provisions of 49 CFR 1131.3. These rules provide that an original and six (6) copies of protests to an application may be filed with the field official named in the FEDERAL REGISTER publication no later than the 15th calendar day after the date the notice of the filing of the application is published in the FEDERAL REGISTER. One copy of the protest must be served on the applicant, or its authorized representative, if any, and the protestant must certify that such service has been made. The protest must identify the operating authority upon which it is predicated, specifying the "MC" docket and "Sub" number and quoting the particular portion of authority upon which it relies. Also, the protestant shall specify the service it can and will provide and the amount and type of equipment it will make available for use in connection with the service contemplated by the TA application. The weight accorded a protest shall be governed by the completeness and pertinence of the protestant's information.

Except as otherwise specifically noted, each applicant states that there will be no significant effect on the quality of the human environment resulting from approval of its application.

A copy of the application is on file, and can be examined at the Office of the Secretary, Interstate Commerce Commission, Washington, D.C., and also in the ICC Field Office to which protests are to be transmitted.

MOTOR CARRIERS OF PROPERTY

WATER CARRIER APPLICATION

W-1117 (Sub-1TA). Applicant: Missouri River Passenger Excursions, Inc., P.O. Box 14181, west Omaha Station, Omaha, NE 68124. Representative: Timothy C. Mason (same as above). By decision entered November 2, 1978, the Motor Carrier Board granted Missouri River Passenger Excursions, Inc., 180-day temporary authority to engage in the business of transportation in Interstate or Foreign Commerce, as a *common carrier* by water in the transportation of passengers between Sioux City, IA and St. Joseph, MO on the Missouri River. Any interested person may file a petition for reconsideration within 20 days of the date of this publication.

By the Commission.

H. G. HOMME, Jr.,
Secretary.

[FR Doc. 78-32700 Filed 11-20-78; 8:45 am]

[7035-01-M]

[Notice No. 744]

ASSIGNMENT OF HEARINGS

NOVEMBER 16, 1978.

Cases assigned for hearing, postponement, cancellation or oral argument appear below and will be published only once. This list contains prospective assignments only and does not include cases previously assigned hearing dates. The hearings will be on the issues as presently reflected in the Official Docket of the Commission. An attempt will be made to publish notices of cancellation of hearings as promptly as possible, but interested parties should take appropriate steps to insure that they are notified of cancellation or postponements of hearings in which they are interested.

MC 106644 (Sub-253F), Superior trucking Co., now being assigned for hearing on February 20, 1979, (1 day), at Chicago, IL in a hearing room to be later designated.

MC 106497 (Sub-154F), Parkhill Truck Co., now being assigned for hearing on February 21, 1979, (3 days), at Chicago, IL in a hearing room to be later designated.

MC 135235 (Sub-6F), Loma Cartage, Inc., now being assigned for hearing on February 26, 1979, (5 days), at Chicago, IL in a hearing room to be later designated.

MC F 13593, Nebraska Transport, Co., Inc.—Control—, G & H. Truck Line, Inc., now being assigned for hearing on February 6, 1979, (9 days), at Scottsbluff, NE in a hearing room to be later designated.

MC 111545 (Sub-250F), Home Transportation Co., Inc., now being assigned for hearing on February 6, 1979, (9 days), at the Holiday Inn Tampa Central, 111 West Fortune Street, Tampa, FL.

MC 115331 (Sub-457F), Truck Transport, Inc., now assigned January 15, 1979, at Chicago, IL, is canceled and reassigned to February 20, 1979, (4 days), at the offices of the Interstate Commerce Commission, Washington, DC.

MC 116254 (Sub-108M1), Chem-Haulers, Inc., now assigned January 15, 1979, at Washington, DC, and continued to February 12, 1979, at Nashville, TN, and continued to April 2, 1979, at Washington, DC, are canceled and the application is dismissed.

MC 114273 (Sub-362F), CRST, Inc., now assigned January 9, 1979, (2 days), at Chicago, IL, will be held in Room 3855A, 230 South Dearborn Street.

MC 8472 (Sub-5F), South End Cartage, Inc., now be assigned for hearing on January 11, 1979, (2 days), at Chicago, IL, Room 3855A, 230 South Dearborn Street.

MC 142706 (Sub-2F), Early Bird Transfer, Inc., now be assigned for hearing on January 15, 1979, at Chicago, IL, Room 3855A, 230 South Dearborn Street.

MC 2890 (Sub-54F), American Buslines, Inc., now assigned for hearing on January 15, 1979, at El Centro, CA and will be held in the Holiday Inn.

MC 144581, Harvey Hayes, an individual doing business as Hayes Trailer Transport, now assigned for hearing on January 10, 1979 at Los Angeles County Courthouse and will be held in Los Angeles, CA.

MC 125433 (Sub-144F), F-B Truck Line Co., now assigned for hearing on January 9, 1979, at Los Angeles, CA and will be held in Los Angeles County Courthouse.

MC 71043 (Sub-10F), Laporte Transit Co., Inc., now assigned for hearing on January 9, 1979, at Chicago, IL and will be held in Room 1319.

MC 113908 (Sub-421), Erickson Transport Corp., now assigned for hearing on December 4, 1978, at Chicago, IL and will be held in St. Francis Hotel.

MC 144533, Frank Pagliughi, an individual, d.b.a. General Transfer Co., now assigned for hearing on December 14, 1978, at U.S. District Court and will be held in Philadelphia, PA.

MC 140024 (Sub-110/f), J. B. Montgomery, Inc., now assigned for hearing on December 13, 1978, at Philadelphia, PA and will be held in U.S. District Court.

MC 140024 (Sub-106F), J. B. Montgomery, now assigned for hearing on December 11, 1978, at Philadelphia, PA and will be held in U.S. District Court.

MC 140024 (Sub-112F), J. B. Montgomery, now assigned for hearing on December 11, 1978, at Philadelphia, PA and will be held in U.S. District Court.

MC 115826 (Sub-300F), W. J. Digby, Inc., now assigned for hearing on December 7, 1978, at Denver, CO, and will be held in Division 2, Court of Appeals.

MC 144140 (Sub-6F), Southern Freightways, Inc., now assigned for hearing on December 6, 1978, at Orlando, FL and will be held in Howard Johnson's Executive Center.

MC 94201 (Sub-161F), Bowman Transportation, Inc., now assigned for hearing on December 6, 1978, at Jackson MS and will be held in Grand Jury Room, U.S. Post Office and Courthouse Building.

MC 143296 (Sub-2F), Peach State Bus Lines, Inc., now assigned for hearing on December 6, 1978, at Atlanta, GA and will be held in Room 305.

MC 56679 (Sub-87), Brown Transport Corp., Alternate Regular Route Authority of General Commodities now assigned for hearing on December 4, 1978, at Atlanta, GA and will be held in Room 202, North Annex.

MC 65920 (Sub-5F), Bishop Motor Express, Inc., now assigned for hearing on December 4, 1978, at Lansing, MI and will be held in room 203, Federal Building.

MC 133659 (Sub-3), Livingston Storage And Transfer Co., Inc., now assigned for hearing on November 29, 1978, at Atlanta GA and will be held in Room 556, Federal Building.

MC 134017 (Sub-7F), R. M. Henderson, d.b.a. H & M Motor Lines, now assigned for hearing on November 28, 1978, at Atlanta GA and will be held in Room 556, Federal Building.

MC 144541F, Baldwin Leasing Co., Inc., now assigned for hearing on December 6, 1978, at Mobile, AL and will be held in Room 440, Federal Building.

MC 56679 (Sub-92), Brown Transport Corp., & MC 56679 (Sub-93), Brown Transport Corp., now assigned for continued hearing on December 18, 1978 (1 day) at Atlanta, GA and will be held in Room 305, 1252 West Peachtree St. N.W.

MC 114632 (Sub-161F), Apple Lines, Inc., now assigned December 11, 1978 for prehearing conference at Washington, DC at

the Offices of the Interstate Commerce Commission.

MC 124083 (Sub-58F), Skinner Motor Express, Inc., now assigned December 4, 1978 at Washington DC for pre-hearing conference is canceled transferred to Modified Procedure.

MC 114273 (Sub-325), Crst, Inc., now assigned January 11, 1979 at Chicago, IL is canceled and application dismissed.

MC 144011, Hall Systems, Inc. now assigned for continued hearing January 15, 1979 at Birmingham, AL (5 days) in a hearing room to be later designated.

MC 105813 (Sub-241F), Belford Trucking Co., Inc., now assigned for hearing on December 6, 1978, at Atlanta, GA and will be held in Room 202, North Annex.

MC 118859 (Sub-11F), Bullock Trucking Co., Inc., now assigned for hearing on December 7, 1978, at Atlanta, GA and will be held in Room 202, North Annex.

MCF 13500, Burlington Northern, Inc.—Control—Frisco Transportation Co., and (FD 28583 Sub No. 1 and 2), now assigned for hearing on January 3, 1979, at Chicago, IL and will be held in Room 1319, E. M. Dirksen Building.

MC 8457 (Sub-6F), Milwaukie Transfer & Fuel Co., now assigned January 10, 1979 (3 days) at Portland, OR in a hearing room to be later designated.

MC-C 10143, O.N.C. Freight Systems, Inc-V-Herbert D. Needel, d.b.a. Tucson Package Delivery now assigned January 5, 1979 (1 day) at Phoenix, AZ in a hearing room to be later designated.

MC 124692 (Sub-204F), Sammon Trucking., now assigned January 15, 1979 (5 days) at Salem, OR in a hearing room to be later designated.

MC 138635 (Sub-50F), Carolina Western Express, Inc., now assigned December 14, 1978 at Los Angeles, CA, is canceled and application dismissed.

MC 263 (Sub-226F), Garrett Freightlines, Inc., now assigned December 12, 1978 for pre-hearing conference at Washington, DC at the offices of the Interstate Commerce Commission.

MC 109173 (Sub-4F), Delta Bus Lines, Inc., now assigned for hearing on January 15, 1979, at Lansing, MI is canceled and application dismissed.

H. G. Homme, Jr.,
Secretary.

[FR Doc. 78-32699 Filed 11-20-78; 8:45 am]

sunshine act meetings

This section of the FEDERAL REGISTER contains notices of meetings published under the "Government in the Sunshine Act" (Pub. L. 94-409), 5 U.S.C. 552b(e)(3).

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[6320-01-M]

1

[M-178, Amdt. 3; Nov. 16, 1978]

CIVIL AERONAUTICS BOARD.

Notice of deletion of items to the November 16, 1978, meeting.

TIME AND DATE: 9:30 a.m., November 16, 1978.

PLACE: Room 1027, 1825 Connecticut Avenue NW., Washington, D.C. 20428.

SUBJECT:

2. Delegation of Authority to the Director, Bureau of Pricing and Domestic Aviation, to act on complaints challenging the reasonableness of fares within the "no-suspend" zones established by PS-80 (Memo 7847-J, BPDA, OGC).

25. Docket 31977, Application of International Developers, Inc. (Japan) d.b.a. Toyo World Enterprises of California, Inc., for indirect foreign air carrier permit (Memo 8097-A, BIA, OGC).

STATUS: Open.

PERSON TO CONTACT:

Phyllis T. Kaylor, the Secretary, 202-673-5068.

SUPPLEMENTARY INFORMATION: Item 2 was deleted from the November 16, 1978, meeting because the staff wishes to make technical adjustments to the delegation to reflect the new Act and is awaiting completion of another item. Accordingly, the following Members have voted that item 2 be deleted from the November 16, 1978, agenda and that no earlier announcement of this deletion was possible;

Chairman, Marvin S. Cohen
Member, Richard J. O'Melia
Member, Gloria Schaffer

Item 25 was deleted because this item relates to international matters, the Board wanted to consider whether to hold a closed or open meeting regarding it prior to discussion of item. Accordingly, the following Members have voted that agency business requires the deletion of item 25 from the November 16, 1978, agenda and that no earlier announcement of this deletion was possible:

Chairman, Marvin S. Cohen
Member, Richard J. O'Melia
Member, Elizabeth E. Bailey
Member, Gloria Schaffer

[S-2352-78 Filed 11-17-78; 3:55 pm]

[6712-01-M]

2

FEDERAL COMMUNICATIONS COMMISSION.

TIME AND DATE: 9:30 a.m., Tuesday, November 21, 1978.

PLACE: Room 856, 1919 M Street NW., Washington, DC.

STATUS: Special open Commission meeting.

MATTERS TO BE CONSIDERED:

Agenda, Item No., and subject

Common Carrier—1—Reconsideration petitions of Commission's action rejecting A.T. & T.'s April 29, 1977, WATS filing.

Common Carrier—2—Inquiry designated to consider whether Inward and Outward Wide Area Tele-communications Services (WATS) are "like communications service" to Message Tele-communications Service (MTS) within the meaning of section 202(a) of the Act.

Common Carrier—3—Petitions to enlarge and delete issues in Docket No. 20690 which is an inquiry into the addition of 1.544 Mbp's speed to A.T. & T.'s Dataphone Digital Service.

Common Carrier—4—Application of Communications Satellite Corporation (COMSAT), A.T. & T., ITT World Communications, Inc., RCA Global Communications, Inc., and Western Union International, Inc., for authority to construct 14/11 GHz communication satellite earth station facilities in the vicinity of Etam and Lenox, W. Va. and a terrestrial interconnecting link via Laurel Mountain for operation with Commission Satellite Systems.

Common Carrier—5—Application by A.T. & T. for authority to construct a domestic satellite earth station at the Goddard Space Flight Center, Greenbelt, Md. (File No. 652-DSE-P-77).

Common Carrier—6—Western Union International, Inc., application to lease and operate 14/56 Kilobit circuits in the Westar and Intelsat satellite systems and construct a domestic satellite Earth station at each Andover, Maine and Etam, W. Va., et al. and Communications Satellite Corp. application to construct and operate an 11 meter antenna associated facilities at each Andover, Maine, and Etam, W. Va., and to use said facilities in conjunction with the Westar domestic satellites et al.

This meeting may be continued the following workday to allow the Commission to complete appropraite action.

Additional information concerning this meeting may be obtained from the FCC Information Office, telephone 202-632-7260.

Issued: November 15, 1978.

[S-2346-78 Filed 11-17-78; 11:00 am]

[6715-01-M]

3

FEDERAL ELECTION COMMISSION.

"FEDERAL REGISTER" NO. FR-S-2287.

PREVIOUSLY ANNOUNCED DATE AND TIME: Thursday, November 16, 1978, at 10 a.m.

CHANGE IN MEETING: The Commission determined by a vote of 6-0 to consider a draft response to AOR 1978-92 and to discuss AOR 1978-90 pursuant to its authority under 11 CFR 3.5(d)(1).

Pursuant to 11 CFR 112.3(b), the Commission determined by a vote of 6-0 to shorten the comment period on AOR 1978-90 and AOR 1978-92 to November 21, 1978, close of business.

PERSON TO CONTACT FOR INFORMATION:

Mr. David Fiske, Press Officer, telephone, 202-523-4065.

MARJORIE W. EMMONS,
Secretary to the Commission.

[S-2345-78 Filed 11-17-78; 11:00 am]

[6750-01-M]

4

FEDERAL TRADE COMMISSION.

"FEDERAL REGISTER" CITATION OF PREVIOUS ANNOUNCEMENT: FR 43, November 13, 1978, page No. 52602.

PREVIOUSLY ANNOUNCED TIME AND DATE OF THE MEETING: 9 a.m., Wednesday, November 15, 1978.

CHANGES IN THE AGENDA: The Federal Trade Commission has deleted an item from the agenda of its previously announced open meeting of November 15, 1978. Because this was the only item scheduled, the meeting has been canceled and rescheduled for an open meeting on Tuesday, November 21, 1978, 1:30 p.m.

[S-2350-78 Filed 11-17-78; 2:08 pm]

[6750-01-M]

5

FEDERAL TRADE COMMISSION.

TIME AND DATE: 2 p. m. Tuesday, November 21, 1978.

PLACE: Room 432, Federal Trade Commission Building, Sixth Street and Pennsylvania Avenue NW. Washington, D.C. 20580.

STATUS: Open.

MATTERS TO BE CONSIDERED: Consideration of proposed trade regulation rule governing labeling and advertising of home insulation.

CONTACT PERSON FOR MORE INFORMATION:

Ira J. Furman, Office of Public Information, 202-523-3830; Recorded message 202-523-3806.

[S-2351-78 Filed 11-17-78; 2:08 pm]

[7600-01-M]

6

OCCUPATIONAL SAFETY AND HEALTH REVIEW COMMISSION.

TIME AND DATE: 1 p.m., November 30, 1978.

PLACE: Room 1101, 1825 K Street NW., Washington, D.C.

STATUS: Because of the subject matter, it is likely that this meeting will be closed.

MATTERS TO BE CONSIDERED: Discussion of specific cases in the Commission adjudicative process.

CONTACT PERSON FOR MORE INFORMATION:

Ms. Patricia Bausell, 202-634-4015.

Date: November 15, 1978.

[S-2348-78 Filed 11-17-78; 11:34 am]

[7751-01-M]

7

POSTAL RATE COMMISSION.

"FEDERAL REGISTER" CITATION OF PREVIOUS ANNOUNCEMENT: 43 FR 53123, November 15, 1978.

PREVIOUSLY ANNOUNCED TIME AND DATE OF THE MEETING: 10:30 a.m., Tuesday, November 21, 1978.

CHANGES IN THE MEETING: Following the previously announced "open" meeting at 10:30 a.m., the Commission will have a "closed" meeting to discuss the draft Opinion and Recommended Decision in Dockets No. MC76-5 and MC77-2. The meeting is closed pursuant to 5 U.S.C. 552b(c)(10).

CONTACT PERSON FOR MORE INFORMATION:

Ned Callan, Information Officer, Postal Rate Commission, Room 500, 2000 L Street NW., Washington, D.C. 20268, telephone 202-254-5614.

[S-2347-78 Filed 11-17-78; 11:00 am]

[8010-01-M]

8

SECURITIES AND EXCHANGE COMMISSION.

"FEDERAL REGISTER" CITATION OF PREVIOUS ANNOUNCEMENT: 43 FR 52359, November 9, 1978.

STATUS: Closed meeting.

PLACE: Room 825, 500 North Capitol Street, Washington, D.C.

DATE PREVIOUSLY ANNOUNCED: Tuesday, November 7, 1978.

CHANGES IN THE MEETING: Additional items to be considered.

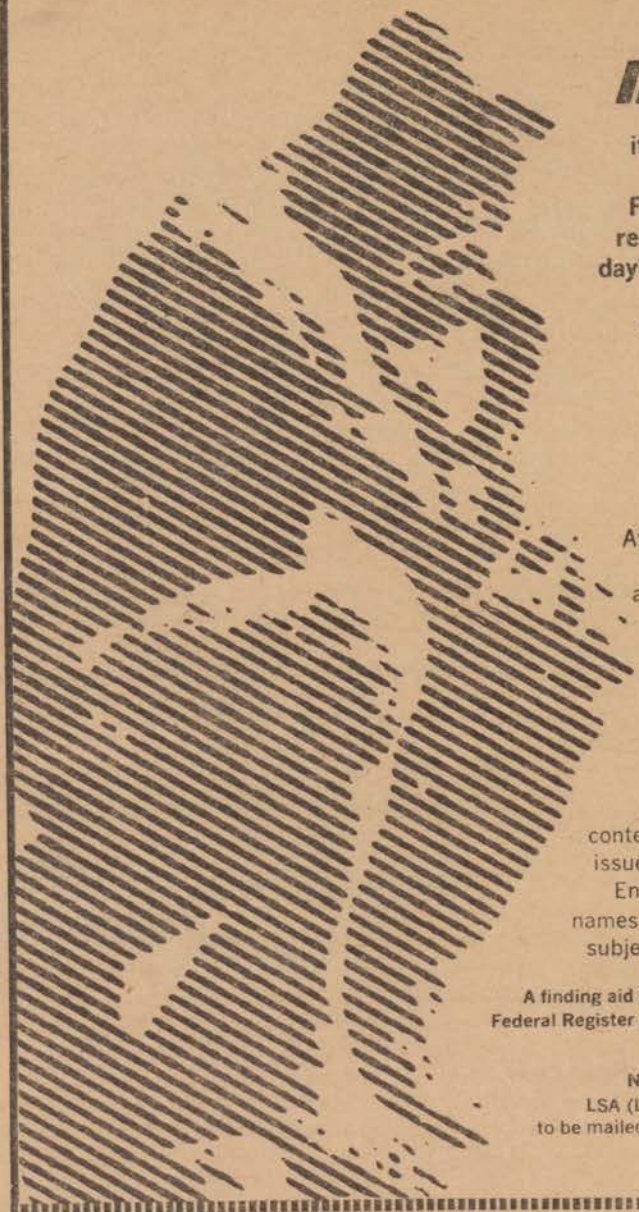
The following additional items will be considered at the closed meeting to be held on Wednesday, November 15, 1978, immediately following the 10 a.m. open meeting:

Disclosure matter bearing enforcement implications.
Regulatory matter bearing enforcement implications.
Settlement of injunctive action.

Chairman Williams and Commissioners Loomis, Evans, and Karmel determined that Commission business required the above change and that no earlier notice thereof was possible.

NOVEMBER 15, 1978.

[S-2349-78 Filed 11-17-78; 2:08 pm]



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Vol. 43—No. 225
11-21-78
BOOK 2:
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federal register

BOOK 2 OF 2 BOOKS

TUESDAY, NOVEMBER 21, 1978
PART II



DEPARTMENT OF LABOR

Occupational Safety and Health Administration

■

OCCUPATIONAL EXPOSURE TO LEAD

Attachments to the Preamble for the Final Standard

[4510-26-M]

Title 29—Labor

CHAPTER XVII—OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION, DEPARTMENT OF LABOR

PART 1910—OCCUPATIONAL SAFETY AND HEALTH STANDARDS

Occupational Exposure to Lead

AGENCY: Occupational Safety and Health Administration, Department of Labor.

ACTION: Final standard for occupational exposure to lead.

SUMMARY: This document contains part VI of the Statement of Reasons (Preamble) accompanying the final OSHA standard for occupational exposure to lead. The regulation and parts I through V of the Statement of Reasons were published in the FEDERAL REGISTER on November 14, 1978.

DATES: The effective date of the standard is February 1, 1979.

FOR FURTHER INFORMATION CONTACT:

Gail Brinkerhoff, OSHA Office of Compliance Programs, U.S. Department of Labor, Room N-3112, Washington, D.C. 20210. Telephone: 202-523-8034. For additional copies of this document, contact OSHA Office of Publications, U.S. Department of Labor, Room N-3423, Washington, D.C. 20210. Telephone: 202-523-8677.

SUPPLEMENTARY INFORMATION:

VI. ATTACHMENTS TO PREAMBLE

ATTACHMENT A—HEALTH EFFECTS

The basis for this revised lead standard is evidence of the toxic effects of lead on the heme, renal, neurological, and reproductive systems at relatively low levels of exposure to lead. This section provides an in-depth analysis of the health effects evidence and is divided into the following sections:

1. *Summary and General Considerations.* A discussion of the symptomatology associated with lead intoxication.

2. *Heme Biosynthesis Inhibition.* A discussion of the effects low levels of lead exposure have on the biosynthesis of heme.

3. *The Neurological System.* A discussion of the effects of lead exposure on the peripheral and central nervous system.

4. *Renal System.* Effects of lead exposure on the kidneys with regard to lead as an etiologic agent in urinary disease and hypertension.

5. *Reproductive System.* The effects of lead exposure on the course of pregnancy with particular reference to the fetus.

6. *Mortality Studies.* A discussion of the mortality experience resulting from lead exposure.

7. *Air/Blood Relationship.* A discussion of the intrinsic relationship between air lead levels and blood lead levels.

1. *Summary and General Considerations.* In the preamble to the proposed lead standard OSHA described the overt manifestations of lead poisoning.

> The primary sources of lead absorption in workers are the inhalation and ingestion of industrial lead. Deposition and retention of absorbed lead in body tissues is variable, but it is found in the brain, liver, kidney, aorta, muscles and bones. Absorbed lead is transported to these tissues via the blood system and some portion is removed from the body primarily through the alimentary tract and urinary system.
>
> Lead intoxication, in its severest forms, can cause permanent damage to the body or cause death. Observed clinical effects include damage to the central nervous system, including the brain, i.e., acute and chronic encephalopathy, damage to the peripheral nervous system, damage to the kidneys and damage to the blood forming process which may lead to anemia. Symptoms which may vary in severity include colic, i.e., abdominal pain; loss of appetite; constipation; excessive tiredness and weakness; nervous irritability and fine tremors. Encephalopathy is the most severe acute clinical form of lead intoxication. It may arise precipitously with the onset of intractable seizures, followed by coma, cardiorespiratory arrest and death. In peripheral neuropathy, the distinguishing clinical feature of lead intoxication is a predominance of motor impairment, with minimal or no sensory abnormalities. There is a tendency for the extensor muscles of the hands and feet to be affected. Lead intoxication has also resulted in kidney damage with few, if any symptoms appearing until permanent damage has occurred. In addition, the use of chelating agents, such as Ca-EDTA, to remove lead from the body increases the risk of kidney damage or failure. (Ex 2, p. 45935)

The overt symptoms outlined above have been described in a number of reviews (Ex. 95; Final Environmental Impact Statement: Inorganic Lead; U.S. Department of Labor, Occupational Safety and Health Administration, (FEIS) April, 1978, Ref. 92: Air Quality Criteria For Lead, Environmental Protection Agency, later cited as EPA Criteria Document: Ex. 1), and will be discussed in more detail in the respective sections. The primary issue which the Health Effects section must address is at which blood lead levels do clinical symptoms and effects caused by lead occur. The proposal raised the issue as follows:

> A number of studies have sought to relate clinical symptoms and effects caused by lead exposure on workers' blood lead levels. There is little disagreement that the risk of clear-cut clinical symptoms related to exposure increases as blood lead levels rise above 80 μg/100 g. In addition, a number of studies have observed symptoms and effects caused by exposure to lead at blood lead levels below 80 μg/100 g. While 80 μg/100 g is a useful lower range for observed clear-cut clinical symptoms, we do not regard it as a sharp delineation above which clear-cut symptoms occur in all workers and below which clear-cut symptoms do not occur. Further workers with blood lead levels above 80 μg/100 g without clear-cut symptoms may have milder symptoms caused by lead exposure. It should be noted that in evaluating studies which seek to relate blood lead levels to symptoms of lead exposure, it is rarely possible in clinical situations to determine the amount of lead absorbed before the onset of symptoms of lead intoxication.
>
> * * * * * *
>
> In summary, it is OSHA's judgment that the probability of clinical symptoms of lead intoxication appearing is increased as blood lead levels rise above 80 μg/100 g. There are also data, however, to suggest that such symptoms may occur at blood lead levels under 80 μg/100 g, although perhaps not under 50 μg/100 g. (Ex. 2, p. 45935.)

In addition, the proposal stressed the importance of considering "subclinical effects" which appear earlier and at significantly lower blood lead levels than seen in cases of severe intoxication. These effects include heme synthesis impairment as manifested by enzyme inhibition, and neurological disease of both the central and peripheral nervous systems indicated by CNS symptoms, behavioral changes and electrophysiologic abnormalities. Lead is a well documented occupational hazard whose effects range from changes in biochemical and pysiological parameters to chronic disease, permanent impairment and death. The record indicates that these effects occur at exposure levels heretofore considered safe for workers, and this same record documents the more modern research which provides the basis for the ultimate conclusions upon which the standard is based. The following sections will evaluate the adverse effects of lead in detail.

2. *Heme Biosynthesis Inhibition.* The effects of lead on the hematopoietic system have been extensively studied. There is little debate that the inhibition of various enzyme systems occurs at PbB levels of 40 μg/100 ml and below. There is no controversy concerning the fact that at this level the buildup of two heme synthesis substrates, aminolevulenic acid (ALA) and protoporphyrin becomes significant. There is, however, controversy concerning what these effects mean. OSHA has evaluated the record and concluded that these effects must be viewed as early steps in a continuous disease process which eventually results in lead poisoning. Such effects are of themselves indicative of physio-

logical disruptions of subcellular processes. Therefore, disruption of such processes over a working lifetime must be viewed as material impairment of health.

Anemia is an established sequelae of lead poisoning, and one of the later steps on the continuum of blood related disease effects described above. Symptoms of anemia are known to occur at PbB levels greater than 80 μg/100 ml, however, the occurrence of anemia at PbB levels below this level was debated. OSHA has concluded that such symptoms may occur at PbB levels as low as 50 μg/100 ml.

Finally, in evaluating the effects of lead, it must be realized that lead does not disrupt heme synthesis exclusively in the hematopoietic system. Lead also disrupts the process of heme synthesis in the mitochondria of every other body cell, including the kidney and nervous tissues. Heme synthesis disruption measurable at PbB levels of 40 μg/100 ml is, therefore, an indirect measure of the disruptive effects of lead in other tissues.

The proposal outlined biohemical and physiological changes which are detectable at blood lead levels lower than those normally associated with clinical symptoms. These changes have been shown to occur in tissues throughout the body, and are the manifestation of lead-induced damage at the subcellular level.

