nevertheless, attempts were made to control occupational lead poisoning by establishing acceptable air levels to guide engineering control measures. Although the point is not documented, it seems that at one time an air limit value of 0.5 mg/m^3 was used. In the 30's and 40's, a value of 0.15 mg/m^3 was a common, but often unachieved, goal based on a recommendation of a 1928 PHS survey of storage battery workers published in 1933.[62]

This value continued to be the one most often accepted until 1957, when the ACGIH increased the TLV to 0.20 mg/m^3, based in part on data of Elkins[5] showing that exposure at 0.20 mg/m^3 would result in urinary excretion at 0.20 mg/liter.

In 1971, the Conference recommended lowering of this value back to 0.15 mg/m^3. This appears to have been based in part on the recommendations of the International Subcommittee for Occupational Health, Permanent Commission and International Association of Occupational Health[63] at a 1968 meeting in Amsterdam, and on the results of the study by Williams, King, and Walford.[47]

The International Subcommittee recommended a time-weighted average concentration for a 40-hour week of 0.15 mg/m^3, on the basis that it corresponded to an acceptable blood concentration of 0.07 mg/100 ml.

The current workroom air standard established under the Occupational Safety and Health Act of 1970 (published in Part 1910.93 of the Federal Register, Volume 36, Number 157, pages 15101-15107, dated August 13, 1971) is 0.2 mg/m^3; this is a time weighted average, and is based on American National Standards Institute Z37.11-1969.[64] This ANSI standard provided no basis for its recommendation.

Basis for Recommended Environmental Standard and Biologic Monitoring

(See Appendix V - NIOSH Testimony Presented at DOL Hearing on a Lead Standard.)

Earlier in this century, efforts to reduce occupational lead poisoning were based on adherence to hygienic workroom air guides. As more knowledge developed, increasing attention was given to blood and urinary lead levels as guides to reduction of occupational poisoning. Concomitantly, there was increasing attention to better lead analyses. There was also an increasing knowledge of the relationship between levels and rates of absorption and excretion, blood lead levels, and health status.

The PHS study by Dreessen et al.[11] was undertaken during the period that the workroom air guide of 0.15 mg/m^3 was accepted,, but failure to achieve control of airborne lead to this level was common, so findings of slight effects among workers in lead-using industries by Dreessen and co-workers did not invalidate the guide. Though not documented, it appears that many industries have rotated their workers to various jobs to keep blood lead levels below 0.08 mg/100 g; thus, exposure to unsafe workplace air levels did not result in adverse effects on health.

Consequently, there is a little definitive information from experience in the United States and other countries on the suitability of 0.15 or 0.20 mg/m^3 as an air-lead level to which workers can be safely exposed over a working lifetime.

However, much experience has accrued to show that absorption of lead in amounts resulting in blood lead concentrations of 0.08 mg/100 g or less will not lead to adverse effects on health, and there is information from studies in other countries relating airborne lead levels to blood lead.

It was previously concluded (III. Biologic Effects of Exposure; Correlation of Exposure and Effect) that a blood lead level of 0.08 mg/100 g is useful for delineating acceptable from nonacceptable lead absorption. While levels below 0.08 mg/100 g are indicative of acceptable occupational lead absorption and, if also representative of past absorption of lead by an individual person, also indicative of insignificant risk of lead poisoning, it should not be concluded that lead poisoning will occur if blood lead levels exceed 0.08 mg/100 g. However, there is an increasing risk of poisoning as levels increase above 0.08 mg/100 g, so absorption of lead should be held to amounts that will result in blood lead levels less than 0.08 mg/100 g. As Kehoe[65] has stated, "...lead poisoning occurs in man only when certain well-defined conditions have been fulfilled" and that this is quantitatively applied by "...the relationship between the current rate and the extent of the absorption of the inorganic compounds of lead, and the concentration of lead in an accessible tissue of the living body, namely, the blood." Thus, a biologic standard of 0.08 mg of lead per 100 g of whole blood is recommended; it provides a margin of safety in adults,

but probably not in children. The extent of this margin of safety is not known, but it seems likely that there will be few, if any, cases of lead poisoning below 0.09 mg/100 g.

Kehoe[65] also pointed out the usefulness of urinary lead as an index of current absorption of lead, but added that it was a quantitatively less certain index than blood lead. It may be consistent with this view that Williams, King, and Walford[47] found that the best correlation between air-borne lead and biochemical index of effect was with blood lead (r = 0.90) and less correlation with urinary lead (r = 0.82). The study of Williams and co-workers[47] indicates that blood levels of 0.08 mg/100 ml is associated with a urinary lead level of 0.20 mg/liter. It has been commonly accepted that 0.20 mg/liter is a safe level in urine, based in part on the findings of Elkins.[5] However, it is important to note that Elkins' studies involved calculation of specific gravity of urine to a value of 1.024. The studies of Williams et al.[47] also calculated urinary specific gravity to 1.024. (Urinary lead levels of 0.20 mg/liter, adjusted to a specific gravity of 1.024, would be 0.133 mg/liter if the specific gravity were calculated to 1.016.) Thus, the conclusion of Zielhuis[51] that urinary lead greater than 0.15 mg/liter, uncorrected for specific gravity, represents unacceptable absorption of lead is consistent with the selection of a biologic standard for urinary lead of 0.20 mg/liter, so long as the specific gravity correction is used.

ALA and coproporphyrin assays, and blood examinations for hemoglobin, reticulocytes, and stippled cells are useful in the assessment of worker health, but are less useful than blood lead as a single criterion for

interpreting the acceptability of lead absorption, since no one of these measurements is a specific index of lead absorption, as is urinary or blood lead.

It should be emphasized that blood lead and urinary lead are good indices of current absorption of lead (in the absence of anemia or absorption of chelating agents), but are not necessarily indications of body burden of lead or of the state of health of the individual. Bone lead is probably more indicative of total body burden than is blood lead, but it is not feasible to sample bone for routine lead assay. As to state of health, overabsorption of lead by an individual in the past may have led to a high body burden of lead and may also have contributed to a state of current ill-health in the individual, all without causing currently high blood or urinary levels of lead.

Since the studies of relationship between health and airborne lead levels are inadequate, it is concluded that an air standard should be recommended from data on the relationship between airborne lead and bio-chemical indices of effect, most importantly, blood lead. There are several studies that point to 0.15 mg/m^3 as the level of airborne lead that will result in biochemical indices showing acceptable absorption of lead, in other words, showing that occupational exposure at 0.15* mg/m^3 will not result in adverse effects on the health of workers.

Tsuchiya and Harashima[46] studied storage battery workers in Japan and compared airborne lead with urinary lead, urinary coproporphyrin, basophilic stippling of erythrocytes, and, as an index of anemia, specific gravity of blood. They recommended airborne lead levels on the basis of acceptable

---

*See appendix V for basis for revised recommendation for an occupational exposure to inorganic lead.

levels of these biochemical indices. On the basis of acceptable urinary lead levels of 0.15 mg/liter, corrected to a specific gravity of 1.024, they recommended a threshold limit value of 0.10 mg/m^3. If a higher urinary lead level is accepted, as recommended in the preceding discussion of the relationship between acceptable lead absorption and urinary lead excretion, a higher air standard would result. It should be noted that the workers studied by Tsuchiya and Harashima worked 8 to 10 hours, 6 days a week, and they observed that a higher air level would have been recommended for a 40-hour week.

The study most directly relevant to the development of a recommended workplace air standard is the study of Williams, King, and Walford.[47] Their data (Table X-5), from studies of storage battery workers stable in their employment (40-hour work week, no job change in the past year, no recent absence or sickness, no change in overtime or productivity), showed that exposure at 0.15 mg/m^3 resulted in a mean blood lead of 0.060 mg/100 ml. Were mean blood lead the criterion of effect, an air standard much higher than 0.15 mg/m^3 could be recommended, but in order to keep most or all workers' blood lead below 0.084 mg/100 ml (0.080 mg/100 g), it is believed that a mean of about 0.060 mg/100 ml should be achieved. The data of Williams and associates[47] does not provide a basis for interpreting the percentage of workers exposed at 0.15 mg/m^3 that will have blood levels above 0.084 mg/100 ml. However, it is believed that a small percentage will have blood lead levels at or above 0.084 mg/100 ml or 0.080 mg/100 g, so it is recommended that workers be monitored biologically, by periodic assays of blood lead, or of blood and urinary lead.