Our understanding of the physiological action of lead in many of these tissues is lacking, as most of the information about lead's effect on heme synthesis is derived from studies on the hematopoietic system. Although the effects of lead on the hematopoietic system may not be the most serious occupationally, this area has been studied in detail for the following reasons:

(1) The biochemical pathway for the synthesis of heme is well understood; and

(2) Clinically, blood samples are relatively easy to obtain compared to brain, kidney or other tissue samples. (Tr. 461)

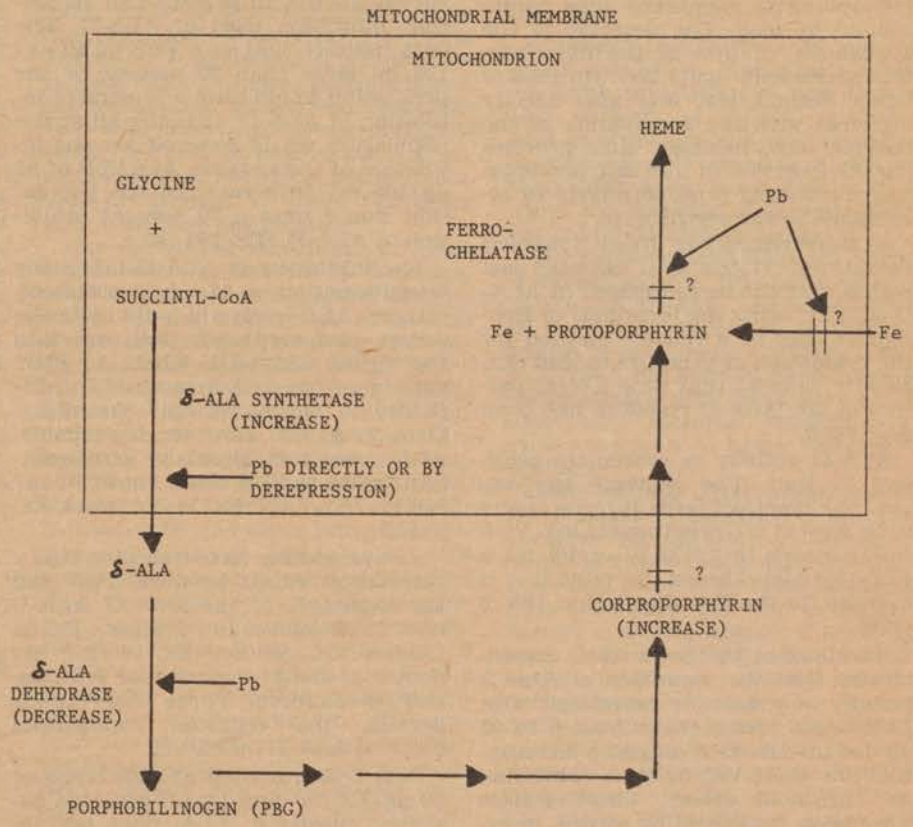
The foundation of our current understanding of the biochemical effects of lead on the hematopoietic system is a knowledge of the processes of red blood cell formation and a specific knowledge of the biochemical pathways of heme synthesis. Heme, a constituent of hemoglobin, is also an integral part of another group of important complex proteins, the cytochromes. These are the proteins of cellular oxygen transport which are located in the mitochondria of all cells. Inhibition of heme synthesis would therefore, not only be expected to affect the production of hemoglobin, but also have an effect on the production of cytochrome proteins. (Tr. 429).

The biosynthesis of heme is a multistep process. Several of the steps of the pathway, including the final step, occur in the mitochondria. In order to appreciate these signs of lead poisoning, a detailed understanding of the biosynthesis of heme and its biological functions is required. The first step in the sequence of reactions leading to the synthesis of protoporphyrin, the immediate precursor of heme, is catalyzed by the enzyme, aminolevulinate synthetase. In this reaction, glycine and succinyl Coenzyme A are converted into 5-aminolevulinic acid (ALA), which is subsequently converted into the ringed structure known as porphobilinogen by a second enzyme, 5-aminolevulinic acid dehydrase (ALA-D).

Porphobilinogen, in a series of reactions (one of which is governed by the enzyme coproginase) is eventually converted into protoporphyrin. In a final step, the enzyme ferrochelatase catalyzes the insertion of iron from ferritin into the protoporphyrin ring to form heme.

At least two of these steps are considered to be directly inhibited by lead. These steps are; (1) The transformation of ALA into porphobilinogen, catalyzed by ALA-D, and (2) The insertion of iron into protoporphyrin, catalyzed by ferrochelatase. Other steps in the process of heme synthesis are also affected by lead, such as A-ALA synthetase and coproginase. However, these effects may only result from feedback depression, rather than from a direct effect of lead. (See figure 1) (Tr. 433-34; EPA Criteria Document, p. 11-10)

FIGURE 1.



It is the third step in the pathway of heme synthesis which converts 5-aminolevulinic acid (ALA) into prophobilinogen. The enzyme mediating this reaction is δ-aminolevulinic acid dehydrase, (ALA-D); its activity is inhibited by lead. The effects of this inhibition can be observed and measured in two ways: (1) The activity of the enzyme ALA-D in the erythrocytes can be measured directly using a method developed by Bonsignore (Ex. 32(20), Ref. (1) in 1965, or (2) when the activity of ALA-D is inhibited, its substrate, ALA, builds up in the serum and spills out into the urine, (ALA-U). ALA-U is, therefore, a reliable measurement of the effect of altered ALA-D activity.

The final step in the heme synthesis pathway is the insertion of iron into protoporphyrin mediated by the enzyme ferrochelatase. This reaction occurs in the mitochondria. There are two possible mechanisms by which lead is considered to interfere with the transport of iron into the mitochondria. First, iron transport across the mitochondrial membrane may be inhibited by lead. The decrease in the availability of iron in the mitochondria necessarily limits the synthesis of heme. Second, lead may also directly interfere with the functioning of the enzyme ferrochelatase, thus preventing the insertion of iron into protoporphyrin, causing protoporphyrin to accumulate in the erythrocyte.

a. *Measurements of Heme Synthesis Inhibition.* (1) *ALA-D.* It has been suggested that the measurement of ALA-D activity, using the technique of Bonsignore may be a reliable method for the evaluation of exposure to lead (Ex. 32(20)), since at that time a clear pattern of the ALA-D response had been developed.

ALA-D activity is extremely sensitive to lead. The evidence suggests that the no-effect level, if there is any such level at all, is extremely low. Zielhuis suggests that it is 10 μg/100 ml, a blood lead level below the value that is average for the U.S. population. (Ex. 6 (179).)

Hernberg et al. (Ex. 6 (20)), demonstrated that the logarithm of ALA-D activity is negatively correlated with PbB levels over a range from 5 to 95 μg/100 ml. His data suggest a direct inhibition of ALA-D by lead, exhibiting no threshold effect. These results have been confirmed by several investigators. (Ex. 6 (118); Ex. 5 (22); Ex. 24 (Alessio).) Other studies have also suggested an exponential negative relationship between ALA-D and PbB. (Ex. 32 (20); (Ex. 23 (64).)

Tola (Ex. 5 (18)), has studied the response of previously unexposed workers to lead. A drop in ALA-D activity levels was observed after a few days of exposure. After 2 months, a new steady-State level of ALA-D was reached. From his data, Tola confirms the dose-effect relationship suggested in the preceding studies.

The relationship between dose (PbB) and effect (ALA-D) is well defined. Since individual variability is small (EPA Criteria Document, p. 11-99), and inter-laboratory measurements of ALA-D are comparable, more so than PbB's, (Ex. 294 (E), Ref. Berlin et al.) these factors do not obscure the relationship.

However, even though the relationship is a well defined one, there is some variability between individuals in the effect that will be observed for any given dose. Using Hernberg's data, Zielhuis has calculated dose-response curves for the 40 percent and 70 percent inhibition level of ALA-D. The data indicate that at a PbB of 40 μg/100 ml more than 20 percent of the population would have a 70 percent inhibition of ALA-D; virtually all of the population would have 40 percent inhibition of the enzyme. At a PbB of 50 μg/100 ml, 70 percent of the population would have a 70 percent inhibition of ALA-D. (Ex. 294 (E).)

The inhibition of ALA-D limits the transformation of ALA into prophobilinogen. ALA levels will build up in the serum, and eventually spill out into the urine, (ALA-U). There is little data on serum ALA because of the difficulty in measuring this parameter. Data on ALA-U, however, is available. ALA-U has been shown to significantly increase at PbB levels above 40 μg/100 ml. (Ex. 5 (9); Ex. 24 (Popovic); Ex. 5 (5).)

Several studies have indicated that a correlation exists between PbB and the logarithm of the level of ALA-U (Ex. 23 (Selander and Cramer); Ex. 24 (Alessio)). Chisholm (Ex. 99 (3)), has shown a similar exponential relationship in children. These observations parallel the reported exponential curve of ALA-D inhibition.

ALA-D is inhibited at PbB levels of 20 μg/100 ml and lower, but this enzymes' substrate, ALA, does not increase in the urine at PbB levels below 40 μg/100 ml. The discrepancy between the blood lead level at which increased ALA-U and decreased ALA-D activity can be detected is partially explained by two factors: (1) There is a larger variability in ALA-U measurements than in ALA-D; and (2) the definition of the normal range of ALA-U is based on controls with average blood lead values up to 40 μg/100 ml. These factors seem insufficient to explain such a large difference. It has been suggested that the different values may in fact indicate a reserve capacity of ALA-D activity. It is only after this enzyme reserve is used up that substrate would begin to accumulate. (EPA Criteria Document, p. 11-11; Tr. 454). Such a reserve capacity is also suggested by Zielhuis' finding that blood hemoglobin levels are not affected at a 30 percent inhibition of ALA-D (Ex. 24 (15), Ref. Zeilhus, 1974).

(2) *Protoporphyrin.* The accumulation of protoporphyrin in the erythrocytes of humans with lead intoxication has been known since 1933. (Ex. 105 (B) Ref. Van Der Bergh and Grotepass). However, until 1972 the technical difficulties associated with measurement of protoporphyrin limited its use as an indication of lead damage. The development in 1972 (Ex. 105 (G), Ref. Piomelli) of simpler and more accurate techniques of testing for free erythrocyte-protoporphyrin (FEP) has made this measurement clinically feasible. In 1974 Lamola and Yamane (Ex. 105 (B) reported that erythrocytic protoporphyrin is not actually "free" but rather chelated with zinc to form zinc protoporphyrin (ZPP). Fluorometric determination of ZPP is another technique which is now in use. (Ex. 105 (E).)

The accumulation of ZPP in erythrocytes in a chronically lead burdened individual is generally presumed to be due to the inhibition of the enzyme ferrochelatase which inserts iron into the protoporphyrin ring to yield heme. (Ex. 105 (C1); Ex. 105 (D); Tr. 434; EPA Criteria Document, p. 11-11). It has also been suggested that lead may have an effect on this step by interfering with the transport of iron across mitochondrial membranes. (EPA Criteria Document, p. 11-11 Tr. 434). By either mechanism, the result is the same; ZPP accumulates in formed erythrocytes of bone marrow and is carried by the circulating blood cells throughout their 120-day lifespan. (Ex. 105 (A); Ex. 105 (C1).) Due to its inhibited incorporation into

heme, excess iron can also be detected in the serum. (Ex. 294 (E).)

The pattern of the protoporphyrin response varies with sex and age. Stuik (Ex. 23 (Stuik)) observed a difference between the protoporphyrin responses of adult volunteers who were given lead acetate. These results have also been confirmed in an epidemiological study. (Ex. 23 (Roels, et al.).) For both groups, an exponential correlation was found between PbB and protoporphyrin (Ex. 23 (Roels, et al.)), similarly found for adults in other studies. (Ex. 24 (Alessio).) A similar exponential response, but at lower PbB levels, has been found in children (Tr. 443; EPA Criteria Document, Ch. 11, Ref. 147; Ch. 11, Ref. 148; op. cit., Ch. 11, Ref. 149).

The concentration of ZPP in the erythrocytes starts to increase at PbB levels of about 20 to 35 μg/100 ml in children, 20 to 35 μg/100 ml in adult women and about 30 to 40 μg/100 ml in adult men (Ex. 23 (Stuik); Ex. 294 (E); Ex. 105 (E); Ex. 24 (Popovic); Tr. 454).

Zielhuis has calculated dose-response curves using Roel's data for adults. At a PbB level of 30 μg/100 ml, 60 percent of the women and 10 percent of the men had FEP levels above "normal" (80 μg/100 ml), while at PbB levels of 40 μg/100 ml, 100 percent of the women and 30 percent of the men were above normal. (Ex. 294 (E).) Piemelli studied 2,000 children and found that at PbB levels of 35 μg/100 ml, more than half had significantly elevated protoporphyrin levels. (Tr. 440.) The duration of exposure has also been positively correlated with protoporphyrin levels by Tomokuni et al. (Ex. 6 (161).)

Elevated protoporphyrin levels persist in the circulating erythrocyte long after the cessation of lead exposure, thus the correlation of protoporphyrin to PbB cannot always be expected to be as close as that of ALA-U to PbB. However, during periods of steady exposure to lead, protoporphyrin levels correlate closely to PbB (r=.91). (Ex. 5 (15), Ref. 17.) Variation in protoporphyrin level due to an iron deficiency, as well as normal variations in PbB level measurements, would also obscure the relationship.

When evaluating the correlation of PbB and protoporphyrin levels, it should be noted that the former is a measure of a dose, while the latter is the measure of an effect. Measurements of an effect, such as ALA-U or ZPP, are expected to be somewhat variable due to the inherent biological differences between individuals. PbB is a measurement which has been available and studied for many years. It is for this reason that we evaluate newly developed biochemical measurements, such as ZPP, in terms of their relationship to PbB. (Tr. 446.) Work by Chisholm (Ex. 99 (B)) and Sassa (Ex. 5 (15) Ref. 17) has indicated that FEP is a better measure of soft tissue lead level than is PbB. The Center for Disease Control has concluded that FEP is a better index of potential toxicity from the body's lead burden and that it provides a better estimate of soft tissue lead. In situations where a child has a low PbB level and a higher than expected metabolic effect (i.e., ZPP level) for that PbB level, the child is considered to be at greater clinical risk. (Tr. 447; Ex. 5 (15).) Children with PbB levels greater than 30 μg/100 g, FEP levels greater than 100 μg and who show symptoms compatible with lead poisoning "should be considered as having poisoning and recognized as candidates for urgent inpatient medical management". (Ex. 5 (15).) Thus a child with a blood lead level as low as 30 to 40 μg/100 ml could be considered to have lead poisoning if the measure of metabolic effect was sufficiently elevated. As was previously discussed, children show effects, such as protoporphyrin elevation, at a somewhat lower level than do adults. Nevertheless, it is important to note that a general pattern is presented; a given blood lead dose may very well have a different metabolic effect on different individuals. A sensitive person may suffer extensive metabolic disturbance at levels well below that of the average person.

The increasing disruption of heme synthesis as measured by increased protoporphyrin has been studied in relation to the occurrence of clinical symptoms of lead poisoning. In children the statistical likelihood of clinical symptoms and permanent damage increases arithmetically with FEP levels greater than 60 μg (Ex. (15)). Fishbein has studied the relationship of ZPP to other symptoms and metabolic effects. Correlation coefficients between ZPP and other laboratory parameters such as hemoglobin, BUN and creatinine, were found to be better than those between the same parameters and PbB. This pattern was specifically found in a population of workers with PbB levels below 80 and no history of chelation therapy (Ex. 105 (E)). An increase in ZPP was found to be positively correlated with various symptoms, such as loss of appetite, weight loss, muscle and joint pain. The prevalence of lead colic increased markedly with elevated ZPP levels. This correlation between ZPP level and clinical symptoms suggests a causative relationship between these biochemical effects and clinical symptoms.

The inhibition of ALA-D and iron incorporation are the most well-studied effects of lead on heme synthesis, although the following effects have also been documented. An increased excretion of coproporphyrin in the urine (CPU) by lead workers and children with lead poisoning is known to occur. (Ex. 5 (5); FEIA EPA Criteria Document, p. 11-13), and CPU measurements are used as an indicator of lead poisoning. It is not known, however, whether this effect results from specific enzyme inhibition or from upstream accumulation of substrate secondary to inhibition of iron incorporation, or both. Alternatively, it has been suggested that lead may directly effect the transport of coproporphyrin across the mitochondrial membrane thereby causing this excess coproporphyrin to be excreted in the urine. (EPA Criteria Document, p. 11-13.) Increases in CPU become measurable at PbB levels of 40 μg/100 ml. (Ex. 96.) Another lead-induced effect on home synthesis has also been observed; ALA—synthetase activity is increased during lead exposure. It is theorized that this effect may be a result of derepression of negative feedback controls. (EPA Criteria Document, Ch. 11, Ref. 171.)

b. *Health implications of heme synthesis inhibition.* The effects of lead on heme synthesis are not disputed. The pattern of ALA-D inhibition, ALA-U excretion and protoporphyrin buildup are well established. However, there has been considerable debate about the meaning of these biochemical and physiological changes.

The proposed lead standard originally suggested that "the point at which subclinical changes become sufficiently serious to represent a threat to health is not clearly defined." (Ex. 2, p. 47735.) Subsequent testimony has demonstrated that there are two fundamentally different understandings of the meaning of heme synthesis inhibition.

Some experts have suggested that the changes in biochemical and physiological parameters are manifestations of homeostatic adjustments to lead. It is implied by these arguments that the body has the capacity to handle a certain degree of lead exposure. Only when the lead dose is large enough to overcome these reserve capacities does the impairment of health occur. Such impairment is referred to as the "clinical" effect of lead. Williams (Tr. 1886) has stated that these biochemical changes are "* * * one of the many thousands of homeostatic mechanisms of the body whereby the effect of an alteration in the external environment is fully compensated by a biological response." Malcolm has stated that "I would submit that so far we have no clear evidence that the suppression of enzyme activities in the blood formation causes any departure from the normal well being unless it causes a fall in the hemoglobin level.

The best way of assuring that this does not happen is to carry out periodic hemoglobin estimations. Changes in enzyme system, in my view, may best be described as biochemical effects of lead and not as subclinical poisoning." (Tr. 2106-2107.)

Finally the Lead Industries Association (LIA) has asserted that, "neither increased ALA nor inhibited ALA-D, nor increased ZPP nor inhibited ferrochelatase is per se harmful. These changes by themselves do not indicate interference with heme production. In fact, the opposite may be true, since there are a number of feedback mechanisms in the biosynthesis of heme which allow the body to compensate for these changes." (Ex. 335, p. 22.)

Other experts have a fundamentally different understanding of the implications of the early biochemical and physiological changes caused by lead. This group rejects the idea that these changes are manifestations of "homeostatic mechanisms", rather, they consider these as the first cellular changes that eventually lead to traditional lead poisoning.

These health effects are considered to be important because they are a measurement of disruption of fundamental cellular processes. This disruption is considered to eventually result in anemia, and also may be related to the development of clinical effects in other tissues.

Piomelli (Tr. 471) discussed the point at which the health effects of lead are considered to occur. He stated that, "I believe personally that the most important issue is to assess objective health effects. And objective health effects like for instance those in heme synthesis which are not at all related to the feeling of the individuals in the case of poisoning. Of course everything in life as I am sure you are familiar happens in a continuous curve and therefore it is a continuous progression and that the earliest indication of poisoning in my opinion is this metabolic effect." (Tr. 471.) He further stated that:

"I would consider that if somebody has any evidence of impairment of heme synthesis caused by lead, this is evidence that lead is interfering with their body function." (Tr. 472.) Teitelbaum has described these metabolic changes as the "obvious laboratory evidence of excessive lead absorption." (Ex. 56, p. 11.) He has described individuals with excessive lead absorption as

> those who have no disease, but who have lead effect demonstrated by metabolic abnormalities which are the stalking horses of future lead intoxication. This group of patients is not lead poisoned in the traditional sense. No physician could, on a clinical basis alone, make the diagnosis of lead intoxication in them. Without sophisticated laboratory studies these individuals would not be recognized as poisoned because they have no obvious clinical findings. However, they have obvious laboratory evidence of excessive lead absorption. (Ex. 56, p. 11.)

OSHA agrees with Piomelli and Teitelbaum and believes that to explain away the subclinical effects of lead exposure as manifestations of the body's reserve capacity seems to be overly simplistic. The complex pattern of lead's effect on heme synthesis does not seem to fit into such a model. The evidence indicates that a worker with a pattern of substrate build-up may eventually suffer overt lead intoxication. Enyzme inhibition is observed at very low PbB levels. For example, it has been established that ALA-D begins to be inhibited at blood lead levels as low as 10 μg/100 ml. Increasing enzyme inhibition leads to the accumulation of substrates which can then be directly measured in the urine and blood. Increased excretion of the substrate ALA-U becomes manifest at PbB levels as low as 40 μg/100 ml for adult men, and increased protoporphyrin is excreted at PbB levels of 25-35 μg/100 ml in adult women and 30 to 40 μg/m 100 ml in adult men. As blood lead levels continue to increase, the enzyme inhibition and resulting substrate accumulation becomes more pronounced.

It is generally accepted that this pattern of increasing inhibition and physicological disruption eventually leads to the clinical symptoms of lead poisoning. Correlations between various clinical symptoms, such as anemia, and physiological changes, such as ALA-D and ZPP, have been found. (Ex. 81 (C); Ex. 105 (E)) Piomelli has discussed these effects stating:

"What is the clinical significance of the effects of lead on heme synthesis? These effects occur in the blood as well as in all other tissues. The inhibition of heme synthesis in the blood forming marrow ultimately leads to anemia which is one of the known symptoms of severe lead intoxication. However, this is not, in my opinion, the most significant clinical effect of heme synthesis, because we know in pediatrics as well as in occupational medicine a severe clinical neurological toxicity may occur much before anemia develops. A much more important fact is that the alteration of the mechanism of heme synthesis reflects the general toxicity of lead in the entire body." (Tr. 458).

It is important to note that for each clinical step in the development of lead-induced disease, there is a degree of variation between individuals as to when a symptom becomes manifest. Each observable symptom is the result of several complex biochemical alterations which are involved in this multistage disease. Individual variation results in a broadening of the range of PbB levels at which various clinical symptoms will develop. In this context, the ability to observe and control biochemical changes is important in order to prevent the progression to overt lead poisoning. Teitelbaum has discussed the problem of individuals who have lead related effects, ("i.e., metabolic abnormalities but no clinical disease") stating, "They show evidence of interference with normal red blood cell manufacture, and interference with normal nerve conduction times and other enzyme systems, which are intimately involved with the maintenance of the human homeostasis. If these abnormalities are ignored in a planned attempt to wait for overt disease, surely no preventive medicine is being practiced!" (Ex. 56, p. 11)

OSHA believes that this standard must be based on the most thorough understanding of the disease process possible. It is the belief of the Agency that the preponderance of evidence indicates that there is a continuum of lead effects, starting at the level of enzyme inhibition, progressing to measurable heme synthesis disruption, and eventually resulting in the established clinical symptoms of lead poisoning. These initial effects must be considered as the early stages of a disease process, not as simply the manifestations of homeostatic mechanisms. The build-up of substrates such as ALA and protoporphyrin is a measurable manifestation of lead's effect on heme synthesis at PbB levels as low as 30 to 40 μg/100 ml. This impairment of fundamental and vital subcellular processes can be demonstrated in a substantial portion of the lead-exposed population. The synthesis of heme is vital not only for the transport of oxygen in the blood via hemoglobin, but also to the functioning of the mitochondria in all cells. OSHA considers that the existence of such a measurable metabolic change in this vital subcellular system over a working lifetime must be considered to be a material impairment of health. The definition of health is necessarily broader than the simple absence of clinical symptoms. Current medical science has given us an understanding of the early stages of the lead disease process, and this knowledge must be applied. Given our current understanding of the continuous nature of lead disease, the promulgation of a standard based exclusively on the prevention of immediate clinical symptoms would be a dereliction of the Agency's duty to provide "the highest health and safety protection for the mployee". When continued exposure will eventualy result in serious disease symptoms, prudent policy requires that the early stages in the disease process be avoided.

c. *The Hematopoietic System.* In addition to the effect of lead on the synthesis of the heme radical itself, hemoglobin production may also be impaired by lead due to an inhibition in the synthesis of the protein portion of the molecule. Kassenar, et al. (EPA Criteria Document, Ch. 11, Ref. 174) and Wada, et al. (EPA Criteria Document, Ch. 11, Ref. 170) have shown that lead impairs hemoglobin synthesis in vitro in human reticulocytes at concentrations corresponding to PbB levels of 20 μg/100 ml.

Studies have revealed that accumulation of non-heme iron in developing erythrocytes in the form of ferruginous micelles which are responsible for the ringed sideroblasts characteristic of lead poisoning and dense aggregations of ferritin in damaged mitochondria. (Ex. 95, pp. 101-103) These accumulations of altered cytoplasmic and nuclear remnants (RNA and altered microsomal and mitochondrial fragments), result in the basophilic stippling of red blood cells. Basophilic stippling is an inconsistent feature of gross lead poisoning. However, microscopic examination of aspirated bone marrow usually reveals stippled sideroblasts in classic lead poisoning. Basophilic stippling has been observed in individuals with PbB levels below 70 μg/100 ml. (Ex. 6(37)).

In addition to affecting hemoglobin synthesis, lead also affects the life span of the circulating erythrocytes. In chronic lead poisoning the shortened life span is moderately severe, although it is much less than that experienced in most hemolytic anemias. According to several studies, survival time is not shortened by more than 60 days. Several investigators (Ex. 24 (Nordberg), Chap. 6, Ref. Rubino; op. cit., Ref. Sroczynski; Ex. 95. Ref. 249) have demonstrated a slight shortening of the life span of erythrocytes (100 days) in lead workers.

The mechanism by which lead shortens the erythrocyte life span is not known. (Ex. 5(5), Ref. 220; Ex. 5(5), Ref. 567; Ex. 5(5), Ref. 240). It has been shown that the osmotic resistance of the erythrocytes of patients with lead poisoning is increased. (Ex. 5(5), Ref. 220; Ex 5(5), Ref. 567; Ex. 5(5), Ref. 240). Increased mechanical fragility has also been reported. However, studies dealing with these phenomena are equivocal. (Ex. 5(5), Ref. 242; Ex. 5(5), Ref. 567).

It is known that lead interferes with a number of membrane functions (Ex. 5(5), Ref. 436). Lead is known to inhibit the activity in Na/K ATPase, an enzyme whose function may be related to the membrane cation pump. Lead has been shown to inhibit this enzyme both in vitro and in vivo. (Ex. 5(23), Ref. 4) Secchi et al. observed a significant negative correlation between PbB levels and the Na/K ATPase activity of erythrocyte membranes. (Ex. 5(23)). Using Secchi's data, Zielhuis has calculated a dose-response relationship. The coefficient of correlation between PbB and Na/K ATPase was low, however, sensitivity and specificity were poor. The study indicates that at rather low PbB levels (30 μg/100 ml) about 50 percent of subjects had moderate inhibition of Na/K ATPase (Ex. 294 (E)).

Roels has (Ex. 294 (E), Ref. Roels et al., 1975), established a negative correlation between PbB and reduced glutathione (GSH), a tri-peptide associated with red blood cell integrity. Zielhuis calculated the dose-response curve using Roels' data. Sensitivity and specificity to PbB levels were rather poor. A slight reduction in GSH was first observable at a PbB level of 30 μg/100 ml. (Ex. 294(E)).

It is not known whether these changes in osmolality, ATP-ase activity or glutathione, are the cause of the shortening of erythrocyte life span but the evidence is suggestive that one or all of these changes may be involved.

A consequence of all these lead-related effects on hematopoiesis is that the production of erythrocytes increases. This is manifested by reticulocytosis which is an increase in the number of large, immature red blood cells. The degree of reduced cell lifespan correlates closely with the reticulocyte count. (Ex. 5(5), p. 110).

Given the dual nature of the mechanism of lead-poisoning anemia, the correlation between decreasing heme synthesis and increasing PbB level is to be expected, but would be obscured by the influence of other anemia inducing effects. Gibson (Ex. 6(59)) found a positive correlation between ALA-U and hemoglobin level (r=.57), and between urinary coproporphyrin and hemoglobin level (r=.54). Fischbein (Ex. 105 (D)) found that ZPP levels and hemoglobin levels were correlated. This evidence, in addition to that previously discussed, documents the shortening of erythrocyte lifespans, and presents a picture of the complex manner in which lead attacks the body on multiple biochemical fronts.

d. *Lead Induced Anemia.* Anemia is the medical term used to describe a condition marked by significant decreases in hemoglobin (Hb) concentration and in the number of circulating red blood cells. Exposure to lead is known to result in anemic disease of varying severity. Lead—induced anemia is mildly hypochromic and microcytic and in some cases, ringed sideroblasts have been observed and the reticulocyte count slightly increased. Basophilic stippling may be prominent, but is not always observed. Erythroid hyperplasia is observed in the bone marrow.

The mechanism of action of lead-induced anemia appears to be a combination of (1) decreased erythrocyte production due to the interference of lead with hemoglobin synthesis and, (2) a shortened erythrocyte lifespan due to damage by lead on the erythrocyte itself. (EPA Criteria Document, p. 11-7; Ex. 284(A); Weintrobe et al., *Clinical Hematology*).