Stankovic[50] also compared airborne lead with blood and urinary lead, and in workmen exposed to lead at 0.15 mg/m^3 and below, the highest individual blood lead found was 0.06 mg/100 g, and the highest urinary lead 0.12 mg/liter. However, the number of workers exposed at or near 0.15 mg/m^3 was not stated, so his finding of 0.06 mg/100 g as the highest individual blood lead is not believed to contradict the previously stated inference that some workers exposed at 0.15 mg/m^3 will have blood lead levels at or above 0.08 mg/100 g (especially those workers absorbing abnormal amounts of lead from nonoccupational sources).

It is of interest that conclusions of experts[63] support the recommended standard, but since data and arguments supporting their conclusions were not presented, their recommendations have not been given weight in deriving the recommended occupational air standard.

The rationale for the recommended work practices and sanitation practices was principally derived from Kehoe.[66] They are normal industrial hygiene procedures used to control occupational exposures to various dusts and fumes.

If worker exposures exceed 40 hours a week, the same TWA of 0.15 mg/m^3 should be used unless exposures so greatly exceed 40 hours a week that nonworking (excretion) time is significantly reduced; exposures up to 50 hours a week should not significantly affect the time for excretion of absorbed lead. However, maintenance of the same TWA means a proportionate reduction in average concentration as exposures exceed 40 hours a week. To achieve a TWA of 0.15 mg/m^3, the average concentration should be 0.15 mg/m^3 for a 40-hour week and 0.12 mg/m^3 for a 50-hour week.

Basis for Environmental Sampling and Analytical Method

Various methods of sampling air and of analysis of these samples have been considered, and recommended methods are presented in Appendixes I and II.

The recommended method of sampling air involves collection of 100 liters of air or more, use of breathing zone samplers with sampling at a rate of 2 liters/min., and collection on 0.45μ cellulose membrane filters. Other sampling rates and other collection media (filter paper, nitric acid impinger, electrostatic precipitation) are capable of giving equivalent results. The recommended procedure is described in Appendix I.

For analysis of lead in blood, atomic absorption spectrophotometry[67-71] and dithizone colorimetry[72,73] were considered. Appreciable consonance can be demonstrated between results obtained with atomic absorption and dithizone methods. Both methods have been used for analysis of air samples, and both are concluded to be capable of giving accurate results. After a review of the several procedures involving atomic absorption spectrophotometry, it was concluded that no one of these procedures has been sufficiently standardized. Individual laboratories get excellent results with a specific procedure, but these procedures have not been compared in a number of laboratories. Dithizone colorimetry, on the other hand, has been used for a long time and has been thoroughly studied. The procedures, interferences, sensitivity, and replicability have been studied and are described by Keenan, Byers, Saltzman, and Hyslop.[72] The recommended procedure is described in Appendix II.

Dithizone colorimetry is a wet chemical method requiring equipment found in most chemical laboratories, but requires meticulous attention to detail and to the prevention of loss and the exclusion of contamination.

Results of lead analysis by this method obtained by well trained technicians are often superior to results obtained by other methods of analysis.

Basis for Biologic Analytical Method

Blood lead was selected as the best method, and urinary lead as an acceptable method, for judging lead absorption, for reasons discussed in earlier sections (see "Basis for Recommended Environmental Standard and Biologic Monitoring").

Specific details for collection of biologic specimens for lead analysis have been described in a booklet Methods for determining lead in air and in biological materials, published by the American Public Health Association. Keppler et al.[75] described the initiation of interlaboratory evaluations of lead in an attempt to improve accuracy and reproducibility of laboratory analyses through a system of accreditation. Subsequent reports[76,77] have described some of the results, from which it is apparent that lead analysis is subject to significant error unless a very high degree of care is used.

Methods for the collection of blood and urine are described by Keenan et al.[72] (Appendix II). While lead-free Vacutainers are convenient, any lead-free tube can be used for collection and shipment or storage of blood prior to analysis. No aliquots can be taken unless blood-clotting has been prevented, either by taking aliquots before clotting or by prevention of clotting, e.g., by heparinization. Single use needles ("throw-aways") are acceptable, but must be lead-free, thus must not be lead-soldered.

Methods for the determination of lead in biological materials include dithizone colorimetry,[72,73,78] spectrography,[79] polarography,[80] and atomic absorption spectrophotometry.[69,71,81] In addition, many biochemical tests, reviewed by Chisolm,[82] have been developed; these depend on the lead-induced upset in heme synthesis. Among these biochemical tests are determination of coproporphyrin excretion,[83] urinary ALA[26,84] and ALA-D in blood.[85,86] Additional methods, such as cell stippling, porphobilinogen determinations, and examination of intranuclear inclusion bodies have received less acceptance. These biochemical indices are not recommended at this time. They can be sensitive, perhaps too sensitive, but they are not specific for lead, and are judged to be less useful than blood and urinary lead determinations for estimating the absorption of lead. However, future developments may resolve some of the present objections to the routine use of these indices of alterations of heme synthesis in the assessment of lead absorption.