While there is no disagreement that anemia is an established symptom of lead poisoning, there is disagreement as to the PbB level above which decreases in hemoglobin and hematocrit are observed. The proposal originally discussed these changes.

Cooper and Tabershaw observed increases in the proportion of workers with abnormally low hemoglobin concentrations at blood lead levels in the 70-99 μg/100 g range compared to workers with lower blood lead levels. Evidence of anemia was most prevalent in workers with blood lead levels above 100 μg/100 g. Observations by Tola. et al. of decreases in hemoglobin and hematocrit levels among workers entering occupational lead exposure for the first time in their lives at blood lead levels in the 40-60 range are also consistent with interferences in the hemoglobin pathway which may occur at blood lead levels of 40 μg/100 g with above. Additional instances of measurable decreases in hemoglobin levels among new lead employees are also available. While these workers may return to preemployment hemoglobin levels at a later date, these changes may still represent decreases in man's physiologic reserve caused by lead. (Ex. 2, p. 45937)

There are several factors which have confused the debate about the occurrence of anemia at PbB levels below 80 μg/100 ml. The first problem is variation in the definition of anemia. The clinical definition of this disease, based on levels of Hb and hematocrit, varies between experts. For adult males, Nelson (Ex. 5(19)) used a hemoglobin level (Hb) below 12.5g/100g to define anemia, Bell (Ex. 81(c)) used a cut-off point of 13g/100g, Fischbein et al., used 14g/100 g, while Wolfe defined anemia as hemoglobin less than 13 gm/100gm and hematocrit less than 41 percent. Others have based their evaluations of the occurrence of anemia on the measurable lowering of the Hb level over a period of time. (Ex. 5(37); Ex. 5(18); Tr. 1945, Tr. 1608). Definitions based on measurements over time are ideal since they depend upon a change in the level of Hb with increasing or continued lead exposure. When studies are done at a single point in time, it is necessary to set a hemoglobin level below which anemia occurs. Such a level is arbitrary, as it is impractical to draw a line in a continuous disease process.

A second factor which confuses the debate about the PbB level at which anemia occurs is the difference be-

tween the way that the various studies were designed. The use of particular cutoff points to define anemia and groupings of data by PbB levels make their results difficult to compare.

For example, Sakurai (Ex. 5(9)) studied 218 workers with lead exposure of less than 65 μg/100 ml. No decrease in Hb was found in workers with PbB levels of less than 50 μg/100 ml. Upon review of Sakurai's data, Cooper and Tabershaw (Ex. 6(37)) observed increases in the proportion of workers with abnormally low Hb levels and PbB levels of 70 μg/100 ml. In their analysis, they placed workers in groups according to PbB levels; 40, 40 to 69, 70 to 90, and 100+μg/100 mls.

Hemoglobin concentrations ranged from 10.9 to 18.0 g/100 ml. The mean was 14.9±072 ml. There was no significant correlation with age. There was a significant negative correlation with blood lead level (r=−0.220. p less than .01). Thus of the 56 employees (18.7 percent of those examined) with hemoglobin concentrations below 14 gm/100g, 28 were in the 114 employees with blood levels of 70 μg/100g or higher (i.e., 25.4 percent compared with 27 in the 183 with blood leads below 70 μg/100g (i.e., 14.7 percent).

Although when lead levels were below 100 μg/100g the mean hemoglobin concentrations were similar for all groups, the proportion with abnormally low levels was greater in both the 70 to 99 and the 100+μg groups. (Ex. 6(37), p. 523)

BLOOD LEAD CONCENTRATIONS (μg/100g)

	Less than 40	40-69	70-99	100+
Mean hemoglobin (gm %)	15.3	15.0	15.2	13.6
No. in this range	30.0	153.0	104.0	12
No. below 14 gm	6.0	36.0	31.0	13
Percent below 14 gm	16.7	19.0	23.0	52.0

It is important to note that the pattern of decreasing Hb vs. increasing PbB does not necessarily start at a PbB level of 70 μg/100 ml. The slight elevation in the proportion of individuals with lowered hemoglobin in the 40 to 69 μg/100 ml group is of particular interest if we accept Sakurai's conclusion that anemia is not detected at PbB's below 50 μg/100 ml. The grouping of data chosen by Cooper and Tabershaw is one which would tend to obscure an effect within the 50 to 70 μg/100 ml range, by diluting it with the unaffected 40 to 50 μg/100 ml group. It is also important to note that while the Cooper-Tabershaw data does indicate an increase in the proportion of individuals with lowered hemoglobin in the 50 to 99 μg/100 ml range, no decrease in the mean Hb was observed in the lower PbB groups may be a reflection of the individual susceptibility to lead-induced anemia.

Wolfe (Ex. 146(A)) reported an elevated incidence of anemia in adult males with PbB levels of 41 to 60 μg/100 ml. Lilis et al. (Ex. 24(14), Table 11) found 38 percent (8 of 21) of workers with PbBs of 40 to 60 μg/100 ml had Hb levels less than 13 gm. These studies confirm the previously discussed findings of lowered Hb in adult males in the PbB range of 40 to 60 μg/100 ml.

Studies of PbB levels and hemoglobin levels in children reflect similar patterns at relatively lower PbB levels than adults. Puschel observed a negative correlation between PbB and Hb in 40 children whose PbB levels ranged from 30 to 120 μg/100 ml. (Ex. 294(E), Ref. Puschel 1972). Others have found similar relationships in children. (EPA Criteria Document, Ch. 11, Ref. 100; Ex. 294(E), Ref. Betts et al. 1973: EPA Criteria Document, Ch. 11, Ref. 102) Studies involving hemoglobin levels in this group are affected by the high incidence of iron deficiency anemia. Nevertheless, these results do parallel those observed in adults. The occurrence of these effects at lower PbB levels in children than adults is to be expected.

In a study that evaluated the effect of lead exposure over time, Tola (Ex. 5(18)) studied 33 workers shortly after they became occupationally exposed to lead. After 100 days, the average blood lead was 50 μg/100 ml and the average Hb level had dropped from 14.4 gm to 13.4 gm. This data is particularly important as it demonstrates the decrease in Hb with increasing exposure to lead; it is not simply a correlation of PbB and Hb at one point in time. This data supports the opinion that lead-induced anemia is clinically apparent at PbB's as low as 50 μg/100 ml. It has been suggested that PbB levels in the cross sectional studies may have been higher at an earlier point in time. Tola's study design eliminates this criticism, while also confirming the results of the cross sectional studies.

e. *Special sub-groups at risk.* Given that exposure to lead may result in a wide range of health effects, from metabolic changes to the clinical signs of lead poisoning, it is to be expected that genetic variations between individuals would influence their response to lead. Some individuals may respond to a lower lead level than others. There are also various factors which have an effect on the hematopoietic system, which introduced together with lead may produce a synergistic effect, and thereby place certain individuals at greater risk. Piomelli discussed several genetic factors which might make a subgroup more vulnerable to lead, stating:

Genetic factors influence the variation in effects observed when several individuals are exposed to the same agent. We are largely ignorant of how these influence absorption of lead. However, a provocative study by Drs. McIntire and Engle has recently shown a significantly greater amount of lead in the blood of individuals defective in the enzyme glucose-6-phosphate-dehydrogenase, despite equal exposure to lead, in this case due to the proximity to lead emitting sources. This observation may appear superficially esoteric and irrelevant. However, it must be noted that this is the most common genetic metabolic defect, and that throughout the world more than 300,000,000 human beings are carriers of it; in this country, at least 2 to 3 million such carriers exist. This observation is at present too limited to warrant direct measures; however, it indicates the possibility that certain individuals may be congenitally more sensitive to the effects of lead. (Ex. 57, p. 20)

I also do not believe that it is necessary to have any special precautions for other individuals, like individuals who are carriers of the thalassemia trait, Mediterranean anemia, because I do not believe that at the present state of our knowledge there is any evidence that these individuals have a greater effect. (Tr. 463)

By contrast, along the same line, it is well established that in individuals with sickle-cell anemia the frequency as well as the severity of lead intoxication is greater than in the general population. These individuals may develop a peripheral neuropathy at much lower levels of lead. Sickle cell anemia is a very rare genetic disorder, which occurs in this country at a frequency of once every 8,000 births. Individuals with this anemia are usally aware of their disorder; however, it must be the responsibility of the employer to examine prospective employees and not to expose to lead individuals with sickle cell anemia. (Ex. 57, p. 21)

With reference to sickle cell trait as compared to sickle cell disease, Zielhuis has suggested that individuals with the trait "may be more affected than normal individuals by lead". (Ex. 294(E))

Okawa and Cromer of NIOSH (Ex. 234 (9)), did not find evidence of an interaction between the effects of lead and sickle cell trait. In a letter to the National Lead Industries they asserted that:

The question of whether lead exposure might have a more deleterious effect on individuals with sickle cell trait is an interesting one. Although this question cannot be unreservedly answered at this time, there are a number of facts which indicate that individuals with sickle cell trait are no more susceptible to the effects of lead than are individuals without the trait. These facts can be summarized as follows:

(1) The primary mechanism by which the hemoglobin molecule is affected differs in lead toxicity and in sickle cell trait. The effect of lead exposure does not enhance the fragility of the red blood cells, which is the major mechanism of anemia in sickle cell disease.

(2) Review of the medical literature on this subject gives no indication that lead produces greater deleterious effects in individuals with sickle cell trait than in those without the trait. However, this observation is by inference since no studies were noted

which addressed themselves to this particular issue.

OSHA agrees with these conclusions and for the purpose of this standard finds no evidence that workers with sickle cell trait constitute a susceptible group. OSHA concludes that there is no evidence to indicate workers with sickle cell are at greater risk from lead exposure than those with it.

Other nongenetic factors may also put various individuals at greater risk. Zielhuis, writing for the Amsterdam Conference, has stated that other environmental factors such as nutrition, chemical exposure, or use of drugs and alcohol might also result in a more extreme lead effect. (Ex. 262)

As mentioned previously, children are known to have anemic lead effects at lower PbB levels than adults. This effect may be related to the fact that iron deficiency anemia is more common in children. Women generally have lower Hb levels than men; the normal range of values for adult males is 14–18g/100 ml, while for adult females it is 12–16g/100 ml. This fact is attributed to the monthly iron loss in normal adult women. It is not clear, however, what this means in relation to lead's effect on women. With the exception of Roel's work showing that there is a greater elevation of FEP at a given PbB level in women than in men, and Zielhuis' review, (Ex. 24) there is very little work comparing lead's effect on adults of each sex.

To reiterate, anemia has long been known to be one of the clinical signs of lead poisoning. The occurrence of anemia above PbB levels of 80 μg/100 ml was presented in the proposal and was generally accepted. The proposal also mentioned data by Tola that were suggestive of anemia at PbB levels of 40 to 60 μg/100 ml. Following the proposal, there was considerable discussion concerning the occurrence of anemia below a PbB level of 80 μg/100 ml. Williams, Cole, and Malcolm testified that anemia was not found below a PbB of 80 μg/100 ml, while Epstein, Wolfe, and Hammond suggested that anemia is found below this level. (Tr. 1885; Tr. 3029; Tr. 2106; Tr. 1060; Tr. 4122; Tr. 229)

OSHA has concluded that the signs of anemia are best understood when they are placed in the context of a continuous disease process; the signs range from minor physiological changes to more extreme clinical symptoms. This understanding of lead disease was discussed at length in the preceding pages. This agency has found that lead-induced anemia may be the result of an adverse effect on heme synthesis, and/or erythrocyte lifespan. Therefore, it follows that there is considerable room for individual variability in the PbB level at which anemia presents itself. In addition, the measurement of hemoglobin levels is a continuous variable resulting in a wide range of values. These factors must be considered when evaluating the PbB level at which lowered hematocrit and Hb become significant in a population. The data of Sakurai, Cooper, and Tabershaw, Wolfe, and Selikoff et al., indicated that a measurable lowering of hematocrit occurs in adult populations in association with PbB levels in the range of 50 to 70 μg/100 ml. The longitudinal study done by Tola showing lowering of the Hb levels at a PbB level of 50 μg/100 ml provides very strong evidence that these effects occur at lower PbB levels than was previously accepted.

The incidence of anemia in the population must be understood not only in terms of a quantifiable lowering of the mean Hb level of a group, but also in terms of the effect that this lowered Hb has on particularly sensitive individuals. Certain subgroups, such as those with the sickle cell trait are relatively large, and the effects which they might suffer must be considered in the promulgation of this standard.

Based on the evidence of small but signfficant changes in Hb levels in the range of 50 to 70 μg/100 ml, and on a specific understanding that the effects of a substance on the more sensitive worker, as well as the typical worker must be considered, OSHA has concluded that there is sufficient evidence to demonstrate significantly lowered hemoglobin levels in populations with blood lead levels as low as 50 μg/100 ml.

The decrease in Hb levels in the lower PbB range is not so extreme that it would normally be considered cause for medical intervention. For an adult male, a lower Hb of 13g/100 ml is significantly, but not dangerously low, at least for the short term. However, when we consider the effects of lead exposure over a working lifetime, we must recognize that an individual with a PbB level of 50 μg/100 ml or above would have significantly lowered Hb levels throughout his/her working life. We have no data to indicate the long term health effect which may be caused from such a prolonged dearth of Hb. We do know, however, that this would result in a lifetime change in the oxygen carrying capacity of the blood, in the blood viscosity, and the cardiac work load. In the absence of specific epidemiological data, the Agency must use prudence and common sense in making a judgment concerning these possible lifetime effects.

OSHA, therefore, has concluded that the long term lowering of hemoglobin levels which is expected to occur within a population at PbB levels of 50 to 70 μg/100 ml, must be considered a significant health impairment, and is unacceptable. An exposure level must be set which assures that PbB levels for most employees will be below that range.

f. *Effects on Mitochrondrial Function.* As was previously discussed, the heme radical is not only a constituent of the hemoglobin molecule, but also a constituent of mitochondrial respiratory proteins called cytochromes. The effect of lead on heme systhesis must be evaluated not only in terms of anemia, but also with reference to the effects associated with the disruption of mitochondrial functions in all body tissues. The research on the mitochrondrial effects caused by inhibited heme systhesis is limited, but the implication of such disruptions are profound and must be evaluated. Piomelli directly addressed this question as follows:

> What is the clinical significance of the effects of lead on heme synthesis? These effects occur in the blood as well as in all other tissues. The heme synthesis in the blood forming marrow ultimately leads to anemia which is one of the known symptoms of severe lead intoxication.
>
> However, this is not, in my opinion, the most significant clinical effect of heme synthesis, because we know in pediatrics as well as in occupational medicine a severe clinical neurological toxicity may occur much before anemia develops.
>
> A much more important fact is that the alteration of the mechanism of heme synthesis reflects the general toxicity of lead in the entire body." (Tr. 458)

There is evidence of heme synthesis inhibition and mitochondrial disruption in other tissues of the body. Secchi et al (Ex. 5(22)), found a direct correlation (r=.67) between the levels of ALA-D inhibition in the red blood cells and in the liver. The PbB levels were in the range that used to be considered "safe", i.e. 16 to 56 μg/100 mls.

In his testimony, Piomelli used a study on the livers of rats who had been exposed to lead concentrations of 35 to 40 μg/100 ml. Lead granules were observed in the mitochondria by electron microscopy. (Tr. 459, Ref. Walton, 1973) He also testified that he was able to detect a disruption in the mitochondria of renal cells of a lead worker. Under the electron microscope, the mitochondria were found to be "bizarre and very broken." (Tr. 459; Ex. 32(30))

Millar et al. found parallel decreases in ALA-D activity in rat brain and blood tissues. The decreases in brain ALA-D activity, however, were only observed at a PbB level of 30 μg/100 ml or above. (Ex. 23(68), Ref. Millar). While this study was not statistically significant it is indicative of the effect.

Thus, the inhibition of heme synthesis as manifested by ALA-D inhibition and the disruption of mitochondrial function, as shown by electron microscopy, have been demonstrated in several body tissues.

This demonstration of subcellular effects of lead in two tissues that are known to be among the main target tissues of lead poisoning, the kidney and the brain, suggests that this lead-induced heme synthesis inhibition may be a part of the physiological mechanism by which these tissues are affected. Piomelli has specifically suggested that mitochondrial disruption resulting in altered cellular respiration may be a mechanism that explains some of the other medical effects of lead. He addresses the relationship between mitochondrial function and neurological effects:

The important adverse effect which lead exerts on every single cell is the decrease in synthesis of heme and its interference with mitochondrial function as ultimately, by these two mechanisms, lead inhibits the ability of each individual cell to respirate.

Different tissues are more or less sensitive to lack of oxygen. For instance, it is very well known that the brain and nervous system are the most sensitive to lack of oxygen, so much so that total loss of brain function may occur in an individual who has no respiration for as little as two to three minutes. This is a very well known effect on the entire organ, but it also occurs at the level of indiviual cells. The effects of lead on heme synthesis do not occur exclusively in the blood but they are seen in the entire body. Let me give you probably an even more dramatic comparison. For instance, when an individual is poisoned with cyanide, instant death may occur. This happens because cyanide attaches to the iron in the heme and makes it incapable of transporting oxygen. The death of the individual exposed to cyanide is not due to the effects on the blood but it is due to the effects of cyanide on the cytochromes in individual cells. Like cyanide lead is a general poison to the entire human body and it does not just affect the blood. (Ex. 57 p. 6)

Decreased respiration, in particular, may be deleterious to certain cells, particularly the neurons, the nervous system cells. As I have mentioned before, the central nervous system is particularly sensitive to lack of respiration; it is not therefore surprising that neurological and intellectual dysfunction should occur with lead intoxication. (Ex. 57, p. 19)

The work of Fischbein et al. provides reinforcing evidence for this theory. The level of ZPP, a measure of heme synthesis inhibition, was related to the occurrence of various lead related signs and symptoms. Central nervous system symptoms such as fatigue, nervousness, sleeplessness or somnolence, memory deficits and slowing of thoughts were studied.

The prevalence of central nervous system symptoms increased markedly with elevated zinc protoporphyrin levels, from 40 percent among those with ZPP levels less than 100 μg/dl to 71 percent of those with ZPP levels in excess of 100 μg/dl. It was also found that peripheral neuropathy was not associated at all with ZPP levels below 100 μg/dl.

Other symptoms considered were loss of appetite and weight loss, and muscle and joint pain. These two groups of symptoms followed the same pattern as the one described with regard to central nervous system symptoms. The relative prevalence of these three groups of symptoms was in accordance with what has been found in large groups of workers with significant excessive lead exposure. The highest prevalence is that of CNS symptoms, followed by muscle and joint pain and then by loss of appetite and weight loss.

Although there is currently no complete explanation of the mechanisms involved in the development of these groups of symptoms, the observations reported here of the correlation between zinc protoporphyrin levels and the prevalence of such symptoms is of considerable theoretical and practical interest. (Ex. 105(D), p. 4)

The relationship of ZPP and the occurrence of lead colic was also studied.

Considering the relationship between colic and zinc protoporphyrin levels, it is of interest to notice that history of lead colic was not given by workers with ZPP levels in the normal range (less than 100 μg/ml). The prevalence of lead colic increased markedly with elevated zinc protoporphyrin levels. (Ex. 105(D))

While an exact relationship is not set between ZPP level and PbB level, the correlation of Fischbein et al would associate the ZPP level of 100 μg/ml discussed above with a PbB level of about 50 μg/100 ml. Thus, the early appearance of these signs and symptoms would be loosely associated with a PbB range at least as low as 50 μg/100 ml. A pattern of biochemical and physiological inhibition resulting in some early clinical lowered hemoglobin levels in the PbB range of 50 to 80 μg/100 ml is paralleled by the manifestation at this PbB level of other clinical effects which may be related to heme synthesis disruption. Others have studied the relationship between PbB level and the occurrence of various symptoms. Beritic (Ex. 6(9)), found lead colic occurring in patients whose PbB levels were in the range of 40 to 80 μg/100 ml. Sakurai (Ex. 5-9) did not find an increase in symptoms in workers whose PbB levels were below 50 μg/100 ml.

The evidence relating the effects of lead on heme synthesis and mitochondrial function to the occurrence of clinical symptoms throughout the body is far from complete. However, due to the serious implication of this theory, it must be carefully evaluated. Evidence of changes in ALA-D levels or of mitochondrial changes associated with lead exposure has been found in both animals and humans in several different tissues. That lead causes the disruption of heme synthesis in renal, neural and liver tissue is well established. Lead is known to effect heme synthesis in these tissues in a manner which parallels that demonstrated in blood forming cells. The data of Fischbein et al., have demonstrated that an increased ZPP level is correlated with the appearance of a variety of lead symptoms and signs in other tissues. In this way, ZPP levels may be an indirect measure of the effects of lead on other systems, such as CNS.

Piomelli gave an excellent summary of the importance of lead's effect on heme synthesis, stating:

It is my understanding that regulations have the purpose of preventing material impairment of health.

Alterations in heme synthesis do not produce subjective evidence of impairment of health, unless they reach the extreme depression in severe lead intoxication, when marked anemia occurs and the individual feels weak. However, it is not any longer possible to restrict the concept of health to the individual's subjective lack of feeling adverse effects. This is because we know that individuals may get adjusted to suboptimal health, if changes occur slowly enough and also because we now have the ability to detect functional impairments by appropriate tests, much before the individual can perceive any adverse effect. In fact, it is the responsibility of preventive medicine to detect those alterations which may precede frank symptomatology, and to prevent its occurrence. The alterations in heme synthesis caused by lead fulfill, in my opinion, the criteria for material adverse effects on health and can be used to forecast further damage. The depression of heme synthesis in all cells of the body is an effect of far reaching proportion and it is the key to the multiple clinical effects of lead toxicity, which become obvious as the exposure continues. (Ex. 57, p. 21.)

This does not in any way suggest that the effect of lead on heme is the only mechanism of lead disease, but it does suggest that it is at least one of the more important ones. An appreciation of the wide range of lead's effect from subcellular changes to overt clinical symptoms, is relevant not only to the occurrence of anemia, but also to the expected pattern of lead induced neurological and renal disease.

In conclusion, OSHA believes that there is evidence demonstrating the impairment of heme synthesis and mitochondrial disruption in tissues throughout the body, and that these effects are the early stages of lead disease in these various tissues. The disruption of heme synthesis measured at low PbB levels is not only a measure of an early hematopoietic effect, it is also a measure which indicates early disease in other tissues. The Agency believes that such a pervasive physiological disruption must be considered as a material impairment of health and must be prevented.

3. *Nervous system.* Neurological and behavioral effects have long been recognized as severe consequences of exposure to lead. In the preamble to the proposed standard OSHA described some of these effects which range from acute and chronic encephalopathy, behavioral effects, severe damage to the peripheral nervous system (peripheral neuropathy), tremors, nervous irritability and early nervous system damage as measured by changes in motor nerve conduction ve-

locities. In its proposal, OSHA stated that there was little disagreement that risk of clear-cut clinical symptoms related to exposure increases as blood lead levels rise above 80 μg/100 g. The agency noted that there were a number of studies which have observed symptoms and effects caused by exposure to lead below 80 μg/100 g. A primary issue which the proposal and this final standard addresses is what levels of lead exposure are necessary to produce specific deleterious neurological effects and whether these effects represent material impairment as defined by the OSH Act. A related and critical issue is whether these effects are reversible and if so what weight should they be given in assessing their health consequences.

In addressing these issues in the proposal OSHA expressed concern with reports of early damage to the nervous system because of limited regenerative capacity in the nervous system. For this reason significant attention was given to the work of Seppalainen who had observed early stages of peripheral neuropathy in lead workers whose blood lead levels never exceeded 70 μg/100 g based upon slowing of the maximal motor conduction velocities of the median and ulnar nerves, and particularly the slowing of conduction velocity of the slower fibers of the ulnar nerve. In particular the preamble stated:

> The results of these studies show effects that were similar to, although milder than the nerve conduction changes seen in an earlier study of workers with clinical symptoms of lead intoxication. While some of the test subjects in the earlier study had a prior history of overt lead intoxication, this was not true in the later study.
>
> The fact that workers whose blood lead levels never exceeded 70 μg/100 g exhibit damage to the nervous system which is similar to, although milder than, that found in workers with a history of clinical symptoms and with higher blood lead levels, suggests that these milder effects are also significant. These data raise questions as to whether nervous system damage caused by increased lead absorption in the subclinical range is reversible and whether there is a continuum of damage of the nervous system in workers with blood lead levels below, as well as above, 70 μg/100 g. Based upon these data, Seppalainen concluded, "* * * that no damage to the nervous system should be accepted and that, therefore, present concepts of safe and unsafe blood lead levels must be reconsidered. (Ex. 2, p. 45935-36.)

There is extensive evidence in the rulemaking record which addresses neurobehavioral effects and peripheral neuropathy in lead-exposed workers at blood lead levels below 80 μg/100 g. There has been considerable debate regarding whether these effects do indeed exist and what is their significance. During the hearings industry representatives raised questions regarding primarily the adequacy of the methodology of some of the research on neurological disease and secondly questioned whether these effects constituted material impairment. Based upon the evidence in the record OSHA has concluded:

1. Since neurological disease must be recognized as a continuum of disease, it is axiomatic that the irreversible stage is preceded at the opposite end of the disease progression by an early, relatively mild, stage of disease. These early developmental stages of neurological disease are pathological states and OSHA finds persuasive the arguments for adopting a lead regulation which protects workers from these consequences of lead exposure. OSHA believes that the neurobehaviorial effects and the slowing of motor nerve conduction velocities (MNCV) do follow a dose-effect relationship and constitute material impairment. OSHA is convinced by the evidence in the record that those many workers who demonstrate slowing of MNCV and neurobehavioral effects may grow progressively worse from neurological disease and therefore must be identified and protected.

2. Both central and peripheral nervous system effects to be described in lead exposed workers appear to occur at blood lead levels as low as 40 μg/100 g. Therefore, OSHA believes the final standard should to the degree feasible maintain blood lead levels at or below 40 μg/100 g.

3. The methodology employed in the studies cited has received critical scrutiny by the scientific community and other interested parties. Many of the studies relied upon have been published in peer review journals which require critical scientific scrutiny before being accepted for publication. The volume and quality of the studies in the literature support the conclusions of the Agency.

a. *Encephalopathy.* Encephalopathy is the most serious form of lead poisoning. It may occur precipitously with epileptic-like seizures, followed by coma, and finally cardiorespiratory failure. In fatal cases, death ordinarily occurs within 48 hours of the first seizure, unless there is a life-support system available. At other times, it may be a more prolonged, fulminant form of encephalopathy in which the individual's state of consciousness vacillates between lucidity and stupor for about a week, and then during the final 48 hours, coma develops which eventually progresses to convulsions and death, (Ex. 95, p. 87)

Even in the absence of death or prolonged unconsciousness, it is now widely accepted that irreversible neural damage typically occurs as one of the sequelae of non-fatal lead encephalopathy episodes. Such permanent neural damage is reflected by signs of continuing CNS impairment ranging from subtle neurobehavioral deficits to severe mental retardation or continuing mental incompetence. What is not yet universally agreed upon, however, are the lead levels sufficient to produce lead encephalopathy and its sequelae.

Terminal lead encephalopathies have given pathologists an opportunity to examine autopsy material from the brain and spinal cord. Through these examinations it has been found that there is a generalized edema of the white matter in both the cerebrum and cerebellum. It is also quite common to observe swelling of the oligodendroghia and accumulations of PAS-positive globules in the perivascular glial cells. (Tr. 106.) At other times, there will be a diffuse atrophy of the gray matter associated with nerves containing fibrillary tangles, which may, in fact, be a reaction that is more directly attributable to the loss of oxygen. This may be due to alterations in the endothelial layers of the capillaries which, in turn, congest the area and shut off the blood supply. In fact, some neurologists consider lead encephalopathy to be a form of vasculopathy. (Final Environmental Impact Statement: Inorganic Lead; U.S. Department of Labor, Occupational Safety and Health Administration, April 1978 (FEIS), Ch. 11, Ref. 92 (later cited as EPA Criteria Document); *Ibid.*, Ch. 11, Ref. 198; Ex. 95, Ref. Cantarow, p. 117). In certain cases of encephalopathy, the center for muscular coordination—the cerebellum—is severely damaged and this could help to explain some of the unusual symptoms experience by the victims. These symptoms have been described innumerable times and begin with the victim's loss of memory, prolonged headaches, hyperirritability, visual disturbances, lack of muscular coordination, and hallucinations. With continued exposure, these symptoms may escalate into convulsions and coma. (EPA Criteria Document, Ch. 11, pp. 11-15; Ex. 95, p. 86; Ex. 95, Ref. Cantarow, p. 121). More permanent health effects resulting from severe encephalopathy may include impaired motor coordination, altered sensory perception, decreased learning ability, and shortened attention spans. In children, the effects are more drastic and include such diseases as severe mental retardation, speech defects, blindness, and cerebral palsy (Ex. 95, p. 97; EPA Criteria Document, Ch. 11, pp. 11-16).

Severe cases of encephalopathy are today so unique that they are typically utilized to characterize patients who have absorbed large amounts of lead in an extremely short period of time. Encephalopathy is usually noted in children with a markedly higher inci-

dence of severe symptoms and deaths occurring in them than in adults. The onset of the disease may be so rapid that the normal clinical manifestations, observed in milder forms of encephalopathy, may also be bypassed.

In general, where blood lead levels have been measured in adults with encephalopathy they ordinarily fall in the 80 μg and above range. However, in children, they have been found below this level. (Ex. 23(45); EPA Criteria Document, Ch. 11, Ref. 231; Ex. 95, pp. 88-98; EPA Criteria Document, Ch. 11, Ref. 208; Ex. 32(15); EPA Criteria Document, Ch. 11, Ref. 435). Such a varibility of blood lead levels, eliciting the same response, should not be surprising since it may simply be an indication of the differences in sensitivity of the child versus the adult. It could also be due to the lack of monitoring data available on PbB levels of adults suffering from encephalopathy. It is important to note that the neuropathologic findings as reported are essentially the same for adults and children. It has only been recently and through extensive study that the recognition of effects at lower levels in childen has been documented. OSHA is very concerned that the lack of evidence in adults may in fact be due to a lack of investigation rather than absence of disease. This may be true of all forms of disease, severe or mild, acute or chronic.

b. *Mild Encephalopathy.*

Historically the division between "severe" and "mild" forms of encephalopathy is mainly of diagnostic signficance for physicians in terms of survival and severity. Patients who are diagnosed as "severe" are usually suffering with epileptic-like seizures or coma—or some combination of both—and have been for more than 24 hours. By the same criteria, patients who are diagnosed as "mild" may still suffer with seizures and coma, but only briefly and without a serious impairment of consciousness. (Ex. 95, p. 87) Correspondingly, there is even a milder form of pathological damage which has been observed in studies of cerebrospinal fluid (CSF). For instance, the examination of CSF from patients suffering with mild encephalopathy, has been associated with a form of meningeal irritation. Since the presence of these cells is normally associated with an inflammation resulting from some source of irritation, it has been suggested that lead has caused the reaction in the meningeal coverings of the brain and spinal cord. (Ex. 95, Ref. Cantarow, p. 165) Coupled with the increased number of lymphocytes, some investigators have found an increase in the CSF pressure, as well as an increase in the lead content of the spinal fluid. (Ex. 23(10); Ex. 95, Ref. Cantarow, p. 136)

Symptomatic of mild encephalopathy are spells of dizziness, shortened attention span, insomnia, and verbal obstruction. Furthermore, patients may complain of forgetfulness and experience changes in their personality. (Tr. 146) Other clinical manifestations are also less severe, anemia and colic are not necessarily present.

In cases of mild encephalopathy the neurological symptoms have been found to remain. For instance, there may be cognitive impairment and profound behavioral changes in the individual which continue. (Ex. 95, p. 87)

c. *Behavioral changes.* One of the major issues addressed at the hearings was whether lower levels of lead exposure (30-80 μg/100 ml) effected neurobehavioral changes in apparently asymptomatic lead workers. That is, in the absence of clinical signs of lead encephalopathy, are there behavioral changes occurring which are manifestations of early neurological disease.

The effects of lead on worker behavior and performance was one of the main issues addressed at the hearings. While the record consists mainly of studies on behavior and reductions in performance levels usually coupled with a rather elaborate series of subjective symptoms, efforts to correlate neurological observations with other biological parameters, or with blood lead levels have been difficult. Earlier work on behavioral effects has been reviewed in detail and will not be repeated here. (Ex. 95, p. 157; EPA Criteria Document, Ch. 11, pp. 11-14; Ex. 262)

There is a growing body of evidence that exposure to lead at exposure levels which produce blood lead levels of 30-80 μg/100 ml in children effect neurological damage especially in the central nervous system. This evidence was the basis upon which the Center for Disease Control issued an updated statement on exposure of lead in children entitled "Increased Lead Absorption and Lead Poisoning in Young Children" in 1975. The document states:

> Lead has even more serious and largely irreversible effects on the central nervous system. It is manifested by severe acute encephalopathy at one extreme and relatively mild neurological disability and possibly hyperactivity at a lower level of exposure. (Ex. 32(15))

Based on this finding and others CDC recommended that the blood lead of children be less than 30 μg/100 ml and indicated that a child with a blood lead between 30-49 μg/100 ml would be considered to have a "minimally elevated" blood lead level requiring reductions of lead intake from all sources. While observations in children cannot be directly extrapolated quantitatively to adult workers because children may be more susceptible to lead, the qualitative similarity of mild nervous system damage in children at blood lead levels below those associated with overt toxicity in adults is worthy of note. In this context, the U.S. Public Health Service concluded for children that, "* * * the statistical likelihood of clinical symptoms and permanent damage increase at least arithmetically with confirmed blood lead levels above 30 μg/100 g. * * *" (Ex. 5(15), p. 3)

Zielhuis as early as 1972 recommended an individual limit of 35 μg/100 ml and a group average of 20 μg/100 ml for children. (Tr. 1078-79) While it is beyond the scope of this section to discuss the evidence relating to the significance of neurobehavioral effects at low lead exposure in children, suffice it to say that the evidence indicates neurological damage as manifested by behavioral deficits does occur in children exposed to lead levels below that set in this standard.

A number of studies suggest that permanent damage to the nervous system may have occurred in children only moderately exposed and in whom no overt symptoms of toxicity had appeared. These effects include behavioral problems such as hyperactivity, difficulty in task performance, deficiency in IQ and nerve conduction deficits. EPA has thoroughly reviewed the neurobehavioral effects in children exposed to low or moderate lead levels. (EPA Criteria Document) EPA concluded that "blood lead levels of 50-60 μg/100 ml are likely sufficient to cause significant neurobehavioral impairments for at least some apparently asymptomatic children". (EPA Criteria Document, Ch. 11, p. 11-20) OSHA agrees with this conclusion of EPA and will discuss the data in the section on reproductive effects. It is important to note that as recently as 10 years ago a blood lead level of 55-60 μg/100 g as compared to less than 30 μg/100 g was considered normal for children. Given the drop in acceptable levels for children as our understanding of the effects at low levels has increased, OSHA believes that a similar situation may be true for adults.

d. *Studies.* The record in these proceedings contain a number of investigations which demonstrate neurobehavioral effects in lead exposed workers whose blood lead levels ranged above and well below 80 μg/100 g. OSHA believes they should be reviewed here since their findings are both significant and in some cases controversial. At this point the blood lead level at which a claim of "no effect" can be made is unknown. In contrast to the hematopoietic system the neurobehavioral data is still incomplete with respect to establishing a threshold level.

In a paper entitled "Effect of Lead on the Central and Peripheral Ner-

vous System" prepared for the Amsterdam Conference (Second International Workshop on Occupational Lead Exposure, Reevaluation of Permissible Limits on Lead) in 1976, Seppalainen, Hanninen, and Hernberg reported on an investigation by Hanninen and Parland who studied 41 lead workers, most of whom had PbB levels in excess of 70 μg/100 ml. Blood lead data was available for 38 subjects and ranged from 60 to 150 μg/100 ml. These authors were able to demonstrate strong indications of CNS dysfunction in six of nine workers with blood lead levels below 70 μg/100 g. (Ex. 24(19)) Seppalainen describes the results of this study at the Amsterdam Conference.

The mean age of the workers was 32.5 years (range: 18-58 years). Healthy unexposed subjects of the same educational level and age range formed a control group (N=50), and two groups with CS_2 exposure (50+50), one of which comprised cases of poisoning, and the other "healthy" exposed workers, were used as additional reference groups. All subjects were examined with a large behavioral test battery including tests measuring intelligence, speed of performances, psychomotor functions and features of emotional reactions.

Compared with the control group (n=50), the lead workers were significantly inferior in two tests of intelligence and in several tasks demanding speed or control of psychomotor functions. In the personality test they showed less emotional reactivity but nevertheless more loss of rational control of behavior. Compared with CS_2 groups, the indications of CNS dysfunction were quantitatively milder than in the group with CS_2 poisoning but more severe than in the group with CS_2 exposure but without poisoning. Qualitatively, the lead syndrome differed from the effects of CS_2 by relatively more accentuated intellectual defects and disturbances of psychomotor control whereas the retardation of performance speed was less advanced. When a discriminant analysis was made between the four groups two of the three discriminant functions were relevant with respect to the differences between the lead group and the other ones. The first function discriminated the groups according to the severity of the CNS dysfunction, and the second function yielded an optimal separation between the lead group and the group with CS_2 poisoning. When each subject was classified to the group he was most likely to belong to according to his discriminant function values, 59 percent of the lead workers were classified into their proper group, 22 percent into the group with CS_2 poisoning, 12 percent into the "healthy" CS_2 exposed group, and 7 percent into the control group. When the sum of the probabilities for belonging to either one of the groups with most behavioral impairment (lead group and CS_2 poisoning group) was used as a measure of CNS dysfunction, there was no correlation between the dysfunction and the PbB levels. Six of the nine subjects with a PbB below 70 μg/100 ml had strong indications of CNS dysfunction. Although the causal connection between lead exposure and behavioral impairment could not be confirmed with certainty in these cases, this result was taken as a warning against considering PbB levels below 70 μg/100 ml as completely harmless. (Ex. 24(19), p. 7-8)

The study serves as an early indication that behavioral effects may be occurring at reduced blood lead levels.

Crockford and Mitran (Ex. 234 (21)) studied the ability of lead workers with slowed nerve conduction velocity to perform a number of psychomotor tasks as compared to a control group. The study was designed to determine if the performance of the lead exposed workers would be inferior to that of the control group. The population was the same as that reported by Lee and coworkers (See section on peripheral neuropathy). This study was limited to the employees who had demonstrated reduced motor nerve conduction velocities in an earlier study by Lee and coworkers. The authors sought to determine the significance of the MNCV reductions by studying the effects on the performance of specific tasks selected to demonstrate particular injury caused by lead absorption. Sixty-seven exposed workers and seventy-nine unexposed persons were tested in July-August 1976 as compared to ninty-four exposed and ninty-four controls studied by Lee over a 6-month period in 1975 and 1976. A battery of psychomotor tests including addition tests, maximum grip strength, reaction times to a visual stimulus and a small electrical stimulus, speed of tapping with a stylus, one hole test and a questionnaire, as administered to exposed workers and control. (Ex. 234 (21), p. 3)

The authors stated that "the results obtained do not indicate that a slowed nerve conduction velocity of the magnitude reported in these lead workers is associated with any decrement in performance. Nerve conduction velocity changes are therefore at the moment of doubtful value in determing exposure limits for lead. The changes in NCV are more likely to originate from training or homostatic mechanisms and are unlikely to indicate damage to the nerves." (Ex. 234 (21), p. 9)

OSHA believes this study is difficult to interpret and contains significant methodological weaknesses which make its findings questionable. First, there was a significant time lag between nerve conduction velocity measurements and psychometric testing. While the lag time might not invalidate the results the fact that the blood lead levels changed from a mean of 60 μg/100 ml in the NCV studies to 51μg/100 ml in this study raises serious question about the results. As stated above, the number of exposed workers (67) in the study by Crockford differed from those of Lee (94). This represents a decline of 27 subjects or 28 percent. Given the difference in experimental conditions it is doubtful that it is valid to compare results and conclude no correlation exists between slowed NCV and performance scores. Seppalainen for example has suggested the authors were at "a no-effect level at 51 μg/100 ml." (Ex. 319) Given the PbB levels and difference in number of subjects it is impossible to determine if the PbB levels were lower because fewer high exposed workers were involved or whether the PbB levels had actually dropped. In other cases these differences render the authors' conclusion meaningless.

The authors do not analyze the results of their clinical findings which appear to indicate significant differences between subject and control groups in terms of subjective symptoms. It is unclear why these authors ignore the clinical findings in these populations. There are significant differences between the exposed population and controls in the following areas: change in temper, sleep difficulty, headache, vertigo, numbness in arms and legs, blurred vision, any sight defect, and abdominal pain. The authors do state:

The exposed workers appear to show a preponderence of subjective complaints. The complaints however could be due to differences in the physical environment of the two groups and interpretation must wait on an investigation of this aspect of the respective working areas. (Ex. 234 (21), p. 8)

In OSHA's view these findings deserve further review especially given subsequent data developed by Fischbein et al. and reported in this section. For example, 13.4 percent of the exposed workers had constant numbness in arms and legs compared to 1.3 percent of the controls and 12 percent of exposed workers had occasional numbness compared to 6.3 percent. Since these are presumably the same workers with MNCV reduction careful followup is indicated.

OSHA must conclude from its analysis of the data that the results of this study are at best inconclusive. The authors' conclusions discussed above cannot be considered valid and the research must be viewed at this stage as a progress report which requires further study and evaluation. It is especially important to further evaluate the subjective symptoms to determine if they are related to lead exposure.

OSHA discussed the work of Repko et al. in the preamble to the proposed lead standard.

The data of Seppalainen agree reasonably well with those of Repko, Morgan, and Nicholson who studied behavioral measures of task performance among workers exposed to lead in storage battery manufacturing companies. While intellectual functions were unaffected by increases in body burden of lead, hand, sensory (hearing), neuromuscular or psychomotor (tremor, eye-hand coordination, muscular strength, and endurance and psychological (hostility, aggres-

sion, and general dysphoria) functions were all influenced by the body burden of lead. The strongest relationships between exposure and effects were found with tests of neuromuscular and psychomotor functions and major changes on the preferred side of the body were observed at blood levels between 70 and 79 μg/100 g. (Ex. 2, p, 45936)

At the public hearings Repko presented a reevaluation of his earlier work. Discussion was limited to the altered functional capacity observed in hand-eye coordination, tremor, strength, auditory acuity, and psychological well-being (mood or affect). He limited his discussion to those specific tests of functioning because "the analyses revealed that the clinical indicators bore no statistically reliable relationship to the types of behavioral functioning assessed through the use of the multiple task performance battery, the test of visual acuity and the digit span test." (Ex. 52, p. 6) Repko acknowledged that while certain measures of performance obtained from the lead-exposed employees suggested significant decreases in functional capacity with increases in PbB or decreases in ALAD there was no evidence which demonstrated differences when compared to the control group. Repko concluded that the failure to demonstrate differences was based on motivational differences between subjects and controls.

However, Repko stated a means to reevaluate the data:

> Recognizing that the utilization of the control data in this research has severe limitations where motivation is a factor influencing performance, or where differences in test behavior are suggested, or where antecedent noise exposure may affect hearing levels, the correlative changes in functional capacity exhibited by the lead-exposed workers must be regarded as more conclusive since the changes are related to biomedical indicators of exposure and effect. The tests involving visual acuity and auditory acuity are not motivationally dependent tests, whereas all other tests utilized in the study require some optimum efficiency in performance, especially in the absence of individual baseline data. (Ex. 52, p. 9)

Based on this finding, Repko disregarded the control data and carried out additional anaylsis of the data utilizing a univariate analysis of variance of the difference between workers with PbB 69 μg/100 ml or below to those with PbB 70 μg/100 ml or above. Analysis of the data led to the conclusion that the original conclusions were indeed correct and that these analyses demonstrate unequivocally that functional capacity is decreased in workers whose ALAD activity is approximately 90 percent inhibited or in workers exhibiting a PbB of 70 μg/100 ml or greater. "Thus measures of exposure, PbB, and of effect, ALAD, clearly delineate a cause and effect relationship between occupational lead exposure and decreased functional capacity." (Ex. 52, p. 10)

Serious methodological questions with these studies were raised during cross-examination by Dr. Lynam especially with respect to the lack of control booths for audiometric testing, the equipment failures during testing, lack of a history of past noise exposures, control group difficulties and problems of overlap between PbB groups and ALAD groups, since they were divided by different criteria. While not discussing these criticisms in detail OSHA has concluded that the concerns have merit and do raise questions with respect to the conclusions in this study. Although indications of behavioral effects do exist in this study OSHA cannot conclude that the data alone conclusively support a finding of behavioral effects at PbBs equal to or greater than 70 μg/100 ml.

More recently Repko et al. reported the results of a study in which 53 behavioral measures of sensory and motor function, 6 measures of nerve conduction velocity, 5 indices of inorganic lead absorption, a clinical electromyogram, a clinical neurological examination and demographic data were obtained from 85 workers from a storage battery industry and 55 controls who worked in a light manufacturing industry. The study groups were statistically identical in terms of age, height, and weight, although there was a slight difference in educational level. The mean blood lead level of the lead exposed workers was 46 μg/100 ml and the value was 18 μg/100 ml for the control group. There was nothing to indicate that stratification of the subjects was a factor that might invalidate any interpretation of the results and based upon the results of a psychological and social assessment the authors argue that the two groups represented a homogeneous population. The worker population had PbB which had been consistently below 80 μg/100 ml and were asymptomatic upon examination.

> Repko reported that "Differences between the two groups were evident in the NCV and behavioral measures. The lead-exposed workers showed a statistically significant lower conduction velocity in the magnitude of 5 to 9m/sec for the MCV of the median, ulnar, posterior tibial, and deep peroneal nerves. Also, the sensory conduction velocity (SCV) of the ulnar nerve was significantly slower for the lead workers; no differences in the conduction velocity of slower fibers (CVSF) of the ulnar nerve were noted. The results of the behavioral measures showed that deficits in visual reaction time, under response control of the ulnar nerve, as well as deficits in auditory functioning, in terms of both pure-tone thresholds and tone-decay, were all adversely affected by low-level absorption. No differences were noted in the strength, eye-hand coordination, or other psychological/social measures." (Ex. 422; Abstract)

A review of this study indicates that the methodological problems which plagued some of the principal authors' earlier work appear to have been resolved and were not apparent here.

Repko concluded: "It is clear from the present study data that the pure-tone thresholds of exposed workers are consistently higher for both the right and left ears at the frequencies tested. Of these differences in pure-tone threshold, 35 percent were statistically significant. These data are further enhanced by the results from the tone-decay test which demonstrate that at threshold and at 5dB above the threshold, the lead workers exhibited a greater amount of decay than nonlead workers. Normal functioning of the auditory system should not produce tone decay. The observed hearing loss is most probably sensorineural, although a central hearing loss cannot be eliminated completely. Sensorineural hearing loss may be attributed to various factors, including drug toxicity.

The second important behavioral finding relates to the visual reaction time test. Reaction times of lead intoxicated workers have been compared to those of non-exposed workers by various Soviet and Eastern European Scientists. Increased reaction times have been reported in leaded workers in response to spoken words or other auditory stimuli to visual stimuli, and to electrical stimuli. The results showing increased reaction times are consistent with findings noted in the literature. The particular motor response involved in the visual reaction time test requires control by the ulnar nerve. The ulnar nerve is the primary motor nerve responsible for lateral movement of the fifth finger. It is also quite interesting that a significant negative relationship was obtained between increases in reaction time and decreases in the maximal motor conduction velocity of the ulnar nerve.

Such findings are impressive, they provide important support to the notion that data derived from behavioral toxicology methods should be utilized in establishing the health status of groups of individuals regularly exposed to lead. The extent of which the neurobehavioral dysfunctions noted in this study and in other epidemiologic studies contribute to increased accidents must be investigated further. From the limited information gained in this study involving accidents, it can be said that the lead workers in the sample group did show significantly more accidents than the workers in the control group. It is clear, in summary, that lead exposure, even at PbB levels substantially below 80 μg/100 ml, may result in various interrelated neurobehavioral dysfunctions; the consequences of such dysfunctions are to detrimentally affect the performance of tasks or jobs involving motor responses." (NIOSH Technical Report, January 1978, Contract No. 210-75-0054; Ex. 422 (A), p. 63)

OSHA has reviewed this work and believes that the results are especially noteworthy with respect to the visual reaction time test insofar as behavioral effects and slowing of the maximal motor conduction velocity of the ulnar nerve are significantly related. In particular as Repko states: "The ulnar nerve is the primary motor nerve responsible for lateral movement of the fifth finger". (Ex. 422 (A), p. 63) This

direct relationship has not previously been reported in the U.S. and bears further investigation. The authors appear to have carefully designed these studies. The slowing of MNCV of the peripheral nerves are in agreement with work to be discussed later in this section. Given the growing body of evidence available to OSHA the Agency has concluded that reduced MNCV in these cases are a result of early peripheral neuropathy and as Repko and others have demonstrated these changes occur at PbB levels well below 80 μg/100 g.

During the hearings Lilis and Fischbein et al. testified on their extensive studies on lead disease among workers in secondary lead smelters. They reported the results of two studies, the first in two secondary lead smelters in Indianapolis, Ind. (Ex. 23(39)) and the second in Vernon, Calif. (Ex. 118(c)) In the first study 158 lead smelter workers were examined of which 113 worked in one plant and 45 in another. In addition, 24 workers without significant lead exposure from two other local plants were examined. The study protocol included a careful review of each individual's experience and history, a broad spectrum of laboratory tests, nerve conduction velocity measurements, reaction times, chest X-rays, and chromosome studies. With respect to CNS symptoms, the authors reported that tiredness, fatigue, nervousness, sleeplessness or somnolence, and anxiety existed in a large percentage of subjects and in addition reported finding other symptoms such as slowing of thought, memory deficits, errors in simple calculations and difficulty in symbol manipulations. A second group of symptoms with high prevalence (38 of 151) was loss of appetite and weight loss. Even for those with less than 1 year of exposure the prevalence was significant.

The third group of symptoms were muscle and joint pain and/or soreness. These symptoms were reported by 70 (46 percent) of 151 active workers. The prevalence of these symptoms increased with duration of exposure. In all three cases the prevalence of central nervous system symptoms correlated with elevated zinc protoporphyrin levels, which is a measure of the effect of lead on heme synthesis (see heme section). Also, the prevalence of symptoms was higher in those workers with elevated blood lead levels. The authors considered it noteworthy that each of the three symptoms followed a similar pattern, that is the prevalence of symptoms showed a marked increase after more than 1 year of exposure which indicates that it takes less than a year to develop these clinical symptoms of lead poisoning under the conditions of lead exposure.

Peripheral neuropathy manifested by weakness of extensor muscles of wrists and/or fingers was discovered in 19 workers. Six of the workers with extensor weakness had had more than 10 years of lead exposure whereas thirteen had had less than 10 years exposure. The blood lead level exceeded 60 μg/100 ml in 14 cases and in 7, the PbB was greater than 80 μg/100 ml. There was no instance of ZPP levels being less than 100 in those cases demonstrating peripheral neuropathy. Nerve conduction velocities (NCV) were measured on the radial nerve of the active extremity and the authors reported 16 out of the 19 workers with symptoms of peripheral neuropathy had decreased NCV's. The mean NCV for the 19 workers was 55.3/sec as compared to 60.1 m/sec for the control group.

In addition, nerve conduction velocity was measured on the radial nerve of the most active extremity and on the left peroneal nerve in 134 lead workers. 61 workers (46 percent) had reduced radial nerve conduction velocities, that is, an RNCV less than 55 m/sec. The prevalence of reduced radial nerve conduction velocities was 25 percent in the control population. While this value of 25 percent appears somewhat high for a control population it is not inconsistent when one considers that this control population was also subject to lead exposure. The authors considered these workers' lead exposure to be insignificant (soldering of food cans) but the fact remains that 10 workers (42 percent) had PbBs of 40–59 μg/100 ml. Seppalainen has found reduced nerve conduction velocities in two or more nerves in workers whose PbB was in this range (13 percent, 50–59; 7 percent, 40–49); 39 of the 61 workers with reduced nerve conduction velocities had a history of high PbB in the past but significant decreases in NCVS was found to occur in young workers as well suggesting early onset of neurological damage. (Ex. 5(13))

The authors conclude this study with the following summary: "A clinical survey of 158 workers employed in two secondary lead smelters in Indianapolis, Indiana (February 2–4, 1976) revealed a high prevalence of lead disease, with symptoms and abnormalities reflecting a wide range of lead induced changes, including central nervous system and gastrointestinal symptoms, muscle and joint pain, history of lead colic, repeated findings of elevated blood lead and low hemoglobin values. Elevated blood lead and zinc protoporphyrin levels at the time of the examination were common. Reduced nerve conduction velocity values were found in a significant proportion of examined workers.

These abnormalities occurred even after short (one year or less) periods of lead exposure. The concurrent finding of elevated blood lead and zinc protoporphyrin levels after similar durations of exposure confirmed the relatively rapid build-up of toxic lead levels. As expected, however, longer lead exposure was associated with greater prevalence of disease, and more severe abnormalities.

Zinc protoporphyrin levels showed better correlation with symptoms and other clinical or laboratory abnormalities than did blood lead levels, indicating that zinc protoporphyrin provides a more accurate index of biological response to lead exposure. Since ZPP is a measure of effect as distinct from a measure of absorption this result is not surprising. It is further indication that there is a relationship between health effects in one system influencing effects in another. The ZPP level in blood at a particular time reflects the lead levels at the site of erythropoiesis averaged over the preceding four months. The blood lead level has an equilibrium time of no more than a few days and reflects recent lead absorption.

It should be noted that the current study was limited in scope, with examination of only 158 smelter workers. Nevertheless, the findings call attention to central nervous system effects, peripheral neuropathy and renal damage as potential results of undue lead exposure." (Ex. 23(39), p. 98–100)

In a followup evaluation Lilis et al. described their findings in a subgroup of the previously described total population who had PbB of less than 80 μg/100 ml at the time of examination and who had never been notified that their blood lead level had been excessive and who had never received chelation. Central nervous system symptoms were found in 56 percent of the lead exposed workers. Once again the prevalence of symptoms correlated with ZPP. The prevalence of loss of appetite and weight was 15 percent as compared to 12 percent for controls, and the prevalence of muscle and joint pain and/or soreness was 39 percent. This subgroup of 48 with PbB levels not exceeding 80 μg/100 g had 26(54 percent) with a duration of lead exposure of less than one year and 18(38 percent) had been exposed for 1 to 3 years. The prevalence of CNS effects is striking given the brief exposures of these subjects. This must be assumed to demonstrate again early neurological damage at relatively low blood lead levels.

Nerve conduction velocity measurements indicated slowing in radial nerve of 20 percent of the lead exposed workers whereas the prevalence in the control group was 25 percent (5 of 20). This result is difficult to assess for two reasons: first the PbB of the controls were slightly elevated (42 percent between 40 and 59 μg/100 ml) and the control group had a much higher mean age (41.1 years versus 28.7 years in the lead-exposed workers). (Ex. 23(14))

Based on these findings OSHA concludes that these studies clearly demonstrate central nervous system symptoms in workers whose blood lead levels are below 80 μg/100 ml and represents definitive work in the study of the relationship between low level ex-

posure to lead and behavioral manifestations to date. OSHA is in general agreement with the author's conclusions:

In this study of lead-exposed workers whose blood lead levels were lower than 80 μg/100 ml and who had had no history of higher blood lead levels in the past, adverse health effects such as significant increase in ZPP levels, anemia, central nervous system symptoms, and muscle or joint pain were found with increased prevalence. This may be related, in some degree, to the rate of buildup of the lead body burden, a factor that has hitherto received little attention. Further, the data indicate that a blood lead level of 80 μg/100 ml is an inappropriate biological guide to control of occupational lead disease and is unsatisfactory if the main goal of medical surveillance, i.e., prevention of lead poisoning is to be achieved. It is clear that adverse health effects of lead occur below 80 μg/100 ml. It is also evident that the data here presented indicate that blood lead levels should not be allowed to exceed 60 μg/100 ml and that monitoring of lead-exposed workers should include ZPP determinations, which give a more sensitive estimate of biologically active lead than simple blood lead examinations, as they show a good correlation with clinical abnormalities. (Ex. 23(14), p. 265)

Dr. Lilis reported the results of the second study of a secondary lead smelter in Vernon, Calif. during the hearings. (Ex. 118(c)) This study was similar to that carried out in the two Indianapolis smelters.

After completion of the Vernon, Calif. clinical field survey of secondary lead smelter workers, a comparison of the findings in this group with those previously reported from the Indianapolis study was undertaken.

It was consistently found that symptoms related to lead toxicity were less prevalent in the Vernon group than in Indianapolis reflecting thebetter control of lead in the former case.

CNS symptoms were found in 64 percent of the Indianapolis workers and in 60 percent of the Vernon lead exposed workers, while in the control group they were present in only 20 percent of individuals.

Muscle and joint pain and/or soreness were also frequent symptoms of lead toxicity. They were reported by 46 percent of the Indianapolis workers as compared to 31 percent in the Vernon group; in the control (not lead exposed) group, only 11 percent had such symptoms.

Loss of appetite and weight loss are the third group of symptoms associated with lead toxicity, and such complaints were reported by 25 percent of the Indianapolis group as compared with 22 percent of the Vernon lead exposed workers, and only 11 percent of the control workers; 40 secondary lead smelter workers in Indianapolis (25 percent of those examined) had had one or more episodes of lead colic and 21 (19 percent) of the Vernon group gave a similar history. This did not occur among controls; 93 of the 158 lead smelter workers in Indianapolis (59 percent) had been notified of high blood lead levels in the past and 50 (32 percent) had had high blood lead levels on several occasions. For the Vernon lead smelter workers, the overall figures were similar: 56 percent had had high blood lead levels, but there had been less workers with repeated high blood leads. (21 percent).

When blood lead levels were found to be high, chelation therapy had been frequently used in the Indianapolis workers; 47 workers in plant 1 and 24 in plant 2 had been given such treatment and 45 (27 percent) had had repeated courses of therapy. In many cases, chelation therapy had been given without removing the worker from his usual lead exposures. Change in job assignment to areas of lesser lead exposure had been used much less frequently.