The dithizone procedure is recommended for analysis of lead in blood and urine. As discussed in the previous section (Basis for Environmental Sampling Method), the method is capable of good results if meticulous attention is given to details, including sources of contamination and loss. Cholak[87] has stated that with careful control the procedure can detect as little as 0.5 µg, with a precision of + 0.5 µg, and that, with modifications, as little as 0.2 + 0.1 µg can be determined. The recommended method as described by Keenan et al.[72] is given in Appendix II. Bismuth is a possible, but uncommon, interfering substance, which can be removed by extraction.

VI. REFERENCES

1. Weast RC (ed): Handbook of Chemistry and Physics--A Ready Reference Book of Chemical and Physical Data, ed 52. Cleveland, Chemical Rubber Publishing Co, 1971-1972

2. Minerals Yearbook 1969: Metals, Minerals and Fuels. US Bureau of Mines, US Government Printing Office, 1969, vol I-II, p 638

3. Gafafer WM (ed): Occupational Diseases--A Guide to Their Recognition, PHS publication 1097. US Dept Health, Education, and Welfare, Public Health Service, 1966, pp 170-72

4. Committee on Biologic Effects of Atmospheric Pollutants: Airborne Lead in Perspective. National Academy of Sciences, National Research Council, Division of Medical Sciences, 1971

5. Elkins HB: The Chemistry of Industrial Toxicology, ed 2. New York, John Wiley & Sons Inc, 1959, pp 49-57

6. Tanquerel des Planches L: Lead Diseases: A Treatise (from Traite des maladies de plomb, ou saturnines, Paris, 1839) as cited in Hunter D: The Diseases of Occupations, ed 4. Boston, Little, Brown & Co, 1969, p 330

7. Burton H: Med-Chir Tr, London, 1840, XXIII, 63-79, as cited in Garrison FH: An Introduction to the History of Medicine--With Medical Chronology Suggestions for Study and Bibliographic Data, ed 4. Philadelphia, WB Saunders Co, 1929, p 444

8. Mayers MR: Occupational Health--Hazards of the Work Environment. Baltimore, Williams & Wilkins Co, 1969, pp 69-70

9. Hunter D: The Diseases of Occupations, ed 4. Boston, Little, Brown & Co, 1969, pp 234-89

10. Lane RE: Health control in inorganic lead industries--A follow-up of exposed workers. Arch Environ Health 8:243-50, 1964

11. Dreessen WC, Edwards TI, Reinhart WH, Page RT, Webster SH, Armstrong DW, Sayers RR: The Control of the Lead Hazard in the Storage Battery Industry, PH bulletin 262. Federal Security Agency, US Public Health Service, US Government Printing Office, 1941

12. Dingwall-Fordyce I, Lane RE: A follow-up study of lead workers. Br J Ind Med 20:313-15, 1963

13. Malcolm D: Prevention of long-term sequelae following the absorption of lead. Arch Environ Health 23:292-98, 1971

14. Henderson DA, Inglis JA: The lead content of bone in chronic Bright's disease. Australasian Ann Med 6:145-54, 1957

15. Lane RE: The care of the lead worker. Br J Ind Med 6:125-63, 1949

16. Symposium on Environmental Lead Contamination, Dec 1965, PHS publication No 1440, 0-210-345. US Dept Health, Education, and Welfare, Public Health Service, 1966

17. Rimington C: Haems and porphyrins in health and disease. I. Acta Med Scand 143(fasc III):161-96, 1952

18. Rimington C: The biosynthesis of haemoglobin. Br Med J 2:189-93, 1956

19. Eriksen L: Lead intoxication--I. The effect of lead on the in vitro biosynthesis of heme and free erythrocyte porphyrins. Scand J Clin Lab Invest 7:80-85, 1955

20. Kreimer-Birnbaum M, Grinstein M: Porphyrin biosynthesis--III. Porphyrin metabolism in experimental lead poisoning. Biochem Biophys Acta 111:110-23, 1965

21. Hernberg S, Nikkanen J, Mellin G, Lilius H: δ-Aminolevulinic acid dehydrase as a measure of lead exposure. Arch Environ Health 21:140-45, 1970