The situation in Vernon is different in this respect. Only 21 (19 percent) of those examined had had chelation therapy and only 6 (5.5 percent) had been given repeated courses of treatment. The practice had been to remove workers with high blood lead levels from areas with excessive lead exposure. Chelation therapy was not administered while lead exposure continued, but in most cases only after hospital admission.

Blood levels, at the time of the examination, were found to exceed 60 μg/100 ml in 21 percent of the Vernon group; only in one case was the level higher than 80 μg/100 ml.

This was much less than the proportion of workers who had been notified in the past of elevated blood lead (56 percent). In contrast to the findings in Vernon were those on the Indianapolis workers; 77 percent had had blood lead levels of 60 μg/100 ml or higher and in 29 percent the levels had exceeded 80 μg/100 ml.

Zinc protoporphyrin was also found to have a strikingly different distribution in the Vernon group, when compared to the Indianapolis group. While the proportion of workers with ZPP in the accepted range (less than 100 μg/100 ml) was more than 3 times higher in Vernon than in Indianapolis, the proportion of those with high levels, in excess of 200 μg/100 ml, was 30 percent in Vernon and 71 percent in Indianapolis reflecting the higher exposures in the latter case with the concomitant higher resulting effects.

Peripheral neuropathy, manifested by weakness of extensor muscles of the wrists and/or fingers in the most active extremity, was found in 23 (21 percent) of the Vernon workers. This finding was related to duration of lead exposure; 13 percent of workers with less than 10 years in the smelter were affected, while the proportion doubled in those with lead exposure of 10 to 20 years and was threefold in those with more than 20 years in the plant. The same relationship had been found in the Indianapolis group, and apparently indicates a dose effect relationship.

The results of the examination of 111 secondary lead smelter workers in Vernon indicate that excessive lead absorption and adverse lead effects were present in a smaller proportion of those examined that had been previously found in a similar group in Indianapolis.

In addition to the clinical evaluations to determine CNS symptoms, Dr. Jose Valciukas, also of Mount Sinai, carried out a battery of behavioral tests on 90 secondary lead smelter workers and 25 nonexposed steelworkers. These behavioral tests included the Block Design test (BD), the Digit-Symbol test (DS), the Embedded Figures test (EF); the Santa Ana Dexterity Test of the dominant hand (DH) and both hands (BH). Subjects were examined at random and examiners did not know whether the subjects were lead exposed workers or controls. In three tests there were significant differences, block design, digit symbol and embedded figures, and which were not related to differences in age or education between subject and control. Dr. Valciukas testified during the hearings on his findings and concluded the following:

(1) Three of the five performance tests (BD, DS, and EF) show according to Student's t-test a statistically significant dependence on ZPP with P ranging from .003 to .02. These tests have been successfully use in the assessment of brain dysfunction. The dexterity tests scores (DH and BH) are not significantly correlated with ZPP levels.

(2) For the BD, DS, and EF tests, scores are correlated with ZPP at P values at least a factor of 10 lower than those for blood levels. This can be understood in terms of the ZPP level representing a 4-month averaging of the lead burden for exposed individuals, as discussed above. It is consistent with the finding that several other lead-related symptoms in a lead-exposed population are better correlated with ZPP levels than with blood lead level.

(3) An observable correlation of the scores with ZPP persisted to fairly low ZPP concentrations (for instance, for EF P .1 for a subgroup having (ZPP) 170 μg/dl). The slopes of the fitted experimental curves suggest that the initial decreases in performance test scores were approximately 16, 7, and 5 percent per 100 μg of ZPP deciliter for the BD, DS, and EF tests, respectively; however, our sample size does not permit establishing statistical significance in this range.

(4) Although the correlation between scores and ZPP levels is statistically significant, the fitted curves have low accountability; that is, the scatter of scores due to individual variability greatly exceeds the effect that ZPP levels have on scores for the population studied here. It is then impossible to draw conclusions about an individual's ZPP

level or lead intoxication from his test scores alone.

This study is based on a group of workers whose blood lead and ZPP levels indicate that a portion of this population meet the clinical definition of lead intoxication. Erythrocyte porphyrin levels for the general (not occupationally exposed) population have been reported (9) to be in the range 20 to 60 μg of ZPP per deciliter. If the correlations are significant at such low ZPP levels, some degree of CNS dysfunction may occur not only in some lead-exposed workers but also in children living in lead-contaminated environments or in other groups with environmental exposures to lead (in water, food, or air). (Science *201*, 467 (1978).)

During cross examination Dr. Cole questioned whether job stratification may influence the results of behavioral testing (Tr. 2735) and suggested a less adept person might gravitate towards dirtier, less desirable jobs. In both Drs. Valciukas' and Lilis' professional opinions, job stratification was not an issue and given that Dr. Cole did not specify in more detail his concern, especially with respect to any particular test, OSHA has accepted the findings of the neuropsychologist as being valid.

An article entitled "Psychological Performance of Subjects with Low Exposure to Lead" by Haenninen et al., published October 1978 in the Journal of Occupational Medicine provides confirmatory evidence to the behavioral studies already discussed. OSHA believes this paper to be of fundamental significance in that the behavioral studies were carried out on subjects whose blood levels had never exceeded 70 μg/100 ml (mean blood lead level was 32± 11 mg (100 ml). The following tables demonstrate the results of the study:

TABLE 1.—*Performance of the Exposed and Reference Groups, Raw Scores*

Variable	Exposed group			Control group		
	(N=49) Mean		SD	(N=24) Mean		SD
Similarities	17.7	+	3.3	19.4	+	2.3
Picture completion (PC)	13.7	±	3.2	14.7	+	3.9
Block design (BD)	34.7	±	9.0	35.5	+	7.7
Digit span (DSp)	9.9	±	1.2	10.3	±	1.3
Logical memory (LogM)	10.8	±	3.4	12.1	±	4.2
Visual reproduction (Vis R)	9.9	±	2.5	10.2	±	2.7
Bourdon Wiersma, speed (BW sp)	33.6	+	6.8	32.9	+	6.5
Bourdon Wiersma, errors (BW err)	22.4	+	29.7	15.8	+	12.3
Benton, time (Ben time)	149	+	55	153	+	56
Benton, errors (Ben err)	2.76	+	2.12	1.91	+	2.15
Santa Ana, preferred hand (SA right)	45.6	+	6.1	46.3	+	7.1
Santa Ana, left hand (SA left)	42.1	+	5.5	41.5	+	6.8
Santa Ana, coordination (SA co)	30.1	+	5.2	27.4	+	6.2
Simple reaction time, preferred hand [1] (SRT right)	1,482	+	262	1,448	+	313
Simple reaction time, left hand [1] (SRT left)	1,385	+	261	1,310	+	251
Choice reaction time (CHRT)	1,711	+	208	1,739	+	226

[1] Cumulative time for 50 reactions in microseconds.

The authors conclude as follows:

In a study of the effects of low lead exposure on psychological performance, 49 exposed workers and 24 controls were given a psychological test battery. All the lead workers had been under regular monitoring during their entire exposure time, and only workers whose maximal blood lead concentration had never exceeded 70 μg/100 ml were included in the study. At the time of the examination, the mean blood lead level of the exposed group was 32+11 μg/100 ml. Comparisons were made both between exposed and nonexposed workers and within the exposed group. In the latter case, the maximal, the average and the actual blood lead concentrations were used as measures of uptake. The most important finding was a significant relationship between impaired psychological performance and lead uptake within the exposed group. The performances that were most affected by lead depended on visual intelligence and visual-motor functions. Age and neuroticism did not explain these relationships. The impairment of psychological performance correlated better with the average than with the maximal or actual blood lead concentration. Considering that no single blood lead concentration had ever exceeded 70 μg/100 ml, these findings indicate that the threshold for impaired performance lies below that level.

Although the impairment of performance found in these workers was mild, the results nevertheless demonstrate that some higher nervous functions are affected by rather low lead exposure. Slight peripheral nervous damage, evident as reductions in conduction velocities, also occurred in the same workers; this impairment also showed a relationship with lead uptake.[32] To what extent such early signs of peripheral and central nervous dysfunction can be regarded as significant enough to warrant reevaluation of the concept of "safe" exposure level remains a matter of judgment. However, a group of experts who met in Amsterdam in 1976 agreed, partly as a result of our preliminary findings on peripheral and CNS effects, that PbB levels should not exceed 60 μg/100 ml and that it was desirable to reduce individual exposure even below this level in order to protect the nervous system.[33]

The extensive research carried out at secondary smelters and reported by Lilis, Fischbein et al. was uncontroverted during the public hearings. Given the soundness of methodology and number of parameters evaluated the studies are without question some of the most significant investigations described during this rulemaking. The evidence of CNS symptoms in lead exposed workers at levels above and below 80 μg/100 ml is particularly striking. The data does not allow the development of a clear dose-response relationship with blood lead levels and therefore there is no clearly delineated no effect level with reference to blood lead levels. There is clear evidence for effects well below 80 μg/100 ml. The finding of significant correlations with ZPP demonstrates the advantage of this biochemical parameter as a biological indicator of long-term lead effects. These authors suggest that ZPP levels correlate better with symptoms of chronic lead toxicity than blood lead levels. The data presented in these studies appears to confirm this point of view. OSHA believes this research documents the existence of central nervous system disorders in lead exposed workers whose PbB levels were below 80 μg/100 ml. Dr. Lilis testified on this point during the public hearings:

MR. KUCHENBECKER. Based on your experience in these studies as well as your prior background, I wonder if you would agree with the conclusion reached in Amsterdam about a blood lead level above which no lead worker should exceed. Do you think that 60 is acceptable, or is there another level?

DR. LILIS. I understand your question. I would say that above 60 one may expect florid lead poisoning, full blown lead poisoning, so nobody should ever be allowed to reach a blood lead level exceeding 60. On the other hand, evidence has been accumulating that even at lower levels than 60, adverse effects are to be found, especially in regard to the hematopoietic system as shown by the zinc protoporphyrin tests which, as you well know, and I think everybody present here knows, now in men would increase at levels around 40–45, or between 40 and 50 anyway. In women, at even lower levels. Since zinc protoporphyrin reflects an adverse effect, and not only that, since zinc protoporphyrin, by our studies and some other studies, was shown to correlate very well with both symptoms and signs of lead poisoning, that indicates that even at lower levels when zinc protoporhyrin starts to go up, there is an adverse effect.

By the same token, I think, effects on the peripheral nervous system have to be considered and they have been shown to occur according to Seppalainen's work—we have no personal experience at such low levels, but I am going to come back to this point—that at levels around 50 micrograms per 100

cc's, you may expect changes in nerve conduction velocity.

Now I am going to come back to the point which I want to make, that we have not had an opportunity to examine any group of workers in which blood lead levels did not exceed 60 micrograms, so we cannot really attest to such findings as Seppalainen's, but we can say that we have seen zinc protoporphyrin to be a very good indicator for biological effects of lead and since zinc protoporphyrin starts going up at around levels of 40 or 45, that means that at those levels you already find something go wrong in the body. (Tr. 2700-02)

And later in her cross examination (SIC) by Dr. Lloyd she reinforced these views:

Dr. LLOYD. They said, "It was agreed that for male workers, individual blood leads should not exceed 60 micrograms per 100 milliliters, in the light of present knowledge available to the group. It is, however, desirable to reduce individual exposure below this level, taking into account the effect on the hematopoietic system at concentrations above 45 to 50 micrograms and on nerve conduction velocity at concentrations between 50 and 60 micrograms. Do you concur with that statement?

Dr. LILIS. Perfectly. (Tr. 2719-20)

OSHA agrees with these conclusions of Dr. Lilis that in order to prevent CNS effects in lead exposed workers PbB levels must be kept at or below 40 μg/100 ml.

(c) *Severe (chronic) neuropathy.* Lead workers with severe peripheral neuropathy are distinguished by extensive pathological changes in the motor nerves. Such pathological changes may include degenerative lesions of the anterior horn cells (motor neurons) of the spinal cord, with concurrent vascular congestion and hemorrhaging of the surrounding capillaries (Ex. 24(19), pp. 13, Ref. Campbell). This degenerative process may also produce fat globules, chromatolysis, and cell vacuolation in the motor neurons. It is also not unusual to see extensive demyelination and sclerosis of the posterior and lateral columns of the spinal cord, as well as thickening of the meningeal covers of the cord.

Projecting from the spinal cord are bundles of nerve fibers that are also damaged as a result of lead poisoning. These nerve fibers show some very specific effects, such as swelling of the axis cylinders, segmental demyelination, and Wallerian degeneration which eventually results in fiber atrophy. Moving away from the spinal cord and into the extensor muscles of the arms and legs, it is also quite common to see the associated pathological changes in these muscles. (Ex. 95, Ref. Cantarow, pp. 65; Ex. 24, (19); Tr. 109; Ex. 95. pp. 89.)

Related to the pathological alterations may be a progressive loss of strength in the extensor muscles to the point of total paralysis. Because such extensor paralysis does consistently appear in the muscles controlling the hands and feet, it has become synonymous with lead poisoning. (Ex. 95, Ref. Cantarow, pp. 126) (Ex. 6, (58).) One of the more typical examples of this phenomenon is radial palsy which as its name implies, results from damage to the radial nerve. It is more commonly referred to as "wrist drop" because the loss of extensor muscle contraction permits unopposed contraction of the flexor muscles, and finally the hand bends or "drops" at the wrist. Workers suffering with radial palsy may experience unilateral or bilateral paralysis, that may result in irreversible atrophy of the etensor muscles. (Ex. 95, Ref. Cantarow, pp. 126.)

If lead exposure continues, and the lead poisoning is not treated or exposure reduced, weakness may eventually extend throughout the arm or leg. Prior to the development of paralysis, workers have been known to complain of hypersensitivity over the affected area, sensations of heaviness in the limbs and finally painful cramping in the muscles (Ex. 95, Ref. Cantarow, pp. 127). Exaggerated tendon reflexes, accompanied by prolonged muscle tremors, may also precede the paralysis and may be caused by the weakening of the muscles or by progressive degeneration in the nerves.

Recent advances in experimental neuropathology have made posssible classification of peripheral neuropathies based on histopathological reactions of the peripheral nerve. However, in spite of extensive studies it is as yet unresolved whether lead induced neuropathy is primarily due to a metabolic derangement of Schwann cells (demyelinating neuropathy), of neurons (cytons and/or axons) or combinations of both.

Axonal atrophy, and segmental demyelination, and changes in the axonal membrane are suggested as pathological lesions. Published findings speak in favor of segmental demyelination but axonal degeneration of myelinated fibers have been reported in guinea pigs, rabbits and cats exposed to lead. It is also possible that the two types of fiber degeneration may occur simultaneously. Demyelination may reduce considerably the nerve conduction velocity, i.e. the propagation speed of nervous impulses while in axonal degeneration the axonal conduction velocity may remain within normal limits or only slow down slightly. It is important to note that histological changes of segmental demyelination has been found in the same nerves that show marked reduction of conduction velocity.

According to Kehoe, severe lead poisoning usually does not occur at PbBs below 80 μg. However, he indicates that under conditions of prolonged and gradual absorption, it is difficult to pinpoint the exact blood lead level at which clinical symptoms appear (Ex. 294 (B)). Other investigators have indicated that peripheral neurological symptoms may appear at PbBs below 80 μg. For example, motor neuron disease has been found in lead workers at blood lead levels of 50 μg/100 g. (Ex. 24 (19), pp. 13, Ref. Campbell).

Reversibility of the patho neurological effect produced by lead has been a topic of considerable debate among neurologists. The majority of investigators agree that the phenomenon of reversibility (in nerve tissue) is directly correlated with the specific nerve components involved. For example, neuron cell bodies, once destroyed, are not replaced by other neuron cell bodies. Axonal damage, however, may be replaced by new growth from the cell.

Recovery from the effects of chronic lead poisoning may be feasible in some cases, if the worker is removed from the source of exposure and therapy is initiated immediately. There are instances, however, when complete recovery is impossible and the pathology is fixed. Even if the worker is removed from the source and therapy initiated, the worker may still experience impairment (Ex. 95 Ref. Cantarow pp. 135) In a recent paper describing his research Dr. R. Baloh a neurologist at UCLA questioned the reversibility of nervous system damage;

Although there are isolated reports of significant improvement in lead induced motor neuron disease and peripheral neuropathy after treatment with chelation therapy, most studies have not been encouraging, and in the case of motor neuron disease, death has occurred despite adequate chelation therapy.

All of this data reinforces a disturbing clinical impression that nervous system damage from increased lead absorption is only partially reversible, if at all, with chelation therapy and/or removal from further exposure. This is not particularly surprising, however, since experience with other heavy metal intoxication has been similar. Nervous system damage from arsenic and mercury responds minimally to chelation therapy. Apparently, irreversible changes occur once the heavy metal is bound by nervous tissue. Although further study is clearly needed, the major point I would like to make this morning is that there is strong evidence to suggest the only reliable way to treat nervous system damage from increased lead absorption is to prevent its occurence in the first place. (Ex. 27(7)pp. 55.)

OSHA agrees with these concerns regarding irreversibility of neurological disease expressed by Dr. Baloh and therefore must establish a standard which will prevent the development of nervous system pathology at its earliest stages.

(d) *Early Peripheral Neuropathy.* OSHA relied heavily on the work of

Seppalainen when discussing early damage to the peripheral nervous system in its proposal. Prior to the proposal and hearings, Dr. Seppalainen had published two papers describing her use of neurophysiological methods, especially nerve conduction velocity studies and electromyography to study the effects of lead. These papers formed the basis for her conclusion that she had observed subclinical neuropathy in lead workers. This work was discussed at great length prior to and during the hearings and for clarity will be reviewed here. (Ex. 2(12)(13).)

Nerve conduction velocity can be measured in motor and sensory fibers by stimulating the nerve with short electrical impulses and by recording the resultant electrical activity in the muscle or low amplitude electrical pulses in sensory nerves elicited by the stimulus. In her testimony at the hearings Dr. Seppalainen stated:

> Slowing of the nerve conduction velocity is a sign of neuropathy. Neuropathy causes also changes in electromyography, namely (1) the number of acting motor unit potentials is reduced (if the neuronal connection to muscle fibers is disrupted or impaired, the muscle fibers cannot work) (2) the duration of the motor unit potentials is prolonged, and (3) spontaneous pathological activity in the form of fibrillations, positive monophasic potentials and fasciculations may be found in the muscles. (Ex. 51, pp. 4)

Seppalainen's work was stimulated by earlier studies which utilized electrophysiological techniques to study nerve damage in lead exposed workers. Sessa et al. showed reduction of the maximal motor conduction velocity (MCV) of the ulnar nerve in patients with lead poisoning but without clinical neurological symptoms (Ex. 5(12), Ref. 1, p. 667-68; Ex. 24 (19), pp. 15, Ref. Behse; Ex. 24 (19), pp. 15, Ref. Vasilescu; Ex. 24 (19) pp. 14, Ref. Feldman; Ex. 24 (19), pp. 15, Ref. Girard; Ex. 24 (19) pp. 15, Ref. Guardriglia; Ex. 51 (B)) also demonstrated slowing of NCV's from exposure to lead. Catton et al. (Ex. 5(12), Ref. 2) presented evidence for a minimal defect of peripheral nerve function in a group of lead accumulator workers without clinical evidence of neurological lesions Of the 19 men examined 13 had blood levels above 80 μg/100 ml and seven had hemoglobin levels below 12 g/100 ml. In these workers, maximal motor conduction velocity was normal but the ratio of the amplitude of the muscle action potential following stimulation of the lateral popliteal nerve at the knee and at the ankle was in some instances smaller than that in control subjects. Catton et al suggested the most likely explanation for this finding is that conduction was slowed in some nerve fibers causing dispersal of the muscle action potential.

Based on this earlier work that demonstrated that the MCV remains normal as long as a portion of the fastest fibers are intact, Seppalainen determined that more sensitive methods were required to detect early or partial damage to peripheral nerves. It was this conclusion which led to the two papers discussed in the proposal and reviewed here.

In the first study 39 male lead workers were studied for peripheral nerve system damage using electrophysiological techniques. A diagnosis of lead poisoning had been made in 31 cases but were without signs of neurological impairment and 8 men had excessive or increased absorption of lead but were without symptoms at time of examination. Previously, 15 men who suffered from lead poisoning in the study and 5 of the symptom-free men had suffered from clinical lead poisoning. The authors reported a standard electromyogram demonstrated fibrillations and/or diminished number of motor units in 24 workers. The lead workers had significantly lower mean maximum conduction velocities of the ulnar and median nerve as compared to an age matched control group. The mean conduction velocity of the slower fibers of the ulnar nerve was 39.0 ± 8.0 whereas the control population was 46.6 ± 3.7 (p is less than 0.001). Based on this result, the authors conclude that measurement of the slower fibers (CVSF) of the ulnar nerve proved to be a very sensitive indicator of lead damage, and a combination of this variable and the distal latency of the median nerve discriminated lead workers from controls better than other combinations. Lastly, they conclude the findings are consistent with slight peripheral neuropathy and further that lead also affects certain portions of the fibers in the proximal part of the nerve as well as in the distal part of the nerve. None of the subjects had paresis at the time of the study, but paresthesiae, myalgia, or muscular fatigue were complaints of some workers.

The second Seppalainen study differed from the first in that the 26 workers (18 males and 8 females) selected for the second study were said to have PbB levels which never exceeded 70 μg/100 ml nor had suffered from clinical lead poisoning whereas in the earlier studies discussed, the subjects blood lead usually exceeded 70 μg/100 ml and symptoms of lead poisoning were prevalent. The authors reported the exposed group which was comprised of 26 workers (18 men and 8 women) from a storage battery factory had a mean exposure time of 4.6 (SD 4.7, median 3.7 years, range 13 months to 17 years). The concentration of PbBs had ranged mostly between 35 μg/100 ml and 60 μg/100 ml and occasionally between 20 μg/100 ml and 70 μg/100 ml.

The results of the conduction velocity measurements from the exposed workers and controls are presented in Table 2. The results indicated the MCV's of the arm nerves (median and ulnar nerves were slower among the exposed workers. There was a slight difference in the MCV's of the nerves in the lower limbs. However, the differences were small and little significance should be given to these small changes. The SCV's in the forearm did not differ. There were marked differences in the CVSF of the ulnar nerve, which is consistent with the earlier study. The CVSF of the ulnar nerve (distribution and mean) among these lead exposed workers were situated between those of the normal controls and those of subjects with lead poisoning from the earlier study. Seppalainen concluded that this finding indicates an exposure-response relationship at the group level between lead exposure and nerve propagation speed.

Table 2 — Nerve Conduction Velocities (msec) of Lead-Exposure and Control Subjects

	Exposed			Controls				
	N	Mean	SD	N	Mean	SD	t	P
MCV* of median nerve	26	54.5	5.2	26	58.5	3.8	3.19	<.005
SCV† of median nerve	25	59.5	5.3	8	56.3	4.1	1.83	>.05
MCV* of ulnar nerve	26	55.0	4.8	26	59.1	3.1	2.75	<.01
CVSF‡ of ulnar nerve	26	42.0	5.0	22	47.1	4.4	3.73	<.001
SCV† of ulnar nerve	25	58.2	4.7	23	60.0	4.5	1.42	>.05
MCV* of deep peroneal nerve	25	50.6	4.4	26	52.0	4.0	1.20	>.05
MCV* of posterior tibial nerve	26	43.3	3.0	19	44.6	3.2	1.32	>.05

*MCV, maximal conduction velocity.
†SCV, sensory conduction velocity.
‡CVSF, conduction velocity of slower fibers.

In both of the above studies needle electromyographic examinations were also performed, in the first case to all 39 subjects, and in the second to 11 exposed workers with abnormal or borderline nerve conduction velocities. Neurogenic EMG abnormalities were frequent in the case of lead poisioning; EMG was abnormal in 24 subjects, and denervation activity (fibrillations) was found in 15 cases. Among lead exposed workers with PbBs not exceeding 70 μg/100 ml, EMG was abnormal in 9 cases out of 11 studied, and in 5 cases fibrillations were found. The authors stated:

"That the neurotoxic effect of lead can also be found in the muscles, which have undergone slight neurogenic degeneration. (Ex. 5(12).)

As previsously stated the findings in this study were qualitatively similar to the previous study but in a quantitative sense they were milder. It is significant that this study indicating changes in maximal motor conduction velocities and electromyographical abnormalities occurred in subjects whose PbB levels had never been above 70 μg/100 ml. When discussing their results Seppalainen concluded:

Of course, in terms of health, the importance of slight subclinical neuropathy can be questioned, too, and we did not find any evidence that the well-being of these workers was influenced by the neuropathy, apart from a few complaints of numbness of the arms. Thus, the term poisoning, in its orthodox sense, cannot be applied to these disorders. But neuropathy, no matter how slight, must be regarded as a more serious effect than the quite reversible alterations in heme synthesis, because the nervous system has a poor regenerative capacity, and the acceptability of such a response must be judged from that point of view. Since the entire question belongs to the diffuse "gray area" between health and disease, it is more than probable that opinions will diverge. We think, however, that no damage to the nervous system should be accepted, and that, therefore, present concepts of safe and unsafe PbB levels must be reconsidered. (Ex. 5(12), p. 183.)

During the hearings Dr. Seppalainen described continuing research on the exposure-response relationship between lead exposure and neurological impairment. She reported the results of an examination of 64 workers with occupational lead exposure ranging from 2 to 20 years. A preliminary report of this research had been given at the second International Workshop on Occupational Lead Exposure, Reevaluation of Permissible Limits. These subjects' blood lead levels had been monitored on a regular basis and had never exceeded 70 μg/100 ml. Nerve conduction velocities of this group were compared to those of 22 controls and to 18 workers whose PbB levels had exceeded 70 μg/100 ml.

Results in this study were similar to those previously described. The conduction velocities in the leg nerves were unchanged save for a small change in the posterior tibial nerve in those whose PbBs exceeded 50 μg/100 ml. As previously described there was a reduction in conduction velocities in arm nerves and a statistically significant linear relationship appeared to exist between the PbB and nerve conduction velocities of the sensory conduction velocities of the sensory conduction velocity (SCV) of the median nerve in the forearm section as well as in the distal section (dsCV), motor distal latency of the median nerve and CVSF of the ulnar nerve. In these groups abnormalities were defined as equal to values below normal mean minus two standard deviations. Abnormalities in two or more nerves appeared more frequently in the higher PbB groups. (See table 3.)

TABLE 3

Number of persons studied	Blood lead μg/100 ml.	Number of exposed persons with abnormality	Percent
22	Less than 20	0	—
15	40 to 49	1	7
23	50 to 59	3	13
15	60 to 69	4	27

The report of the Second International Workshop on Permissible Levels for Occupational Exposure to Inorganic Lead drew the following conclusion on this work of Dr. Seppalainen:

> It is not known whether the maximum blood lead concentration or the integrated average concentration is the determining factor in the development of changes in nerve conduction velocity. However, the Group concluded from the data presented by Seppalainen et al. and the data reported in the literature that changes in nerve conduction velocity occur in some lead workers at blood levels exceeding 50 μg/100 ml. It was thought that no conclusion could be drawn from the one case in the blood lead range 40–49 μg/100 ml.
>
> It is not possible to decide what any given measured small deficit means in terms of specific nervous damage. However, it is generally recognized that a clear deficit in the nerve conduction velocity of more than one nerve is an early stage in the development of clinically manifest neuropathy. There is no evidence that these changes progress. Reversibility should be studied. Although slight changes may be measured in persons experiencing no symptoms, it was the concensus of the Group that such changes should be regarded as a (Critical effect is a defined point in the relationship between dose and effect in the individual, namely the point at which an adverse effect occurs in cellular function of the critical organ). (Ex. 262, pp. 6–7.)

Seppalainen reported at the hearing that the number of controls had been increased and the lead exposure data had been rechecked. In this most recent study the authors carried out statistical calculations which compared the results of workers with occupational lead exposure from 2 to 8 years to those of controls. They only accepted workers whose PbBs had been determined from the onset of exposure; 1–2 times per year up to 1970 and from then 4–6 times yearly thereafter. As in the previous studies statistically significant reduction in several nerve conduction velocities were noted (p less than 0.01) at PbBs 50–59 and 60–69 μg/100 ml.

The nerves with reduced NCV's included the motor distal, latency, SCV, dSCV of the median nerve, and the CVSF of the ulnar nerve. The latter was slowed at PbB levels below 50 μg/100 ml. Again, dose-response relationship was operative in this recent work.

During the hearings there was considerable critical testimony of Seppalainen's research which focused on two general issues: The first, criticism as articulated by LIA was:

> Even if Seppalainen's findings were accurate and reliable—it is clear that the slight reduction in nerve conduction velocities which she found does not constitute "material impairment of health" and does not affect the functional ability of lead workers who have blood-lead levels below 80 μg/100 ml. (Ex. 335, p. 25.)

Second, there was criticism of her methodology; questions were raised regarding the reliability and accuracy of her results. We shall address the latter criticisms first. (Ex. 3 (72); Ex. 335). It should be pointed out at the outset that Seppalainen's research has been in the literature for a number of years. The published work has been subjected to peer review, for several years without any major reported challenge. Multiple investigators, some already cited have confirmed her work. One point regarding her work needs to be stressed. That is, unlikely many cross sectional studies in the record in which the only PbB levels determined were done at the time other parameters were measured, Seppalainen utilized data over a period of years to insure that PbB levels were always below 70 μg/100 ml. The reliability of her PbB determinations was excellent. This type of study should serve as a model for other investigators.