22. Millar JA, Cumming RLC, Battistini V, Carswell F, Goldberg A: Lead and δ-aminolaevulinic acid dehydratase levels in mentally retarded children and in lead-poisoned suckling rats. Lancet 2:695-98, 1970

23. Maxfield ME, Stopps GJ, Barnes JR, Snee RD, Azar A: Effect of lead on blood regeneration following acute hemorrhage in dogs. (Accepted for publication by Am Ind Hyg Assoc J)

24. Goldblatt MW, Goldblatt J: Some Aspects of Industrial Toxicology, in Merewether ERA: Industrial Medicine and Hygiene. London, Butterworth Co Publishers Ltd, vol 3, p 459, 1956

25. Baikie AG, Valtis DJ: The oxygen consumption of the blood in experimental lead poisoning. Br J Exp Pathol 35:434-38, 1954

26. Haeger-Aronsen B: Studies on urinary excretion of δ-aminolaevulic acid and other haem precursors in lead workers and

lead-intoxicated rabbits. Scand J Clin Lab Invest 12(suppl. 47):1-128, 1960

27. Vannotti A, Imholz A: Die Beziehungen des Reticulumendothels zum Umsatz des Nichthamoglobineisens. Z Gesamte Exp Med 106:597-611, 1939

28. Afonova VN: The metabolism of proteins in the blood serum in experimental lead poisoning. Sb Nauch Tr Ryazan Med Inst 12:129-32, 1960

29. Camerada P, Congiu M, Leo P: Comportamento del seromucoide e dell'acido sialico (sangue e tessuto nervoso) nell'intossicazione sperimentale da piombo nell ratto. Rass Med Sarda 66:253-63, 1964

30. Caccuri S: Primi risultate sull'azione dell'acido nicotinico sulla porfirinuria nell'saturnismo sperimentale. Folio Med 26:345, 1940

31. Fonzi S, Pengue L, Raddi R: Processi immunitari nella intossicazione sperimentale da piombo--Nota VI. Variazioni degli anticorpi antitifici (completi ed incompleti) in corso di immunizzazione attiva specifica. Lav Umano 19:324-28, 1967

32. Ambrosio L, Mazza V: Comportamento del lisozima nel siero di sangue e negli organi nelle intossicazioni sperimentali (piombo, benzolo, manganese). Riv Ist Sieroter Ital 29:252-62, 1954

33. Rubino GE, Pagliardi E, Prato V, Giangrandi E: Erythrocyte copper and porphyrins in lead poisoning. Br J Haemat 4:103-07, 1958

34. Kosmider S, Grabski J, Stradowski J: Plasma sodium, calcium and potassium levels in rabbits during experimental acute lead poisoning. Arch Immunol Ther Exp 11:303-06, 1963

35. Kanner NL: [Influence of lead on the functional state of the adrenal cortex.] Probl Endokr Gormonoter 11:83-88, 1965 (Rus)

36. Gontea I, Sutescu P, Stanciu V, Lungu D: [Vitamin C in lead poisoning in the guinea pig.] Igiena 13:501-09, 1964 (Rum)

37. Sandstead HH: Effect of chronic lead intoxication on vivo I[131] uptake by the rat thyroid. Proc Soc Exp Biol Med 124:18-20, 1967

38. Goyer RA: Lead and the kidney, in Current Topics in Pathology. New York, Springer-Verlag, 1971, vol 55, pp 147-76

39. Six KM, Goyer RA: The influence of iron deficiency on tissue content and toxicity of ingested lead in the rat. J Lab Clin Med 79:128-36, 1972

40. Gusev MI: [Limits of allowable lead concentrations in the air of inhabited localities], in Ryazanov VA (ed): [Limits of Allowable Concentrations of Atmospheric Pollutants], Bk 4, pp 5-31, 1960, translation, US Dept Commerce, Off Tech Serv, 1961 (Rus)

41. Shalamberidze OP: [Limit of allowable concentration of lead sulfide in atmospheric air], in Ryazanov VA (ed): [Limits of Allowable Concentrations of Atmospheric Pollutants], Bk 5, pp 29-38, translation, US Public Health Service, 1962 (Rus)

42. Xintaras C, Johnson BL, Ulrich CE, Terrill RE, Sobecki MF: Application of the evoked response technique in air pollution toxicology. Toxicol Appl Pharmacol 8:77-87, 1966

43. Xintaras C, Sobecki MF, Ulrich CE: Sleep: Changes in rapid-eye-movement phase in chronic lead absorption. Toxicol Appl Pharmacol 10:384, 1967; abst

44. Rosenblum WI, Johnson MG: Neuropathologic changes produced in suckling mice by adding lead to the maternal diet. Arch Pathol 85:640-48, 1968

45. McClain RM, Becker BA: Placental transport and teratogenicity of lead in rats and mice. Fed Proc 29:347, 1970; abst

46. Tsuchiya K, Harashima S: Lead exposure and the derivation of maximum allowable concentrations and threshold limit values. Br J Ind Med 22:181-86, 1965

47. Williams MK, King E, Walford J: An investigation of lead absorption in an electric accumulator factory with the use of personal samplers. Br J Ind Med 26:202-16, 1969

48. Selander S, Cramer K: Interrelationships between lead in blood, lead in urine, and ALA in urine during lead work. Br J Ind Med 27:28-39, 1970

49. Lane RE, Hunter D, Malcolm D, Williams MK, Hudson TGF, Browne RC, McCallum RI, Thompson AR, deKretser AJ, Zielhuis RL, Cramer K, Barry PSI, Goldberg A, Beritic T, Vigliani EC, Truhaut R, Kehoe RA, King E: Diagnosis of inorganic lead poisoning: A statement. Br Med J 4:501, 1968

50. Stankovic MK: Biochemical tests for the appraisal of exposure to lead. Arch Environ Health 23:265-69, 1971

51. Zielhuis RL: Interrelationship of biochemical responses to the absorption of inorganic lead. Arch Environ Health 23:299-311, 1971

52. Kehoe RA: The Harben Lectures 1960. The metabolism of lead in man in health and disease—Present hygienic problems relating to the absorption of lead, lecture 3. J R Inst Public Health 24:177-203, 1961

53. Brandt AD, Reichenbach GS: Lead exposures at the government printing office. J Ind Hyg Toxicol 25:445-50, 1943

54. Ruf HW: Studies on the lead hazards in certain phases of printing. I. Actual lead exposures as measured by the amount of lead in printing atmospheres. J Ind Hyg Toxicol 22:445-63, 1940

55. Belknap EL: Studies on the lead hazards in certain phases of printing—II. Actual lead absorption as measured by physical examinations, blood and urine studies. J Ind Hyg Toxicol 22:463-71, 1940

56. Pegues WL: Lead fume from welding on galvanized and zinc-silicate coated steels. Am Ind Hyg Assoc J 21:252-55, 1960

57. Tabershaw I, Ruotolo BPW, Gleason RP: Plumbism resulting from oxyacetylene cutting of painted structural steel. J Ind Hyg Toxicol 25:189-91, 1943

58. Survey of Lead in the Atmosphere of Three Urban Communities, PHS publication 999-AP-12. US Dept Health, Education, and Welfare, Public Health Service, 1965

59. Berg BA, Zenz C: Environmental and clinical control of lead exposure in a non-ferrous foundry. Am Ind Hyg Assoc J 28:175-78, 1967

60. Ruhf PC: Lead exposure control in the production of leaded steel. Am Ind Hyg Assoc J 24:63-67, 1963

61. American Conference of Governmental Industrial Hygienists: Transactions of the thirty-third annual meeting of the American Conference of Governmental Industrial Hygienists, Toronto, Canada, May 24-28, 1971. Lead, pp 129-132

62. Russell AE, Jones RR, Bloomfield JJ, Britten RH, Thompson LR: Lead poisoning in a storage battery plant, PHS bulletin 205. US Treasury Dept, Public Health Service, 1933

63. Permanent Commission and International Association of Occupational Health: Report on the Meeting of International

Subcommittee for Occupational Health in International Conference on Inorganic Lead - 28-29 Nov, 1968, Amsterdam. Ind Med Surg 38:10-11, 1969

64. USA Standard Acceptable Concentrations of Lead and its Inorganic Compounds, USAS Z37.11-1969 (revision of Z37.11-1943). New York, United States of America Standards Institute Inc (now American National Standards Institute Inc), 1969

65. Kehoe RA: Standards with respect to atmospheric lead. Arch Environ Health 8:348-54, 1964

66. Kehoe RA: Industrial Lead Poisoning, in Patty FA: Industrial Hygiene and Toxicology, revised ed 2. New York, John Wiley & Sons Inc, vol II, pp 941-80, 1963