This research has also been quoted widely and discussed in great detail and apparently accepted as valid at scientific meetings, e.g. The Second International Workshop on Permissible Levels for Occupational Exposure to Lead. In OSHA's view this research is widely accepted in the scientific community and has indeed been the stimulus for subsequent studies in the use of electrophysiological techniques to investigate early neurological/damage.

The following criticisms were raised by LIA. (Ex. 335, p. 28):

> (1) The control group and the lead-exposed group were observed under different circumstances and tested at different times. The control subjects were not specifically chosen as controls and were tested over the period between 1970 and 1973, whereas all of the lead-exposed subjects were tested only in 1973. Different rooms were used for the studies. As a consequence of these differences, it is difficult if not impossible to compare meaningfully the test results from the two groups." (Ex. 335, p. 28.)

There is no inherent reason why controls and subjects need be tested at the same time since the real issue is whether the conditions such as temperature were standardized and controlled. If the conditions are standardized then this point is moot. Seppalainen did testify at the hearings that subjects and controls were in fact intermixed in the work reported in Amsterdam and there were no inconsistencies between this study and others.

LIA also criticized:

> (2) Although the study suggests that the lead-exposed subjects were biologically monitored during the "entire period" of their exposure to lead, 6 of the 28 subjects—or more than one out of every 5 workers—had been occupationally exposed to lead prior to the time the monitoring began. It is therefore entirely possible that some or all of these workers had previously had higher blood-lead levels, and that the slight neurological changes observed actually occurred and were caused when blood-leads were higher, not at the lower levels which existed at the time of the monitoring. (Ex. 335, p. 29.)

This point was addressed by Seppalainen in her original paper in 1975 as follows:

> Only six of the subjects studied had a working history of more than 5 years; they were included despite defective monitoring data, only because there was sufficient reason (i.e., no change in working methods and the working environment), to presume that exposure had not been higher in the past than during the period of frequent monitoring. (Ex. 5 (12), p. 180.)

In addition, there is no evidence in subsequent work that there were inconsistencies in the subject population and therefore any evidence that the research was flawed.

A third criticism of Seppalainen's methodology by LIA was:

> (3) There is a serious question as to whether skin temperatures were adequately monitored throughout the testing period and were maintained at the correct level. (Ex. 335, p. 29.)

Seppalainen discussed the issue of temperature in great detail during the public hearings, especially in response to questions from Dr. Jerome Cole, Lead Industries Association. In her prepared testimony Seppalainen stated:

> According to my large clinical experience nerve conduction velocities begin to slow down significantly, when the skin temperature at the stimulation point is lower that 30° C. In my previous studies I have not published detailed information on skin temperatures, which, however, always have been measured. In the last study all the skin temperature measurements were taken into statistical analysis and no difference could be found between any exposed group and the control group. (Ex. 51, p. 9.)

Dr. Cole pursued this issue during cross examination when he pointed out the significance of temperature changes on nerve conduction velocities. Seppalainen agreed that nerve conduction velocities are very much affected by differences in temperature and proceeded to describe her method for determining temperatures:

Dr. SEPPALAINEN. The way I regularly do it and which I have used in all of my studies concerning lead—concerning the peripheral nervous system and lead is such that I start with nerve conduction velocity measurements, motor and sensory nerve conduction velocity measurements of the arms where the nerves are stimulated at the elbow level and at the wrist level. This procedure, when I am performing this thing, it lasts about 7 to 10 minutes and the final step in this measurement is the measurement of skin temperature at the proximal stimulation point at the elbow level which is used with an appliance which is called Sekunda Thermometer, made by Testoterm, I think it is of German make and also at the same time I record the temperature on the tip of the third finger. I do not think that there are major changes during these seven or ten minutes on the skin temperature. I just can hold, I mean, the probe all of the time on the nerve. (Tr. 134-36.)

The discussion of temperature control will not be repeated since it is apparent that Dr. Seppalainen has rigorously controlled for temperature. OSHA has therefore concluded that the methodology employed by her was not only adequate but was designed with care and precision.

More recent studies on the relationship between occupational lead exposure and peripheral nerve conduction velocities have confirmed Seppalainen's work. For example, Araki and Honma (Ex. 51 (B)) studied 36 male workers who had been exposed to lead occupationally from 3 months to 46 years and whose PbB ranged from 29 to 73 μg/100 ml. There was a statistically significant relationship between the maximal motor nerve conduction velocities, mixed nerve conduction velocities of the median nerve of the forearm and with the MCV of the posterior tibial nerve and PbB of 29 to 73 μg/100 ml when compared to an age matched control group. The authors in this study concluded that the nerve conduction velocities were affected primarily in the forearm, and "the diminished MCV of the median nerve in the forearm may be a manifestation of motor peripheral neuropathy due to lead absorption." (Ex. 51 (B), p. 231.)

Lee and co-workers conducted electrophysiological studies on 94 workers whose average blood lead at the time of testing was 60 ± 15 μg/100 ml. The authors used a 1-tailed, paired t-test in comparing conduction veolcities of the lead workers and age-matched controls. The results indicated that all maximum motor nerve conduction velocities of lead workers were significantly decreased but maximum sensory nerve conduction velocities were not affected. Both fast and slow fibers were affected. In contrast to the previously described studies these authors did not demonstrate a significant correlation between biochemical parameters such as PbB, ALAD, FEP, Hb, PBU, ALA, and CP and reduced nerve conduction velocities. The data as analyzed by multiple regression analysis did indicate that length of exposure was significantly correlated (p less than 0.05) with ulnar nerve conduction velocity. Length of exposure did not correlate with other conduction velocities. No explanation for the differences was advanced. It is important to note that conduction velocities were significantly decreased in men whose exposure had been less than 2 years (1.5 years average) and whose mean blood (lead) at time of testing was 58 ± 16 μg/100 ml.

Professor Lee's work has been criticized by Dr. John C. Steiner who is assistant Professor of Neurology at the University of Cincinnati College of Medicine. (Ex. 234 (21)). Dr. Steiner states in his testimony:

Lee stated that "all maximum motor conduction velocities measured were significantly decreased in lead workers." This appears to be a misstatement, and perhaps he meant that the mean value of the motor conduction velocities for all nerves examined were abnormal, but on the basis of his data, it is not possible for me to conclude that one could differentiate individually affected persons, and that therefore all persons would have abnormal conduction velocities. While it is not stated whether temperature was controlled, his values for the 94 normal persons' ulnar nerves was considerably less than those reported by Payan (22) who did control for temperature: Lee—55.6 ± 4.3 M/sec, Payan, 69 ± 5.5M/sec.

The percent mean conduction velocity difference in Lee's controls vs. lead workers in ulnar, median and peroneal nerves was 4, 2.5 and 3.1 percent, respectively, while the radial nerve conduction velocity was reduced by 12% in workers and controls. Again, in the peroneal nerve, the ratio of amplitude to the action potential of extensor digitorum brevis muscle when stimulated at the knee vs. at the ankle was reported as being significantly different in workers. The large standard deviation and known effect of chronic pressure of the shoe across that muscle, plus assumed difference in temperature would invalidate the significance of the finding. (Ex. 234(21), Ref. Steiner, p. 9.)

These issues were not addressed during the hearings. OSHA believes the questions raised do not invalidate Professor Lee's conclusions that there was a significant relationship between lead exposure and reduced motor conduction velocities. In fact, industry has accepted these findings as being accurate (Ex. 234(21)). There is little if any doubt that a real effect was measured and that it was directly relatable to the subjects' exposure to lead.

In addition to the research already described there have been a number of recent studies which relate peripheral neuropathy to the reduction of nerve conduction velocities. (Ex. 97; Arch. Phys. Med. Rehabil. *56*, 312 (1975); Nervenartz, *46*, 674 (1975))

Landigran and coworkers (EPA Criteria Document, Ch. 11, Ref. 231) carried out an extensive epidemiologic, hematologic and neurologic study on children who lived near a lead smelter. Neurologic examinations were undertaken on a cohort of 5- to 9-year-old children who were selected from the study and geologic control areas. As many children with blood lead levels (greater than or equal to) 40 μg/100 ml were selected as could be matched with controls. Pair members were matched in terms of age, sex, and socioeconomic stratum. A total of 123 pairs were selected. Peripheral nerve conduction velocities were measured in blind fashion on the right peroneal nerve of each child and the results indicated a statistically significant negative correlation between peroneal nerve conduction velocity and blood lead level.

($r = -0.38$, $t = 2.12$, p is less than 0.02 by 1-tailed t test.) The authors concluded that while the data do not allow for clear statements of threshold effect, the "findings are consistent with a growing body of data which indicates that various subtle neurologic and psychologic abnormalities may develop in children with increased lead absorption."

In a letter to the editor of the Journal of Pediatrics, September 1977, Gartside and Panke critiqued the findings of Landrigan et al. While acknowledging that the matched pair approach is a powerful technique, these authors argued that matching should be carried out utilizing all the concomitant variables which were significantly associated with the variable of interest. In this study only four of the concomitant variables were used in creating the matched pairs. The authors suggest that those left unused could account for the differences observed. Gartside and Panke examined several subsets of the original matched pairs and found that the correlations between blood lead and nerve conduction velocities was less significant. This reevaluation of the data, however, suffers from a classic biostatistical problem which, simply stated, is that by reducing the overall number of the population studied, any likelihood of establishing a statistically significant result is also concomitantly reduced. The question of utilization of all concomitant variables, while not being invalid, must be perceived as being highly speculative, and without further research OSHA believes that the conclusions of the Landrigan study are

valid, especially when considered in light of other data in the literature.

NIOSH has conducted a major health hazard evaluation of the effects of lead exposure on workers employed at the Bunker Hill Co. lead smelter and zinc plant (Ex. 300). One aspect of that study included an evaluation of neurological and psychological measures. The neurologic and psychological tests consisted of measures of the following:

(1) Maximum motor nerve conduction velocity (NCV) of the ulnar nerve;

(2) Maximum motor NCV of the peroneal nerve;

(3) Eye-hand coordination;

(4) Choice reaction time; and

(5) Subjective feelings, as measured by the multiple adjective affect checklist (MAACL).

The following observations can be made from the data collected:

(1) Ulnar conduction velocities are lower in smelter employees.

(2) Peroneal nerve conduction velocities are lower in smelter employees.

(3) Choice reaction time (RC mean) is increased by approximately 10 percent for male smelter employees and by the same amount for production females.

(4) Eye-hand coordination did not differ for smelter employees, with the exception that clerical females exhibited superior performance on this particular test.

(5) No pattern of hostility, depression, or anxiety is evident in either male or female smelter groups.

Results from multiple regression analyses indicate that the lower nerve conduction velocity for lead males and zinc males are inversely correlated to age and to ZPP, the latter is consistent with the earlier described work of the Mount Sinai group, (Ex. 23 (39)). With the exception of age, no other independent variable achieved statistical significance for lower NCV in production females if ZPP was used in lieu of blood lead as an independent variable. However, if blood lead, not ZPP, was used in the regression analysis, it was found that peroneal NCV was significantly correlated ($r^2 = .29$, p is less than .01) with age (p is less than .01) as well as blood lead ($p = .08$). No pattern was evident regarding an association between RC mean and any independent variable other than age.

Nerve conduction velocities are consistently lower in smelter employees than controls but the decreases are small, and achieved statistical significance only for the peroneal nerve in female smelter workers and male zinc smelter workers. The latter result, however, is inconsistent with lead absorption indices (ZPP, blood lead) which indicate lower ZPP and blood lead values for male zinc smelter workers than for male lead smelter workers. Production females show a 3.2 m/sec decrement in peroneal NCV compared to female controls. This 3.2 m/sec difference is the largest decrement in NCV found in the study; however, the ZPP and blood lead values are only half of comparable values for male lead smelter workers. It cannot be explained whether this inconsistency means that: (1) Females are more susceptible to lead than males, or (2) ZPP and blood lead may be poor indices of nervous system effects, or (3) the results are an anomaly. NCV means (ulnar and peroneal) show a consistently greater decrement for females than males. However, indices of lead absorption for females were only about half of comparable values for males.

Mean choice reaction times seemed to be consistently lengthened by about 10 percent in both male and female smelter workers. However, "dose-effect" relations are not evident, since lead males do not differ from zinc males, whose mean blood lead was lower by about 43 percent.

Performance on the test of eye-hand coordination did not reveal any association of impaired performance with lead exposure. The only significant result that was found showed clerical females to be more proficient in eye-hand coordination than any other group. This result could easily be due to selection processes inherent in hiring clerical/secretarial employees, or enhancement of eye-hand coordination skills through typing or other similar job-related duties.

The psychological data (depression, anxiety, hostility) showed no major differences between smelter workers and control groups. However, if published norms are used as a basis for comparison, the following results are obtained:

(1) Production females are more depressed than normal ($t = 2.18$, d.f. = 143, p is less than .01).

(2) Lead males and zinc males are more depressed than normal ($t = 2.58$, d.f. = 262, p is less than .01; $t = 2.48$, d.f. = 292, p. 05, respectively).

(3) Lead males, zinc males, and spirit lake males are more hostile than normal ($t = 3.43$ d.f. = 262, p is less than .001; $t = 2.41$, d.f. = 292, p is less than .01; $t = 2.44$, d.f. = 184, p is less than .05).

Multiple regression analyses showed that subject's age was a factor consistently correlated with neurologic, psychometric, or psychological data. ZPP levels were also correlated with peroneal NCV in lead males and zinc males, but not for production females. In general, it was found that ZPP was a better variable for inclusion in regression analyses than was blood lead level. This has been found to be true in other studies described in this section.

The final study of lead neuropathy to be discussed in this section is a recent paper by Feldman and coworkers entitled "Lead Neuropathy in Adults and Children", Archives of Neurology *34*, 481 (1977). In one aspect of this study Feldman examined six adults, all of whom displayed clinical symptoms of nervous system disease. Their blood lead concentrations were 58, 80, 87, 160, 180, and 233 μg/100 gm and their right peroneal motor nerve conduction velocities were 41, 40, 49, 44, 38, and 37 m/sec respectively. These adults had all worked in environments with high lead exposure for extended periods of time and therefore had increased body burdens of lead. These adults manifested a wide variety of neurologic symptoms and signs such as encephalopathy, myelopathy, and peripheral neuropathy.

The authors suggest that MNCV determinations as an additional screening exposure to lead would be efficacious in clinical practice because it provides data on probable accumulative effects of exposure to lead.

A blood lead level may reflect current exposure, but tells little about past exposure or about total body lead burden. Circulating levels of lead in whole blood have no relation to the concentration of lead in the nervous system. Postprovocation excretion of lead is a better reflection of past exposure and total body burden than the blood lead but provides little information about the possible accumulation of lead or its toxic effects in the central and peripheral nervous systems. (Arch. of Neurol 34: 483)

In a second study described in this paper, Feldman et al. reported on 19 lead exposed steelworkers who were involved in the dismantling of an elevated train track network in Boston—13 workers were burners and 6 were nonburners who were also exposed to lead but to a lesser degree. The 19 workers were studied for whole blood lead, hematocrit, hemoglobin, protoporphyrin and calculated FEP concentrations as well as motor nerve conduction velocity of the peroneal nerve. Both groups of workers wore respiratory protection. The nonburners had been exposed from 4 to 10 months whereas the burner workers had as little as 1 month exposure before symptoms became evident. The burner workers had a mean MNCV of 43.2 and the six nonburners was 49.0 as compared to the control mean of 54.09m/sec ± 5.96. This study demonstrates again the relationship between neuropathy and motor conduction velocities, and indicates slowing of MNCV at blood leads well below 80 μg/100 ml.

In the third section of this paper the authors developed the following hypothesis:

If the observation of slowed MNCV in cases of symptomatic lead intoxication and in subclinical cases of lead neuropathy have validity as a possible factor for measuring increased absorption of lead, then by measuring MNCV it should be possible to identify those affected from a group of individuals at risk. (Arch. of Neurol. 34: 485)

Children were selected for study from a public housing project in a city in Massachusetts where there was evidence of lead in window and door casements and on the outside walls, and 26 parents volunteered to have their children tested. Children with peroneal MNCV below 47.63 m/sec (1 SD below the control mean) were considered suspect of having an increased lead body burden. These children were admitted to a hospital for edetate disodium calcium provocation, and 10 of 26 had MNCV's—one or more SD below the control mean. (See table 4.)

Table 4. — Blood and Urine Lead Levels and the Results of Edetate Disodium Calcium Provocation

Patient	Motor Nerve Conduction Velocity, M/sec	Screening Blood Lead Level, ug 100 gm	24-Hour Urine Lead Excretion Level		
			Before Edetate Disodium Calcium Therapy, ug/Liter	After Edetate Disodium Calcium Therapy (50 mg/kg)	
				ug/Liter	u mole of Lead/u mole of Edetate Disodium Calcium
4	43.5	23	10	260	0.094
6	45.0	14	20	180	0.152
7	38.5	23	10	700	0.422
15	38.5	17	20	50	0.076
20	31.0	41	10	683	0.566
21	38.0	33	16	1,036	0.957
22	40.5	83	14	695	0.297
25	44.5	41	--	Not chelated	-----
17	40.0	22	--	Not chelated	-----
10	47.5	29	--	Not chelated	-----

The mean urine concentration of 514.9 μg/liter/24 hr for the seven chelated patients is more than triple the mean control level of 165 μg/liter 24 hr for unexposed children. The difference between the two means is highly significant (t=4.38, p is less than .001). The 24-hour collections before and during edetate disodium calcium provocation show a remarkable outpouring of lead in four cases: subjects 7 (700 μg/liter), 20 (683 μg/liter), 21 (1,036 μg/liter), and 22 (695 μg/liter). The authors conclude:

> Although the size of the sample is small, it can be observed that of the seven children initially selected, on the basis of slowed MNCV at a level of at least 1 SD, as being suspect of increased body lead burden, a possible relationship exists between the MNCV slowing and the quantity of lead excreted in the urine after edetate disodium calcium provocation. The four children with provoked urine excretion of greater than 600 mg of lead all had MNCVs of less than 2 SDs below the control mean; in the remaining three children the results of provocative chelation were less striking. Yet, two of them showed lead concentrations in urine exceeding the mean for control subjects.
>
> The individual differences in MNCV values among the at-risk groups and control subjects may be small, but, as more data are gathered, it becomes clear that subclinical neuropathy, measured by nerve conduction velocity, is a concomitant of increased lead absorption in children. (Arch. Neur. *34*, 486-87, (1977))

These three studies illustrate motor nerve conduction velocity reductions in three distinct lead exposed populations, demonstrate effects at low levels of lead exposure and further indicate the effects on children as well as adults.

Based on the studies described in detail and others only referenced, OSHA has concluded there is extensive evidence in the record that there is statistically significant reduction of certain nerve conduction velocities in both male and female lead workers whose blood lead levels are 50 μg/100 ml or greater. OSHA also concludes that the slowing of MNCV follows a dose-response relationship although the issue of whether the pathological findings bear any relation to past PbB levels is as yet unresolved. The record evidence is insufficient to determine whether slowing occurs in the 40 to 50 μg/100 ml range. It is OSHA's view that the research cited has withstood the challenges to the methodology employed, in particular to the questions of temperature control, control groups, blood lead determinations, instrument use, and testing protocols. While Seppalainen's work is the most extensive to date, numerous other investigators already mentioned have shown NCV reduction in lead exposed workers and others.

The principal issue raised with respect to the reduction of NCV velocities was whether this effect constituted "material impairment" of health, (Ex. 335). That is, is there a continuum of disease associated with exposure to lead so that it is axiomatic that the chronic irreversible stage is preceded at the opposite end of the disease progression by a relatively mild, apparently reversible stage of disease which is characterized by the MNCV reductions described herein. Based upon the voluminous evidence in the record concerning MNCV reduction OSHA believes that these reductions do constitute material impairment and do represent early indicators of a neurological disease process.

This earliest stage is characterized by varying subjective and/or objective symptoms that may not at first unduly alarm the worker or present a physician with clear-cut diagnosis. Nevertheless, this early developmental stage of neurological disease is a pathological state, and OSHA finds persuasive the arguments for adopting a lead regulation which protects workers from the early consequences of lead exposure. Evidence in the record convinces OSHA that it is necessary to protect the many thousands of workers who exhibit reduced nerve conduction velocities. Witnesses on behalf of industry testified that these changes in MNCV were not cause for concern (Tr. 1885, 1903, 2108, 3040, 6577). However, industry testimony failed to disprove the significance of these acute effects as manifestations of neurological disease which, with continued exposure, progresses inevitably to the chronic neurological diseases associated with lead exposure. OSHA believes that motor conduction velocity decrements reflect nerve damage and are significant health effects in themselves and should be prevented since prevention is the only reliable treatment for the irreversible changes which occur once lead is bound by the nervous tissue.

OSHA believes that the scientific evidence indicates that reduced motor nerve conduction velocities are manifestations of peripheral neuropathy classified clinico-pathologically as a demyelinating neuropathy, although there is also evidence for axonal atrophy and changes in the axonal membrane. The predominant type of myelinated fiber pathology is segmental demyelination. In general the morphologic normality of the neuron in the presence of widespread segmental demyelination speaks in favor of a generalized damage of Schwann cells than of neuronal damage. It is therefore consistent that histological changes of segmental demyelination are found in the nerves that showed marked reduction in conduction velocity.

There is evidence that a lead worker may in some cases revert to a normal state of health if exposure to lead is discontinued although OSHA believes complete recovery is unlikely, if not impossible, and therefore OSHA is convinced by the evidence in the record that those many workers who will grow progressively worse must be identified and protected.

In reaching this conclusion OSHA has relied on the work of Seppalainen, whose technique has been described by Dr. Macolm of Chloride Incorporated as "immaculate." (Tr. 2123). Seppalainen describes her view of neuropathies as follows:

> Thus, the main importance lies in the implications for the prevention policy at the place of work, as well as for the setting of safety norms. It is commonly thought that PbB levels in excess of 70 μg or even 80 μg/100 ml are obligatory for the development of poisoning. Biochemical changes that do occur at lower lead levels, i.e., depression of erythrocyte ALA dehydratase and a slight increase in urinary coproporphyrin and ALA, are often said to be of no clinical importance. Of course, in terms of health, the importance of slight subclinical neuropathy can be questioned, too, and we did not find any evidence that the well-being of these workers was influenced by the neuropathy, apart from a few complaints of numbness of the arms. Thus, the term poisoning, in its orthodox sense, cannot be applied to these disorders. But neuropathy, no matter how slight, must be regarded as a more serious effect than the quite reversible alterations in heme synthesis, because the nervous system has a poor regenerative capacity, and the acceptability of such a response must be judged from that point of view. Since the entire question belongs to the diffuse "gray area" between health and disease, it is more than probable that opinion will diverge. We think, however, that no damage to the nervous system should be accepted, and that, therefore, present concepts of safe and unsafe PbB levels must be reconsidered. (Ex. 5(12), p. 183)

During the hearings Seppalainen concluded:

> The slowing of nerve conduction velocities shows an effect of occupational lead exposure upon peripheral nerves. Although this slowing was slight in degree, it should be considered a harmful effect. In previous studies on neurotoxicity of lead, we have demonstrated abnormalities also in muscles in subjects with a slight slowing of nerve conduction velocities. Electromyography in many of them showed changes of denervation type. Furthermore, nervous tissue is slow and at times imcomplete in regeneration, if damage in it is advanced.
>
> As in the case of exposure to a neurotoxic chemical (carbon disulfide) the slowing of the nerve conduction velocity precedes clinical palsy, I consider that such slowing should be regarded as an early sign of beginning clinical neuropathy. The same phenomenon is a possibility which has to be kept in mind when dealing with human exposure to lead. When a worker with lead exposure shows abnormally slow nerve conduction velocities in two nerves, better hygienic measures are advisable to decrease his individual exposure in order to prevent more profound damage. Normal hemoglobin in these cases is no proof that the nervous

system is safe, since the pathological signs in the hematopoietic system appear at significantly higher PbB levels than nervous system signs. (Ex. 51, p. 7)

Dr. Kenneth Bridbord of NIOSH divided response to environmental exposure into five categories: normal, physiologic change of uncertain significance, pathophysiologic change, morbidity (fairly severe disease), mortality. (Tr. 1795-1801). He places nerve conduction velocity impairment in the "pathophysiologic change" category which can be described as:

. . . a change that is very closely associated with disease but may not, in and of itself, be called disease." I think the earliest sign that I would consider adverse, would be the decreased nerve conduction velocites in which case, in adults we begin to see this as blood lead levels rise about 50. One reason why I think that is clearly a pathophysiologic response or should be categorized as such, is that the ability of the nerous system to repair itself is fairly limited. That's not to say that there couldn't be any reversibility in some of these indicators but clearly there is very limited capacity to repair damage once such damage has occurred.

I think another point on the nerve conduction velocities is we're still measuring a fairly simple function and that to perform complex functions requires some integration of a number of circuits, maybe an electrical analogue might be a good example and that has to involve a certain amount of feedback and any decrease that one might find in a simple straight path I would think would tend to be accentuated to some degree as you get into more complex task and integration of many switching points, etc.

But I would clearly put the nerve conduction velocity in the pathophysiologic change. I think, in terms of dose response relationships in adults, I'm not sure we have a great deal of evidence to find where the pathophysiologic change clearly becomes, a morbidity change. Again, it's probably a continuum. It's probably that triangle break is going to vary from individual to individual in terms of when the pathophysiologic change begins to be considered morbidity.

Certainly, once someone has had wrist drop, unequivocally that's morbidity and that's a very distinct disease entity. In the case of damage to the nervous system, it is quite well established that at fairly high levels of exposure, that can be the cause of death even in adults. (Tr. 1795-1801)

The record of this proceeding contains numerous examples of clinical symptoms behavioral changes and reductions in nerve conduction velocity at blood lead levels as low as 40 μg/100 g and OSHA believes a standard should be established to prevent these changes from occurring in lead exposed workers. The standard must not be based only on prevention of foot or wrist drop, but rather on prevention of early neurological disease as manifested by behavioral and MNCV changes. It should be recalled that the Second International Conference on the Reevaluation of Permissible Exposure Limit for lead described reduction of MNCV as a "critical effect". (Ex. 262) OSHA believes that the characterization of MNCV reductions as "pathophysiologic changes" or as a "critical effect" are accurate representations of a disease process which is likely to be irreversible and which must be considered to represent material impairment even at these early stages.

OSHA agrees with Dr. Seppalainen in her conclusion on protective blood lead levels:

Mr. Becker. "Could you suggest a level under which a nerve conduction test would not be required?

Dr. Seppalainen. If I want to be safe, I would say 50 micrograms per 100 milliliter in blood." (Tr. 147)

However, in order to establish a reasonable margin of safety and to prevent other early neurological effects OSHA believes that blood lead levels should be maintained at or below 40 μg/100 g for lead exposed workers throughout their working lifetime.

4. *Renal System.* a. *Introduction.* Occupational lead nephropathy has until recently not been recognized as an industrial hazard in the United States. The NIOSH criteria document, "Criteria for a Recommended Standard: Occupational Exposure to Inorganic Lead", 1972, stated: "In the United States, there have been few reports of renal disease in lead workers, although the PHS survey discovered an increased incidence of albuminuria in affected workers". (Ex. 1, p. III-9) In the preamble to the proposed lead standard OSHA addressed the issue of the lack of information on renal disease in lead workers:

The relative absence of renal disease seen among workers in recent years has at times been interpreted as evidence that renal damage is not likely to occur among workers at current levels of exposure. Two recent observations, however, suggest that this may not be so. Vitale, et. al., observed evidence of lead nephropathy based upon renal functions and biopsy studies in three workers with blood lead levels below 80 μg/100 g. Cramer, et. al., observed effects of exposure to lead upon the kidneys after four or more years' exposure, including a decreased ability of proximal tubular cells in the kidneys to form inclusion bodies and a decrease in the ability of the kidneys to excrete lead accompanied by a moderate degreed of fibrosis. These changes were not characterized by any gross impairment of renal function. The authors considered it doubtful, however, that these changes were completely reversible. A critical question raised by this study is whether these changes could increase the risk of lead nephropathy with frank renal failure as has been observed in some instances. (Ex. 2, p. 45937)

In recent years evidence has accumulated indicating lead nephropathy is a significant problem to lead exposed workers. It is apparent from the record in these proceedings that lead nephropathy caused by prolonged exposure to lead is a consequence of crucial importance. Whereas the effect of lead on the hematopoietic, reproductive, and the nervous systems have been widely studied in recent years the record indicates that kidney disease associated with chronic lead exposure, while of equal significance, has been inadequately investigated.

Unlike the hematopoietic system, the means available to diagnose the early onset of renal disease is far more limited thereby making prevention more difficult but essential. Routine clinical tests will not indicate the presence of kidney dysfunction until lead nephropathy is advanced. Blood tests, such as the blood urea nitrogen (BUN) and serum creatinine (S-Creat) are increased only when two-thirds of kidney function is lost. In early lead nephropathy urinalysis is normal and therefore early renal disease is not routinely detected under clinical examination. Blood lead determinations have been found to be of limited value in diagnosing renal disease because they are primarily a measure of absorption when sampled close to the time of exposure. Blood lead levels are themselves not a measure of body burden of lead or the cause of symptoms or signs after lead exposure has ceased. Due to this insensitivity the lead standard cannot be based on the ability to detect organ damage through symptoms. When symptoms of renal failure are present "it is too late to prevent or correct the disease. Progression to death or dialysis is likely." (Tr. 1732) Given these conclusions, this agency must approach the prevention of kidney disease while recognizing the limits of certain biological parameters. Therefore, OSHA believes that any standard established for lead must provide some margin of safety. The Agency agrees with Dr. Richard Weeden, a nephrologist who testified for OSHA during the hearings, that "40 μg/100 ml (in blood) is the upper acceptable limit." (Tr. 1771).

b. *Renal Disease.* The fundamental unit of the kidney is the nephron. A nephron is made up of a glomerulus and a tubule. The blood is filtered in the glomerulus and this plasma filtrate enters the tubule where it is modified and eventually leaves the body as urine.

The arteriole supplying the glomerulus of a nephron also surrounds and supplies the tubular portion of that nephron. This vascular arrangement enables the proximal convoluted tubular cells to remove needed body substances (H_2O, glucose, salts, nutrients, etc.) from the urine and return them to circulation, as well as to assist the glomerulus in the removal of toxic substances (lead) from the blood for urinary excretion.

Whether excessive exposure to lead results in a form of chromic nephro-

pathy has long been debated. A sequence of histological changes extending from the occurence of lead-induced intranuclear inclusion bodies to diffuse interstitial fibrosis and renal failure has been described in animal studies by Goyer. (Ex. 6(38), Ref. Goyer (1971a)). It was concluded that there was dose response relationship between lead exposure and chronic nephropathy.

The study of humans with prolonged lead exposure suggests that there may be two or three stages in the reponse of the human kidney to chronic lead exposure. In an early phase, lasting less than 1 year, the proximal renal tubular cells form nuclear inclusion bodies similar to those found in animals who are experimentally lead poisoned. These inclusion bodies have been seen so frequently in cases of lead intoxication, that they have become one of the diagnostic criteria of lead poisoning. In fact, the lead within the inclusion bodies is 60 to 100 times more concentrated than in the entire kidney. Although it is an likely pathological reaction, it has been suggested that the inclusion body serves as an adaptive or protective mechanism during the transcellular transport of lead by the tubule cells. This mechanism has the effect of maintaining a relatively low cytoplasmic concentration of lead, and thereby, reducing its toxic effects on sensitive cellular functions, particularly mitochondria.

It is also not unusual during this early stage for workers to experience hyperaminoaciduria, i.e., the excessive excretion of various amino acids due to the loss of the absorptive ability of the proximal tubular cells (Ex. 23 (Weeden), Ref. Goyer). It is also well established that simply removing the worker from exposure will allow him to recover, although some workers continue to experience hyperaminoaciduria that eventually requires chelation therapy. Renal biopsies taken from those workers who are removed from exposure illustrate some interesting changes. While most of the abnormal cells in the glomeruli, arterioles, and tubules disappear and are eventually replaced by normal cells, intranuclear inclusions may still be seen. Although no definitive explanations have been given for their continued appearance, there is speculation that these intranuclear inclusions could be early indicators of the existence of a body lead burden (Ex. 23 (Weeden), Ref. Goyer). During this phase there is also a relatively high urinary output of lead but no impairment of renal function. The ultra-structural changes are probably reversible. In a second phase, after 4 or more years of exposure, the proximal tubular cells apparently have decreased their ability to form nuclear inclusion bodies and the kidneys excrete less lead and morphologically have a moderate degree of interstitial fibrosis. This phase is not characterized by any gross impairment of renal function but is doubtful whether the morphological changes are completely reversible. (Ex. 5(30))

If the lead exposure is prolonged and not alleviated, there is a progressive pathological change which eventually results in the third and final stages of frank nephropathy. Human and animal renal biopsy speciments demonstrate extensive interstitial fibrosis, sclerotic glomeruli, as well as dilated, atrophied proximal tubules. (Ex. 23(67), Ref. Goyer; Ex. 23(67); Ex. 95; Tr. 1732) At the same time, the worker may also experience hyperuricemia, blood pressure increases, with or without associated hypertensive disease, gout, or uremia, which signal the possibility of total kidney failure. (Tr. 1729; Ex. 23(67); Ex. 95) With reference to hypertension some investigations have found that arterial hypertension which is accompanied by chronic renal failure may actually precede the observed increase in blood pressure by a few years. In fact, when the biopsy evidence is evaluated in conjunction with intrarenal vasoconstriction, lead-induced hypertension becomes even more suspect. Likewise, the observed renal dysfunctions also may be associated with the vasoconstriction of renal blood vessels, which is, in the final analysis, part of the overall diagnostic picture of generalized vasoconstriction indicative of lead poisoning. (Ex. 118 (E)). Less controversial, is the occurence of saturine gout which may be the direct result of prolonged exposure to lead. That exposure affects the ability of the renal tubules to excrete uric acid into the urine, thereby accumulating in the blood to form a number of undesirable salts. Those salts are deposited in joint spaces and, hence, the worker suffers with the arthritis called gout. (Ex. 95, pp. 114–115).

Reversibility of the pathological damage and the restoration of the functional capacity of the kidney is reduced as lead exposure continues. Ultimately, the worker becomes uremic and it is increasingly more difficult to maintain the vital signs. In fact, at this point, the worker can only be kept alive through hemodialysis, the use of the artificial kidney. (TR. 1729)

c. *Kidney Function Tests.* Kidney function is clinically usually measured by the glomerular fileration rate (GFR), the level of blood urea nitrogen (BUN), and/or the serum creatinine (S-Creat) value.

The GFR is a measure of how much plasma is filtered by the kidney each minute. A reduction in GFR represents a reduction in the number of functioning nephrons which make up the kidney. Under normal circumstances, the material will pass through the kidneys at a rate of 130 ± 15 ml/1.73m^2 body surface area. (Tr. 1729) Wedeen has defined renal disease by a glomular filtration rate of less than 90 ml/min. and said there is abnormal kidney function when the GFR is less than 100 ml/min.

Elevated BUN and S-Creat values are indicative of dysfunctioning nephrons, since the two biochemical compounds normally would be removed by glomerular filtration and tubular excretions (in the case of creatinine). It should be pointed out that clinical tests of BUN and S-Creatinine are gross indicators of renal disease as they do not become elevated until over two-thirds of the kidney function is gone. (Tr. 1727) Wedeen compared this loss of kidney function to the loss which would be incurred by actually cutting away two-thirds of the kidneys. (Tr. 1776) Moreover, in uncomplicated lead nephropathy, the urinalysis is usually normal. In order to detect early renal failure, elaborate laboratory procedures, such as GFR and biopsy, are necessary.

Normal BUN values range from 10-20 mg/100 ml and normal s-creatinine values are 1.2–1.4 mg/100 ml (different investigators describe normal as being from 1.2–1.8 mg/100 ml).

d. *Studies.* Three significant studies were described during the public hearings. However, in addition to the studies described in the proposal there are present within the Record several additional studies that clearly indicate the prevalence of renal disease among workers. For example, Lilis et al. in a study of 102 lead-poisoned patients discovered signs of impaired renal function in a significant number of cases. While impairment of urea clearance appeared to be the earliest sign of renal dysfunction, creatinine clearance also deteriorated with increasing exposure. Finally, high creatininameia and persistent urea retention, which usually was accompanied by high blood pressure, developed in those workers who were chronically poisoned. Lilis et al. concluded that the functional impairment resulted from a marked vasoconstriction of the renal blood vessels (perhaps indicating the generalized vasconstriction of lead poisoning), and was probably transitory in the early stages. Prolonged exposure, however, may lead to progressive and irreversible renal damage with the subsequent development of organic lesions. These investigators noted that such clinical effects ordinarily indicated severe lead nephropathy. Undercompensated and decompensated renal failure was also found in 18 patients, most of whom had been exposed to lead for more than 10 years, and many

with a history of lead colic attacks. Arterial hypertension accompanied chronic renal failure in 13 of the cases, however functional renal impairment usually preceded the development of hypertension by several years. (Ex 118E)

In the series of 102 cases of lead poisoning studies by Lilis et al., where 18 cases of clinically verified chronic nephropathy were found, the mean blood lead level was approximately 80 μ/dl, with a range of 42 to 141 μg/dl for the whole series. (Ex 118 E.)

Another study by Lilis et al. noted the appearance of chronic lead nephropathy after a protracted evolution of lead poisoning without the presence of lead colic attacks. In this study of 14 patients with occupational lead poisoning, these investigators found a reduction in renal plasma flow. They did not, however, find evidence of generalized vasoconstriction, which emphasized a considerable and specific reaction to lead solely by the renal blood vessels. Furthermore, this reduction of renal plasma flow was even found in patients with less than 5 years of lead exposure. An additional finding of significantly lowered creatinine clearance values, only in cases with more than 5 years of exposure, indicated the progressive nature of the deterioration of the renal function. Six of 14 patients were investigated during CaEDTA treatment, and the results showed that administration of the chelating agent did definitely improve the renal plasma flow. This difference in response to treatment was dependent on the duration of exposure since those with shorter exposures to lead showed the greatest improvement to chelation therapy. (Ex. 118F).

Additional studies by other investigators also appear in the Record and provide evidence of renal disease in lead workers. (Ex. 95; Ex. 95, Ref. 84; Ex. 6(104); Ex. 27(7); Ex. 97; Ex. 284A; Ex. 6(99); Ex. 24(15); Ex. 6(33))

The results of three major studies were reported during the hearings. In the first Lilis, Fischbein et al. reported the results of a clinical field study from two secondary lead smelters during the rulemaking hearing. They examined 158 secondary lead smelter workers; 24 control workers without significant lead exposure were also studied. The experimental protocol has been partially described in the neurological section of this preamble and will not be repeated here. Suffice it to say that a careful review of each individual's occupational experience was undertaken and a broad spectrum of laboratory tests were performed, including BUN and S-Creatinine.

BUN levels were elevated (greater than 21 mg/100 ml) in 29 (18 percent) of the lead exposed workers, and there was a strong correlation with duration of exposure. A similar correlation was seen with S-creatinine. (Table 1.)

TABLE 1.—*BUN and Creatinine Levels and Duration of Lead Exposure in Secondary Smelter Workers*

Duration of lead exposure	Number examined	BUN levels			
		>21 mg/100 ml		>25 mg/100 ml	
		Number	*Percent*	*Number*	*Percent*
Less than 10 yr	137	18	13	6	4
More than 10 yr	20	11	55	4	25
Total	157	29	18	10	6

Duration of lead exposure	Number examined	Creatinine levels			
		>1.2 mg/100 ml		>1.4 mg/100 ml	
		Number	*Percent*	*Number*	*Percent*
Less than 10 yr	137	19	14	6	4
More than 10 yr	20	9	45	7	35
Total	157	28	18	13	8

A total of ten workers had (BUNs) greater than 25mg. Workers exposed for less than 10 years had experienced elevated blood lead levels at some time in the past. As a rule, more than one-third of the group had also experienced lead colic, and only a small number had been given chelation therapy. (Ex. 23 (Lilis, Fischbein))

In general, blood lead levels in the Mt. Sinai group were distributed as follows:

1. Twenty-nine percent were over 80 μg;
2. Forty-eight percent were over 60 μg;
3. Twenty-two percent were over 40 μg; and
4. One percent was less than 40 μg/100 ml.

In those workers with less than 1 year of exposure, blood lead levels were found, in varying degrees, to range from less than 40 to 80 μg/100 ml. Conversely, those workers with over 3 years of exposure showed blood lead levels in the 80 μg range, and workers with more than 10 years exposure were even higher.

Because of the rapid build-up of blood leads in some workers, as well as the widespread practice of chelation therapy, blood leads could not be significantly correlated with length of exposure. Blood leads could, however, be correlated with ZPP determinations, which in turn showed a strong relationship to the length of exposure.

Furthermore, ZPP elevations also showed some correlations to BUN and S-Creatinine increases. This is significant given that ZPP is a measure of effect rather than absorption. Therefore, an indirect relationship does exist between blood lead levels and increased renal disease. What is more important, though, is the strong relationship between length of exposure and renal disease. *At less than ten years of exposure, 24 workers have lost approximately 66 percent of their renal function—as evidenced by the dramatic increases in BUNs—and, concurrently, elevations in S-Creatinines.*

The authors reported that 26 workers were hypertensive (systolic greater than 150mm Hg and/or diastolic greater than 95mm Hg). The percentage of hypertensives increased with duration of exposure. In the group of 26 hypertensives there were 12 workers with slight or moderatley elevated BUN and 10 with elevated creatinine. The authors concluded:

> The concurrent finding of elevated blood lead and zinc protoporphyrin levels after similar durations of exposure confirmed the relatively rapid build-up of toxic lead levels. As expected, however, longer lead exposure was associated with greater prevalence of disease, and more severe abnormalities. In some cases, evidence of kidney damage (elevated BUN and creatinine levels), hypertension and clinical signs of peripheral neuropathy were found. (Ex. 23 (Lilis, Fischbein), pp. 98–99)

A subgroup in the study was defined as all workers who were found at the time of examination to have (1) blood lead levels of less than 80 μg/100 ml (2) who had never been notified in the past that their blood lead level had

been excessive and, (3) who had never received chelation therapy. BUN levels in this proup of 48 workers were found to be in the normal range, defined as not exceeding 20 mg/100 ml, with 2 (4 percent) exceptions (23 and 25 mg/100 ml. There was no correlation of BUN levels and blood lead values in this range (r=0.019). Nevertheless, when the relationship of BUN and ZPP levels was analyzed, it was found that with increasing ZPP levels the Bun values tended to be more elevated, with a statistically significant correlation factor of 0.20. For the entire group of lead smelter workers, the prevalence of an elevated BUN was found to be 18 percent, and there was a correlation with duration of exposure (r=0.37). Most individuals with elevated BUN had registered high blood lead levels in the past, many had experienced lead colic, sometimes repeatedly, more than two-thirds had undergone chelation therapy.

Since only 4 percent of the workers had elevated BUN in the subgroup described above (blood lead levels of less than 80 μg/100 ml and no history of high blood lead levels in the past), and since the majority of them had had very short duration of exposure, it was not surprising that their BUN was, for the majority of cases, in the normal range.

In the subgroup six cases (13 percent) the creatinine value exceeded 1.2 mg/100 ml, but in only one case (2 percent) was creatinine in excess of 1.4 mg/100 ml found. Among the six cases there was a weak correlation between blood lead levels and creatinine levels; blood creatinine. A level of 1.3 was found in only one worker with a blood lead level of less than 59 gm/100 ml, had while in five workers with blood lead levels of 60 to 79 gm/100 ml creatinine levels over 1.2 mg/100 ml.

Since elevation of BUN and S-creatinine occurs only when about two-thirds of kidney function is lost , these results must be taken very seriously. That is, there were two cases of BUN elevation and six cases of creatinine levels over 1.2 mg/100 ml in men whose blood leads were below 80 μg/100 ml.

The authors concluded:

> When the relationship of creatinine and ZPP levels was analyzed, it was found to be similar to that described for BUN, with a possibly stronger correlation (r=.28).
>
> These observations concerning a possible correlation of BUN and creatinine levels with ZPP concentrations in this group of workers with blood lead levels of less than 80 μg/100 ml and no history of elevated blood lead levels in the past are of interest and suggest the need for further investigations along these lines, to help delineate the course of renal disease associated with lead exposure. (Ex. 24 (Lilis et al.), p. 9)

* * * * *

> Evidence has been reported concerning the development of nephrophathy with long-confirmed lead absorption.
>
> The results of this survey suggest that metabolically active lead may have an earlier impact on renal function than heretofore believed. While in the majority of cases BUN and creatinine were in the normal range, there was nevertheless a correlation between ZPP levels and both BUN and creatinine. The mechanism through which the nephrotoxic effect occurs is not yet clear; one possiblity is vasoconstriction affecting the afferent renal arterioles predominantly. (Ex. 24 (Lilis et al.), p. 15)

The second major study which demonstrated lead nephropathy in workers was carried out by NIOSH. NIOSH reported the results of a Health Hazard Evaluation at Eagle Picher Industries, Inc. in November 1975. They determined that symptoms consistent with lead intoxication as well as signs of anemia, peripheral neuropathy and kidney disease were present in workers exposed to lead. A discussion of that report and a supplemental medical study follow.

Eagle Picher Industries' Joplin, Mo., plant produces lead oxide, lead peroxide, lead sulfate, lead silicate and blue lead. Medical evaluations of 53 production workers at this plant revealed blood lead levels ranging from 39 to 135 μg/100 ml, with 44 (83 percent) greater than or equal to 60 μg/100 ml and 19 (36 percent) greater than or equal to 80 μg/100 ml (Ex. 38C). Comparable levels of erythrocyte protoporphyrin were noted. Blood urea nitrogen (BUN) levels were elevated in 17 workers. (Table 2)

TABLE 2.—*Workers With Elevated BUN Levels*

Worker numbers	BUN		Creatine	Years employed	Number of courses of EDTA
	March	May			
1	—	44	—	—	8
2	—	30	—	—	0
3	28	30	1.1	23	1
4	30	28	1.2	23	4
5	26	24	1.3	7	2
6	21	27	.9	16	9
7	23	27	1.0	13	0
8	21	23	1.2	25	0
9	—	26	—	—	0
10	25	—	1.3	20	0
11	23	—	1.0	20	0
12	21	18	1.0	21	0
13	23	18	1.0	7	0
14	23	17	1.2	29	1
15	17	28	.9	4.5	1
16	20	23	1.4	31	13
17	18	24	1.2	20	4

Given these findings NIOSH made the following statements with respect to those men with elevated BUN:

> Findings consistent with those noted in lead intoxication were noted in the exposed workers examined. These included symptoms of lead toxicity, anemia, peripheral neuropathy and renal disease.

* * * * *

> The results of additional studies are needed to determine if significant kidney disease exists in these workers and if it is related to occupational lead exposure or to EDTA therapy. These studies are currently in process. (Ex. 38C, p. 6)

Based upon the BUN results in this earlier study NIOSH conducted a followup medical evaluation to determine the extent of renal functional impairment in these workers and the role of occupational lead exposure in the etiology of this disease. The 19 workers (including 2 borderline cases) were referred to a board certified nephrologist for outpatient diagnostic studies.

Following complete history and physical examination, blood and urine tests were performed on specimens from each worker. In evaluating renal concentrating ability, the osmolality of a urine sample collected after a 12-hour water fast was determined. Creatinine and lead clearances were determined using 1-hour timed urine collections and simultaneously collected blood samples. Blood lead levels were determined. Blood chemistry tests (including creatinine, BUN, and uric acid) were performed.

Five of the 19 workers tested had elevated BUN levels (greater than 22 mg/100 ml) and one had an elevated serum creatinine concentration (greater than 1.5 mg/100 ml). However, 8 (42 percent) had decreased creatinine clearance (less than 91 ml/min/1.73 sq m BSA). Impaired urine concentrating ability (i.e., inability to concentrate the urine above 800 mosm/liter after

an overnight water fast) was found in 8 of 15 workers tested.

Lead clearance tended to decrease with the increasing duration of exposure to lead. This inability to clear lead was independent of the age of the worker: analysis of data for 45 to 55 year-old men shows the same negative relationship between duration of exposure and clearance rate. (Table 3)

TABLE 3.—*Results of Renal Function Test, Missouri, 1976*

Subject	Age	Duration of lead exposure (years)	Blood lead level (μg/100 ml)	Creatinine clearance (ml/min/ 1.73. sq. m BSA)	Fasting urine osmolality (mosm/liter)	Lead clearance rate* (ml/ min)
1	56	7	154	85	.....	0.07
2	43	20	66	142	.....	.82
3	37	20	35	82	871	1.46
4	47	23	71	72	588	.10
5	45	8	87	128	1,020	.61
6	53	23	61	91	1,025	.97
7	52	26	61	115	650	.70
8	38	20	123	109	608	.94
9	60	25	75	75	278	.30
10	42	21	66	96	1,180	.36
11	47	7	48	108	820	1.09
12	53	29	96	89	708	.10
13	35	13	56	109	966	1.13
14	62	16	105	97	912	1.01
15	52	25	78	73	286	.25
16	53	20	92	108	912	.04
17	51	7	80	65	704	2.04
18	52	31	55	43	652	.80
19	29	4.5	58	112	1,114	.92
Normal range			60	91	800–1,300	

Given these results, NIOSH concluded:

"These studies clearly demonstrate a significantly increased prevalence of mildly to moderately severe kidney disease in employees at the Eagle Picher plant in Joplin, Missouri. Although further studies are needed to clarify the cause of these disorders, lead nephropathy is a likely etiology for several reasons. These workers have been heavily exposed to lead for prolonged periods (5–30 years) and manifest other toxic sequelae of lead exposure including anemia, recurrent colic and joint symptoms. Both renal glomerular and tubular dysfunction were noted in these men, a pattern previously noted in other studies of lead nephropathy. A positive relationship exists between the degree of renal dysfunction (impaired lead clearance) and duration of exposure to lead; and effect which is independent of age. In view of these findings, other etiologies of renal disease seem unlikely but must be ruled out with further testing.

This study illustrates the insensitivity of blood urea nitrogen and serum creatinine determinations and routine urinalysis in detecting renal disease. This finding has been well-documented in the medical literature. More sensitive measures should be used in screening for lead nephropathy in exposed populations; such tests could include measurement of lead clearance, creatinine clearance, and urine concentrating ability as were done in the current report. All testing was performed over several hours in a doctor's office and could be adapted to in-plant screening. (Ex. 79, p. 3)

OSHA considered the results of these clinical investigations to be of extreme importance for three reasons. First, the data indicates that the workers had damage to the tubules of the kidney which negatively affected the kidney's ability to concentrate urine and to excrete lead. Since lead excretion decreases with kidney damage, the use of urinary lead levels to monitor workers is impossible and in fact potentially dangerous. If kidneys were impaired and as a result urinary lead appeared normal, damage might go undetected and expose the worker to further insult.

Second, 7 of 53 people (13 percent) had renal impairment. (Tr. 1348). Third, these results further indicate the insensitivity of BUN, S-Creat, and routine urinalysis in detecting early renal disease.

Dr. Richard P. Wedeen, a board-certified specialist in internal medicine and nephrology, testified on the role of lead in the development of nephropathy in the United States during the rulemaking hearings:

To the best of my knowledge, we have identified the only well documented cases of occupational lead nephropathy in the U.S. We have used sophisticated physiological techniques called "clearances" requiring from 4 to 12 hours of the patients' time and many more hours of laboratory analyses to measure kidney function. In selected cases, we have performed renal biopsies in order to confirm the diagnosis of lead nephropathy and to exclude other possible causes of kidney disease.

We have identified 19 cases of nephropathy among 51 lead workers whose kidney function was examined. Thirteen of the nineteen men worked in a lead smelting plant, three worked as lead burners, two cleaned up spent bullets in pistol firing ranges, and one prepared solder creams from molten lead. All of these workers lived and worked in northern New Jersey and had been occupationally exposed to lead for from 3 to 34 years. All had been removed from exposure to lead for at least a few weeks at the time we examined them. (Tr. 1735–36)

Wedeen testified that this method was used for two reasons:

First, physical signs and symptoms of renal failure ordinarily are not seen until more than three-fourths of kidney function is lost.

The clinical tests of renal function normally available in any physician's office are too inaccurate or insensitive to detect moderate decreases reliably in GFR. The blood-urea-nitrogen and serum creatinine levels are only increased when more than about two-thirds of kidney function is lost. Moreover, in uncomplicated lead nephropathy, the urinalysis is usually entirely normal. So, in order to detect early renal failure, elaborate laboratory procedures are necessary.

What about advanced renal failure? This is, of course, what we are interested in preventing. The great difficulty with end-stage renal disease due to lead is that there is no way of proving the cause of the disease once it has progressed to the point at which dislysis is required to sustain life. The techniques we have used are essential to detect kidney damage up to 60 percent loss of function. Between 60 and 85 percent loss of function can be detected by routine laboratory procedures. More than 85 percent loss of function results in symptoms of kidney failure called uremia. However, when the disease has progressed to this point, it is extremely difficult to establish the cause, and reversibility is unlikely. (Tr. 1737–38)

In these 19 workers, 4 had a blood lead level above 60 μg/100 ml, and one had a blood lead greater than 80 μg/100 ml at the time of examination. Most of these men had been removed from lead exposure for some period of time. Based upon this evidence, Wedeen and coworkers concluded that blood lead levels were an inadequate measure of lead absorption for purposes of predicting renal disease(s) in workers no longer exposed to lead. (Tr. 1738)

Nineteen of the fifty-one workers whose kidney function was examined had reduced GFR's and in 10 men there was renal biopsy evidence of tubular damage consistent with lead nephropathy. Wedeen eliminated from consideration those who had other possible causes of renal dysfunction such as age over 55 (4) or hypertension (2). This left 13 of the 19 cases. They then eliminated those referred because of medical symptoms, leaving nine (13 percent) medically unselected lead workers who had lead nephro-

pathy. This is the same incidence of renal impairment as that seen in the Eagle Pitcher study.

The blood lead levels of these workers disgnosed as having lead nephropathy were, with one exception, below 80 μg/100 ml and with four exceptions, below 60 μg/100 ml. Blood lead levels may have been elevated at some time in the past and there was some evidence of this from records obtained. These authors point out that PbB is "of little value to the physician seeking to determine the body burden of lead or the cause of symptoms or signs after lead exposure has ceased." (Tr. 1444) Dr. Wedeen's conclusions are given here in their entirety since OSHA believes they most accurately describe the issues associated with prevention of mortality and morbidity from lead produced nephropathy.

I would now like to address myself to the question of why occupational lead nephropathy has not previously been detected in the United States. I think occupational lead nephropathy has been overlooked because:

1. When lead nephropathy becomes symptomatic, the patient leaves his job and is lost to follow-up.

2. When lead nephropathy is advanced, hypertension develops and is considered the cause of renal failure.

3. Routine clinical tests will not indicate the presence of kidney disease until lead nephropathy is advanced. Blood tests such as the blood-urea-nitrogen and serum creatinine are increased only when two-thirds of kidney function is lost. The urinanalysis which is routinely used to screen for kidney disease is normal in early lead nephropathy. Thus, moderate renal disease is not routinely detected in the physicial's office.

4. Perhaps of most importance in preventing diagnosis of lead nephropathy has been the reliance on blood lead concentrations to make the diagnosis of lead poisoning. The astute physician who sees lead workers with renal disease will naturally consider the possibility of lead nephropathy. He will then go to his reference books to look for the criteria for diagnosing lead poisoning. When he finds that the textbooks and Federal guidelines agree that when the blood lead is under 80 μg percent there is no lead poisoning, he discards that diagnosis. Exactly the same error occurs when he looks at urine ALA or urine lead concentration in workers whose illness has taken them away from lead exposure. He has excluded the diagnosis of occupational lead nephropathy and is usually compelled to make the diagnosis of pyelonephritis or hypertension. Even the most competent physician has no way of knowing that his criteria are inappropriate, since they come from the most authoritative sources. The physician has extreme difficulty in diagnosing lead nephropathy as long as the criteria for diagnosis are misleading.

I have reported today 19 lead workers who have lost 30 to 50 percent of their kidney function. Since they showed no symptons and had no routine laboratory evidence of kidney disease, it may be asked why this kidney function loss should be viewed as material damage. Lead nephropathy is important because the worker has lost the functional reserve, the safety, provided by two normal kidneys. If one kidney becomes damaged, the normal person has another to rely upon. The lead worker with 50 percent loss of kidney function has no such security. Future loss of kidney function will normally occur with increasing age, and may be accelerated by hypertension or infection. The usual life processes will bring the lead worker to the point of uremia, while the normal individual still has considerable renal functional reserve. Loss of a kidney is therefore more serious than loss of an arm, for example. Loss of an arm leads to obvious limitations in activity. Loss of a kidney or an equivalent loss of kidney function means the lead worker's ability to survive the biologic events of life is severely reduced. By the time lead nephropathy can be detected by usual clinical procedures, enormous and irreparable damage has been sustained. The lead standard must be directed towards limiting exposure so that occupational lead nephropathy does not occur. (Tr. 1747-1750.)

Dr. Wedeen concluded that a *minimum* of 10 percent of American lead workers have occupational lead nephropathy.

It can therefore be anticipated that at least 10 percent of the American lead workers have occupational lead nephropathy. This is assuming of course that the exposure in other lead workers is comparable to what exists in New Jersey. This is in fact a minimal estimate. We have excluded from renal function studies 19 workers with gout, hypertension, or kidney stones, although each of these conditions is a well known complication of lead poisoning.

We also eliminated from detailed study six lead workers with other medical conditions possibly associated with renal failure. Thus 25 additional men of the 141 workers screened for excessive body lead burdens may have had renal disease, but it would have been impossible to exclude causes other than lead.

Renal function was therefore not studied in these 25 workers. The statement that 10 percent of unselected lead workers have occupational lead nephropathy therefore represents a minimal estimate of the incidence of this disease assuming only that industrial health precautions in the northern New Jersey plants in which our patients work are typical of the United States.