67. Analytical Methods for Atomic Absorption Spectroscopy. The Perkin Elmer Corp, 1971

68. Slavin W: Atomic Absorption Spectroscopy. New York, Interscience Publishers, pp 118-22, 1968

69. Farrelly RO, Pybus J: Measurement of lead in blood and urine by atomic absorption spectrophotometry. Clin Chem 15:566-74, 1969

70. Hwang JY, Ullucci PA, Smith SB Jr, Malenfant AL: Atomic Absorption Spectrometric Determination of Lead in Blood by the Solid Phase Sampler Technique. Application Report, Lexington, Mass, Instrumentation Laboratory Inc, 1971

71. Pierce JO II, Cholak J: Lead, chromium, and molybdenum by atomic absorption. Arch Environ Health 13:208-12, 1966

72. Keenan RG, Byers DH, Saltzman BE, Hyslop FL: The "USPHS" method for determining lead in air and in biological materials. Am Ind Hyg Assoc J 24:481-91, 1963

73. Cholak J, Hubbard DM, Burkey RE: Microdetermination of lead in biological material with dithizone extraction at high pH. Anal Chem 20:671-72, 1948

74. Subcommittee on Chemical Methods, Committee on Ventilation and Atmospheric Pollution, Industrial Hygiene Section: Methods for Determining Lead in Air and in Biological Materials. New York, American Public Health Association, 1944

75. Keppler JF, Maxfield ME, Moss WD, Tietjen G, Linch AL: Interlaboratory evaluation of the reliability of blood lead analyses. Am Ind Hyg Assoc J 31:412-23, 1970

76. Weil CS: Critique of interlaboratory evaluation of the reliability of blood-lead analyses. Am Ind Hyg Assoc J 32:304-12, 1971

77. Donovan DT, Vought VM, Rakow AB: Laboratories which conduct lead analysis on biologic specimens. Arch Environ Health 23:111-13, 1971

78. Vinter P: The estimation of lead in blood. J Med Lab Technol 21:281-86, 1964

79. Cholak J, Story RV: Spectrographic and analysis of biological material--III. Lead, tin, aluminum, copper, and silver. Ind Eng Chem Anal Ed 10:619-22, 1938

80. Cholak J, Bambach K: Determination of lead in biological material: A polarographic method. Ind Eng Chem Anal Ed 13:583-86, 1941

81. Berman E: The determination of lead in blood and urine by atomic absorption spectrophotometry. Atomic Absorp Newsletter 3:111-14, 1964

82. Chisolm JJ Jr: Disturbances in the biosynthesis of heme in lead intoxication. J Pediatr 64:174-87, 1964

83. Askevold R: Routine analysis of porphyrines in urine. Scand J Clin Lab Invest 3:318-19, 1951

84. Mauzerall D, Granick S: The occurrence and determination of δ-aminolevulinic acid and porphobilinogen in urine. J Biol Chem 219:435-46, 1956

85. Granick S, Mauzerall D: Porphyrin biosynthesis in erythrocytes: II. Enzymes converting δ-aminolevulinic acid to coproporphyrinogen. J Biol Chem 232:1119-40, 1958

86. Bonsignore D: [The erythrocyte aminolevulinic (ALA) dehydratase activity as a diagnostic test in occupational lead poisoning] Med Lav 57:647-54, 1966 (It)

87. Cholak J: Analytical methods for determination of lead. Arch Environ Health 8:222-31, 1964

VII. APPENDIX I

METHOD FOR SAMPLING OF LEAD IN AIR

Lead dust or fume is collected on 0.45 µ cellulose membrane filters mounted in either 2- or 3-piece filter cassettes. Air is drawn through the filter by means of a pump at a rate of 2 liters a minute (not less than 1 nor more than 4 liters per minute). A minimum sample of 100 liters shall be collected. Larger sample volumes are encouraged provided the filters do not become loaded with dust to the point that loose material would fall off or the filter would become clogged.

With each group of samples, one filter, labeled as a blank, shall be submitted; no air shall be drawn through this filter.

The sample cassettes, if shipped, must be packed in a suitable container to prevent damage in transit. Loss of sample shall be prevented; loss of loose deposits on the filter can be prevented by mounting a clean filter in the cassette on top of the sample filter.

Ash the filter and analyze for lead as described in Appendix II.

Other collection methods shown to be equivalent may be used.