NIOSH estimates that about one million American workers are exposed to lead. According to this estimate, there may be 100 thousand cases of preventable renal disease due to occupational exposure to lead in this country.

It might be worth contemplating the national cost in dollars if hemodialysis had to be provided to this group of lead workers, compared to the cost of supporting definitive research and prevention. If only 10 percent of these hundred thousand workers with occupational lead nephropathy came to chronic hemodialysis, the cost to Medicare would be about $200 million dollars per year. (Tr. 1740-1742.)

OSHA concurs with the findings of these three studies described and believes that kidney disease associated with lead exposure is far more prevalent than previously recognized. In addition to this general conclusion OSHA also recognizes the difficulty in early detection of the disease given the insensitivity of routine screening tests, the inapplicability of the GFR for routine purposes and the limitations of blood lead determinations. Therefore it is incumbent on the Agency to set a standard which in general reduces the exposure to the worker in order to prevent development of the disease as well as establishes a reasonable margin of safety. These points were clearly articulated by Dr. Bridbord of NIOSH during his cross examination in the hearings. In discussing the insensitivity of these tests Mr. Becker, USWA, questioned Dr. Bridbord about the need for some margin of safety in protecting workers from renal dysfunction.

Mr. BECKER. Dr. Bridbord, you stated the other day a personal feeling that perhaps a level of somewhat lower than 60 would be more appropriate to protect lead workers * * * what do you feel the risk is of renal dysfunction at blood lead levels, at continued exposure of blood lead levels of say 60 micrograms and 80 micrograms.

Dr. BRIDBORD. I think that would be really hard to put into quantitative terms. I think it's more of a question of what I feel would protect against that kind of effect.

In other words, I would view those effects as extremely severe. Some individuals, physicians included, might debate the true health significance for example, of increase in zinc protoporphyrin or increased ALA in the urine etc. I think there can be very little question of the severity of the effect involved with damage to the kidneys which is one of the most vital organs in the entire body.

So I think it's not so much that I have data or that anyone has data which would say precisely here is the actual quantitative risk. * * * I think that the important point from the data we see that at least there appears to be a legitimate question and a legitimate suggestion that such effects certainly occur below 80 blood lead and may well occur below 60 and that the point is if blood lead maximum in my personal opinion, of 60 just does not provide a margin of safety to assure that people are being protected against an extremely severe effect. * * *

Dr. BECKER. Dr. Epstein said yesterday that he felt a standard should apply a margin of safety of 10 times. That he felt, and I don't know what background or scientific basis he was relating to. If I take it then from your comments that certainly the standard of 60 would not provided a 10 fold margin of safety against renal disease.

Dr. BRIDBORD. Certainly not. A 10 fold margin of safety I'm not even sure a standard of 40 would give you a 10 fold margin of safety but there would at least be a certain rationale in terms of protecting against the earliest changes that we feel might be significant in the case of lead but that would be the mechanism that would provide that increased margin of safety but it certainly would not be a 10 fold margin of safety. (Tr. 1839-40)

It is important to note that in the Eagle Pitcher study by NIOSH (Ex. 38(6)) there were a number of workers who had blood lead levels which were

below 80 μg/100 ml, and these men were currently exposed to lead. There was no indication of major changes in the plant over a number of years. In noting these facts Dr. Bridbord questioned Dr. Wedeen on the issue of chronicity of exposure.

Dr. Bridbord. Well, looking at a group of workers, currently employed, having a bloodlead level on that worker and having some information, that to the best of our knowledge there were no major changes in that particular plant during the past number of years. Would that not be a somewhat better index of what the blood lead levels might have been in the past. Considering too, that these workers are currently employed.

Dr. Wedeen. Sure I think that the blood level measured close to the time of exposure is probably more reflective. I worry very much, that this may occur after a few months of exposure and the blood lead level may remain the same for the next 20 years, despite the fact that the individual is continually accumulating lead in the body.

Dr. Bridbord. Would you think that the chronicity of lead exposure, apart from precisely whether the blood lead was above or below 80 or above or below 60 for example, might be an important factor in determining the eventual development of renal disease in lead workers.

Dr. Wedeen. Yes, That is just what I meant, that the accumlative effects and the cumulative body burden may be very different from the blood lead level at any moment in time.

In other words, one could certainly imagine that a blood lead level of 80, for 2 years, may be very similar to a blood lead level of 40, for 4 years. I don't have that data, but something like that may well exist in terms of the danger of the different levels of exposure.

Dr. Bridbord. Alright. Paricularly, in view of that, and given the requirements of the Occupational Safety and Health Act, that sets standards which protect during the working lifetime, would you have some reservations about a blood lead maximum standard, even at 60.

Dr. WEDEEN. I certainly would. And I think I just expressed the basis for it. You will note that in my recording of these patients, very very few of them had blood lead levels over 60. I just feel that while the blood lead level is maybe better than nothing, it may be very practical. It probably doesn't do the job we are trying to do and certainly not from the physicans point of view, who has seen the individual patient, who may or may not be a current exposure at the level that got his disease. (Tr. 1765-1766)

Based on the studies presented and the subsequent comments by Bridbord and Wedeen OSHA agrees with Dr. Wedeen's assessment of the level required to prevent lead nephropathy. "40 μg/100 ml is the upper acceptable limit." (Tr. 1771) Caution must be used in interpreting Dr. Wedeen's statement since he has previously stated blood lead levels are inadequate measures of possible lead nephropathy. OSHA interprets Dr. Wedeen's statement to mean that in order to prevent the development of renal failure over a working lifetime an absorption which results in PbB levels of 40 μg/100 ml is required as an upper limit.

In estimating that there may be 100,000 cases of preventable renal disease due to occupational exposure to lead in this country, Wedeen failed to mention mortality from hypertension or related disease, focusing instead on renal disease. OSHA recognizes that it is difficult to separate one from the other. As Wedeen testified:

"Occupational lead nephropathy is that renal disease is associated with a number of complications and these complications may also cause renal disease. In paricular I would like to mention hypertension, a very important problem, well recognized in this country.

High blood pressure can cause renal disease, but renal disease often causes high blood pressure. This means that in the presence of high blood pressure it can be very difficult to prove what caused the disease. (Tr. 1731)

OSHA's interpretation of the Cooper-Gaffery study reinforces this concern. The agency concludes there is good evidence in smelter populations that mortality due to CNS vascular disease and hypertensive cardiovascular-renal disease is excessive in smelter workers and probably has a work related etiology. OSHA believes there is the possibility of excess mortality from hypertensive vascular disease in the battery manufacturing population as well. The Mt. Sinai group also noted in increased prevalance of hypertension in their study population as well.

It is apparent that further investigations are required in this area although OSHA acknowledges that there are other contributing causes to hypertension which are confounding. The reverse problem is also true however. Physicans may exclude a diagnosis of occupational lead nephropathy because of the lack of clinical indicators. This compels the physician to make a diagonsis of essential hypertension unrelated to lead. OSHA believes that hypertension and renal disease associated with lead exposure has been underestimated and thereby has been the number of workers afflicted with renal disease in the U.S.

During the hearings Dr. Charles Hine, Medical Director of ASARCO, criticized Dr. Wedeen's study in some detail. His testimony and Dr. Wedeen's response is summarized as follows:

Hine:

The kidney, like other organs of the body, has a considerable reserve. At any particular time, only about half of the approximately one million nephron units are functioning. Therefore, the normal kidney can function entirely satifactorily with less than 60 percent of its units working. For example, the removal of one kidney as is done in kidney transplants, with a resulting reduction of 50 percent of pre-surgical function, does not impair the renal function of the donor. (Ex. 218A, p. 4)

Wedeen:

Dr. Hine implies that loss of 50 percent of renal function is not a loss of renal function. This is patently absurd. The decision to accept 50 percent loss of kidney function (as in a living transplant donor) should properly be left to the individual potential donor. (Ex. 257, p. 1)

Hine:

In his publication in the American Journal of Medicine (1975) and throughout his presentation to this group, Dr. Wedeen has referred to occupational lead nephropathy, describing his observations on some 69 lead workers. I believe that this is an incorrect term for the following reasons: Nephropathy, by definition, is a disease of the kidney. None of the men he examined had symptoms of kidney disease, although four were reported to have symptoms and signs of lead poisoning. (Ex. 218A, p. 4)

In other words, Dr. Wedeen is not telling us about kidney disease, he is simply documenting a decrease in kidney function as measured by one test. This decrease in function may be temporary and reversible or permanent and irreversible. (Ex. 218A, p. 5)

Wedeen:

As for the term "nephropathy," this simply means "disease of the kidney". In lay as well as medical circles, loss of function is commonly considered disease. Moreover, many diseases are relatively asymptomatic, particularly in their early phases, e.g., hypertension, diabetes, etc.

It should further be pointed out that lead nephropathy was not determined by "one test" (p. 5). In each case, the significance of the reduction in GFR was supported by comparable reductions in effective renal plasma flow (CPAH). In nine patients, the diagnosis of lead nephropathy was confirmed and other possible etiologies further excluded by renal biopsy.

At the time of my presentation at the OSHA hearings, I reported that one lead worker had shown definite improvement, and three showed no deterioration in kidney function following prolonged chelation therapy. Newly acquired follow-up data on eight treated patients show that all have had a progressive improvement in kidney function. The increase in GFR in response to EDTA therapy both confirms the etiology and indicates the importance of detecting lead nephropathy before it has reached the endstage. (Ex. 257, p. 1)

Hine:

Dr. Wedeen refers to "abnormal EDTA mobilization tests". What is an abnormal EDTA test? Anyone who receives EDTA in sufficient quantities will excrete an increased quantity of lead beyond that which he normally excretes. In my opinion, any quantity of lead greater than 500 μg/l suggest a past exposure to lead greater than that of the general population. An "excessive burden" then is a matter of clinical judgement and experience. (Ex. 218A, p. 8)

Wedeen:

What is an abnormal EDTA mobilization test (p. 8). This was defined in our own control group and the literature in the 1975

American Journal of Medicine paper as less than 650 μg Pb/day. Greater than 1000 μg Pb/day during the EDTA lead-mobilization test was considered abnormal, i.e., suspect, in our OSHA testimony.

The value of the EDTA lead-mobilization test was well documented in my OSHA presentation and need not be reiterated here.

Dr. Hine's statements concerning EDTA therapy (pp. 9–10) are not supported by our findings. (Ex. 257, p. 2)

Hine:

The response of the kidney to the adverse effects of injurious substances is limited and a number of different etiological factors will produce the same type of chemical, biochemical and physiological manifestations of disease. Dr. Wedeen described some observations in his patients which are pertinent to his conclusions, regarding the extent of change due to lead per se, the validity of the diagnosis of nephropathy and the overall significance of his findings. These are:

(i) Specific lead-induced intranuclear inclusion bodies reported by others were not observed by him in biopsy of the kidneys in his group.

(ii) Glomerular changes, arteriolar damage and loss of proximal tubular brush borders were absent in all but the most severe case.

(iii) Clearance data did not reflect defects of tubular transport, of sodium, water, phosphate, or urate.

(iv) In contrast to others who have observed that lead poisoning actually enhances PAH excretion, a tubular defect in excretion, was observed in his patient.

(v) Aminoaciduria of a low degree has been reported as a functional manifestation of increased lead absorption. On the contrary, in the one patient on whom amino acid excretion was measured, only 50% of the maximum quantity of amino acids appeared in the urine. (Ex. 218A, p. 9)

Wedeen:

Dr. Hine makes six final points he believes raise questions about the diagnosis of lead nephropathy. I will respond to each of these briefly.

Page 9, No. 7 (i): Re "intranuclear inclusions." The disappearance of intranuclear inclusions in lead nephropathy has been noted by Cramer, Goyer, *et al.* in adults (Brit J Indust Med 31:113, 1974), and by Goyer in experimental animals (Lab Invest 32:149, 1975).

ibid (ii): Histologic changes in proximal tubules consistent with lead nephropathy were present in all ten biopsied kidneys.

ibid (iii): The absence of *multiple* renal defects detectable by clearance methodology undoubtedly reflects only the limitations of this physiologic technique.

ibid (iv): The PAH transport defect may be a transient phenomenon peculiar to acute lead poisoning.

ibid (v): Aminoaciduria has been demonstrated only in large groups of lead exposed individuals compared to unleaded subjects. These findings have no bearing on an individual case. Aminoaciduria is *not* a criterion for diagnosis for lead nephropathy in adults, although it is rather consistently found in children.

ibid (vi): Normal renal concentrating ability is characteristic of early lead nephropathy and helps distinguish this disease from other renal disease. (Ex. 257, pp. 2-3)

Hine:

We were surprised to see no data on creatinine clearance, since this commonly utilized procedure lends itself to less exacting control of the patient and is more adaptable to the screening of large numbers of persons. (Ex. 218A, pp. 5–6)

Wedeen:

Creatinine clearances were not reported because they showed no correlation with the more accurate measure of GFR used. The well known error of creatinine clearance measurements even under "metabolic ward" conditions is increased under the outpatient field conditions used in this study. (Ex. 257, pp. 1–2)

Hine:

On page 8 of his presentation, Dr. Wedeen refers to measurement of GFR in 41 unselected lead workers. In 28 workers who had normal EDTA tests, apparently no measurement was made. Unfortunately, other negative controls were also not studied at the same time with the same technique. Comparable data on the GFR of persons with similar characteristics but with different occupations is thus not known. Such measurements assume a special significance when comprehensive studies of this type are being carried out in a group suspected of having a problem. The importance of negative control data cannot be emphasized too strongly. (Ex. 218A, p. 6)

Wedeen:

The importance of negative control data cannot be emphasized too strongly (p. 6). By current standards, it would be clearly unethical to obtain clearance data in subjects in whom no disease was suspected. It is therefore appropriate and necessary to use "normal" data taken from the literature. In eight patients we now have better than "negative control data." These patients served as their own controls, and each showed improvement in kidney function following specific therapy.

Because of the obvious limitations in obtaining physiologic data in humans, diagnosis by exclusion necessarily remains the mainstay of clinical medicine. In six patients diagnosis was established by exclusion, in eight by specific therapeutic response, and in seven, other possible causes of decreased renal function were recognized. (Ex. 257, p. 2)

Hine:

Dr. Wedeen's reference to the estimated numbers of lead workers who may be in need of hemodialysis—10,000 or his estimated 100,000 with "lead nephropathy" seems incongruous in terms of:

(i) The improvement in lead hygiene which has occurred throughout the country in the last 20 years.

(ii) The few cases of documented lead nephropathy which are known to occur.

(iii) The suggestion that a modest decrease in GFR is going to result in any significant number of cases of persons with end-stage kidney disease. (Ex. 218A, pp. 10–11)

Weeden:

My comments on the national impact of occupational lead nephropathy were indeed speculative. However, my estimate of at least a 10 percent incidence of the disease were made after excluding symptomatic lead workers from consideration. Other American workers may therefore have lead exposure comparable to that in New Jersey despite "the improvement in lead hygiene which has occurred throughout the country in the last 20 years (p. 11).

Dr. Hine's interpretation of the other medical literature on this subject is also highly questionable, in my view. As long as lead exposure is evaluated only by blood lead levels and lead nephropathy is recognized by inappropriate diagnostic criteria, the impact of this disease on lead workers will remain obscure. (Ex. 257, p. 3)

These arguments in essence stand on their own in OSHA's view and little comment is required. In evaluating these remarks OSHA has given significant weight to Dr. Wedeen's background and expertise in the field of kidney disease. OSHA believes that Dr. Wedeen has more than adequately responded to the issues raised by Dr. Hine and the agency accepts the testimony of Dr. Wedeen as being an accurate representation of the state of occupationally related kidney disease from exposure to lead.

In conclusion, OSHA believes that Dr. Wedeen and coworkers have presented important data that demonstrate that lead exposure is related to kidney disease in long term exposures. This work, coupled with the studies described earlier in this section, elucidates the presence of lead induced renal disease. These investigations raise the specter of a high prevalence of a lead induced renal disease previously thought to be rare in occupational settings in the U.S. While accepting the uncertainties associated with the few modern studies available, OSHA believes that a standard which maintains PbB levels at 40 μg/100g is both prudent and reasonable, and would provide a reasonable margin of safety against the development of chronic renal disease.

5. *Reproductive System.* a. *Introduction.* During the hearings, extensive testimony was presented concerning the effect lead exposure has on the reproductive process prior to conception, at conception, during pregnancy, on the fetus and newborn child, and on the children of lead exposed workers. The evidence indicates that lead has a profound, adverse effect on the course of reproduction. Lead exposure affects the reproductive system of both males and females, by causing genetic, gametotoxic, intrauterine, and extrauterine effects. In lay terms, this means that lead can: (1) Adversely affect the chromosomes; (2) damage the sperm or egg cells prior to conception; (3) affect the developing embryo during pregnancy; or (4) affect the developing baby of an exposed mother who is breast feeding.

Ms. Andrea Hricko prepared a flowsheet for the hearings which describes the chronology of potential adverse effects:

FIGURE 1.

CHRONOLOGY OF POTENTIAL ADVERSE EFFECTS OF JOB EXPOSURES ON REPRODUCTION OR ON THE ABILITY TO HAVE NORMAL, HEALTHY CHILDREN

PRIOR TO CONCEPTION	AT CONCEPTION	DURING PREGNANCY	ON THE NEWBORN	ON THE CHILD
Menstrual disorder - women Interference with sexual functions - men Lowered fertility - men and women Genetic damage in male and female germ cells, can be passed on to children and result in disease or birth defects. Can also cause miscarriage or stillbirth.	Difficulties in conceiving a child (for example, by interference with sperm's ability to fertilize the egg)	Miscarriage, stillbirth, cancer, disease, or birth defects--as a result of substances crossing the mother's placenta and reaching the developing fetus	Toxic effects on development of baby as a result of chemicals transmitted to child in mother's breast milk	Toxic effects on development of child from exposur to substances inadvertently brougl home on parents' workclothes

Evidence has existed for over a century that lead has profound reproductive effects:

> During the late nineteenth and early twentieth centuries, women in the pottery and white lead industries felt lead was an abortifacient. Over 100 years ago, they knew that women in lead work were more likely to be sterile; that if they became pregnant they were more likely to miscarry; that if the pregnancy went to term it was more likely to end in stillbirth; and that if the child was born living, that death was more likely to come in the first year of life. (Ex. 233; p. 1)

This older work, coupled with more recent data on adverse reproductive effects, caused OSHA to address the issue of increased susceptibility of women of childbearing age in its proposed lead standard.

> Recent studies of the toxicological effects of exposure to lead indicate certain groups of adult workers may have greater susceptibility to lead intoxication than the general worker population. One such group is female employees of childbearing age. It is known that lead absorbed into the bloodstream of pregnant women crosses the placental barrier and enters the blood of the fetus. This is of great concern because excessive exposure to lead during pregnancy has caused neurological damage in children. As noted in the Academy's report, the risk to the fetus from intrauterine exposure to high levels of lead in the mother's blood is maximal in the first trimester of pregnancy when the condition of pregnancy may not be known with certainty. It has also been established that the umbilical blood lead concentration in the fetus is similar to that found in the mother's blood. This raises the serious possibility that the blood lead level in the mother might harm the fetus, without producing any clinical symptoms of lead exposure in the mother.
>
> The extensive data on lead intoxication in children indicate that for several reasons, including their rapid growth, children may be susceptible to lead intoxication at lower blood lead levels than adults. The U.S. Public Health Service considered this and other factors when it recommended, in March 1975, that blood lead levels in children be kept below 30 μg/100 g. (Ex. 2, p. 45935).

There was evidence presented at the hearings that exposure to lead is associated with adverse reproductive effects, and that females per se are not more susceptible than males, as was maintained in the proposed standard. The principal issues addressed were:

(1) Whether low blood-lead levels were associated with adverse effects on male fertility.

(2) Whether genetic damage in male and female germ cells occurs from exposure to lead and whether the results can cause failure of implantation, miscarriage, stillbirth, or be passed on to children and result in disease or birth defects.

(3) Whether OSHA should establish a standard which protects the fetus from the harmful effects of lead, and if so, what blood lead levels are required to protect the fetus.

There was extensive discussion on the issue of equal employment for women in the lead industry. The lead industry argued that OSHA is not obligated to set a health standard which would insure equal employment for all persons; that is, OSHA is not obligated to protect the fetus. This issue will be addressed in the permissible exposure limit section following a review of the health effects here.

Following an evaluation of the record, OSHA has concluded that the agency must set a standard which, to the degree feasible, protects the fetus as well as working adults. This conclusion is based on the knowledge that lead crosses the placental membrane and can adversely affect the fetus. Given the health effects data, in order to protect the fetus, the blood lead level of the parent should be kept below 30 μg/100 g. In addition, OSHA has concluded that the fetus is not most vulnerable during the first trimester, rather, the growing fetus is vulnerable whatever its stage of development.

OSHA also concludes that the record provides evidence which is indicative of adverse effects on males prior to conception. In particular, OSHA believes that Lancranjan has demonstrated an adverse effect on spermatogenesis—including teratospermia, asthenospermia and hypospermia—in workers exposed to lead at low blood lead levels. These same workers demonstrated difficulties in erection and ejaculation, a reduction of orgasm, as well as decrease in libido. OSHA agrees with Lancranjan's conclusion that:

> It is our impression that the endocrine system is one of the more sensitive structures to the noxious agents of places of work. Among the components of the endocrine system, the male gonad is one of the glands most sensitive to the noxious environment. (Ex. 23 (Lancranjan), p. 396)

This final standard must be set to protect men from the effects of lead on fertility, as well as to protect the fetus and women from lead-induced effects.

Both human and animal studies suggest that mutagenic effects occur from exposure to lead. There is evidence of chromosome aberrations in both humans and animals exposed to lead. While these chromosomal abnormalities do not have a clearly defined biological significance, they may be related to reproductive failure.

Data from both human and animal studies indicate that lead exerts genetic, gametotoxic, embryotoxic, and teratogenic effects that impact on the pre- and postnatal survival of the fetus and newborn, respectively. In addition, the viability and development of the fetus may also be markedly affected by the transplacental passage of lead, and the newborn may be affected by lead in the mother's milk. In summary, OSHA believes that the record in this rulemaking demonstrates adverse reproductive effects in males and females, and adverse development effects in the fetus and newborn at blood lead levels at least as low as 30 to 40 μg/100 g, and perhaps below 30 μg/100 g. OSHA concludes, therefore, that it would be prudent to keep blood lead levels of the fetus below 30 μg/100 g.

The remainder of this section will review the record evidence of the effects of lead on reproduction and development. OSHA will follow the outline described by Hricko which establishes a "chronology of potential adverse effects of job exposures on reproduction or on the ability to have normal healthy children." (Ex. 27(11), p. C-4.) That is, we shall address the chronology as follows: (1) prior to and at conception (2) during pregnancy, and (3) fetal and neonatal effects.

b. *Reproductive Effects.* (1) *Prior to and at Conception.* (a) *Females.* In women, the first point at which the effect of lead on the reproductive system is expressed is in the ovarian cycle. Cantarow et al. presented an extensive review of the literature up to 1944. (Ex. 24 (Zielhuis, Wibowo), Ref. Cantarow et al.) This review article contained many references to the older literature and in particular summarized the effects of lead on female gonads and the uterus as follows:

> Disturbances of menstruation occur commonly in women with lead poisoning, including irregularity of the menses, amenorrhoea, dysmenorrhoea and menorrhagia. There may be transitory periods of sterility with the occurrence of normal pregnancy after withdrawal from exposure; this important fact has been demonstrated in man and in experimental animals indicating that lead injures the germ cells which are formed during the period of gestation. (Ex. 24 Zielhuis, Wibowo), Ref. Cantarow et al.)

Modern studies on animals and humans have also demonstrated adverse effects on the ovarian cycle. A well designed study by Vermande—Van Eck and Meigs demonstrates the gametotoxic effect of lead in rhesus monkeys. (Ex. 95, Ref. 564) Eleven monkeys were injected with lead until clinical signs of lead intoxication had been present for several months. Laparotomy was performed and the right ovary removed. Lead injections were discontinued and the animals were allowed to recover. Three animals were chelated, and 8 months later the left ovary and uterus were removed. Menstruation stopped in all monkeys during lead administration, and the sex skin lost its color by the end of the 6th month. The monkeys gradually recovered and menstrual periods resumed 5 months after the injections

ceased. The sexual skin color redeveloped in 1 to 4 months. The ovaries appeared macroscopically normal following recovery.

The most important change in ovarian function was a depression of estrogen effect. There was almost no indication of gonadal function after 8 months of lead exposure. Microscopically, while the ovaries showed damage to the primary oocytes, there was inhibition of follicle development. Only a few follicles were found growing in the ovaries and these degenerated in the early secondary stages before maturity was reached. Therefore, ovulations failed to occur. It is important to note that the animals recovered after lead exposure ceased. The same effect has been shown in humans. This is logical since the reproductive cycles of both species are very similar, i.e.—a 28-day menstrual cycle, similar processes of oogenesis, ovulation, and menstruation. (Tr. 631-32)

Hilderbrand et al. studied lead effects on the reproduction of male and female rats. (Ex. 27(13), Ref. Hilderbrand et al.) Although all the experimental animals received the same amounts of lead (either 5 or 100 micrograms of lead acetate for 30 days), the females developed higher blood lead levels than males fed the same dose: 30 μg/dl for females and 19 μg/dl for males at the 5 μg dose; 53 μg/dl for females and 30 μg/dl for males at the 100 μg dose. Both sexes, however, showed adverse reproductive effects at both dose levels. At both the 5 and 100 μg doses, female rats developed irregular estrus cycles; follicular ovarian cysts developed when blood leads reached 50 μg/dl. At the lower dose, impotence and prostatic hyperplasia were noted in the males; testicular damage and inhibition of spermatogenesis occurred when blood lead levels reached 50 μg/dl.

Both Maisin et al. (Ex. 24 (Zielhuis, Wibowo), Ref. Maisin) and Jacquet (EPA Criteria Document, Ch. 11, Ref. 380) noted decreases in the numbers of pregnancies for female mice fed varying amounts of lead (0.1 percent to 0.5 percent) for 16-18 days after the day of vaginal plug. Moreover, the numbers of embryos dying after implantation increased.

Panova reported a study on the luteal disorders detected in female workers exposed to lead. The study of 140 female printshop workers, exposed for 1-12 months to less than 7 μg Pb/m³, reported a 13 percent increase in menstrual cycle disorders when compared to 100 textile worker controls. Such disorders were particularly noted in the 20-25 years of age bracket. Unfortunately, the study also reported unexpectedly high ALA-U levels, which makes its quantitative validity difficult to assess. While it is difficult to predict the exact level of lead at which luteal disorders began to manifest themselves, the data indicates that the menstrual disorders occur far below those levels at which lead intoxication is readily detectable. The author concluded that chronic exposure to low air lead levels is related to a disturbed funcional state of the ovaries, and that a dose-response relationship exists. (EPA Criteria Document, Ch. 11, Ref. 354)

(b) *Males.* Reports on the effects of lead on male reproductive function are found in the early literature as well. Cantarow reported the observation of disturbances in male potency as a result of exposure to lead. (Ex. 24 (Zielhuis, Wibowo), Ref. Cantarow) These findings included testicular atrophy and reduced sperm motility. In addition to the Hilderbrand study previously cited, other animal studies have focused on lead effects on paternal reproductive functions. For example, the data from studies of rabbits, guinea pigs, and rats indicates that paternally transmitted effects from lead can occur, including reductions in litter size, in weights of offspring, and in survival rate.

The paternal effects of lead were first confirmed by Cole and Bachuber. (Ex. 23 (Lancranjan et al.), Ref. 25) Litters sired by lead-poisoned rabbits were found to be smaller than those sired by controls. Weller (Ex. 23 (Lancranjan et al.), Ref. 26) also found reductions in the birth weights and survival rates of newborn guinea pigs sired by lead-toxic males.

Varma et al. (Ex. 6(168)) placed each of 14 male mice, which had been fed a solution of lead subacetate for 4 weeks (a total mean intake of 1.65 g), with 3 virgin nonleaded female mice for 1 week. The rate of pregnancy was 52.7 percent in controls compared to 27.6 percent in the lead-treated group, indicating a decrease in male fertility. The mutagenicity index (number of early fetal deaths/total implants) was 10.4 for the lead/exposed mice compared to 2.9 for the untreated controls.

In the study by Maisin et al. (Ex. 24 (Zielhuis, Wilbowo), Ref. Maisin), the percentage of abnormal spermatozoa increased with greater exposure. Ultrastructural changes were present.

Stofen (Ex. 233, Ref. 24) reviewed several studies conducted in Russian laboratories. Injecting 2 μg/kg doses of lead six times within a 10-day period, Egorova et al. found damaged testes and spermatozoa. Morphological changes in the testes of rats receiving 2 mg lead/kg were reported by Golubova et al. (EPA Criteria document, Ch. 11, Ref. 369); no such changes were found in rats receiving 0.2 mg/kg.

In 1976, Mt. Sinai conducted a survey in order to evaluate the reproductive history of workers, and questionnaires were administered by physicians to employees of two lead smelters in Indianapolis, Ind. Of the 153 workers questioned, 131 were married, and 102 of them had 304 children. It was found that 81 percent of those children had been born prior to the workers' initial employment in the lead smelter. Many workers had sought medical assistance for general tiredness and decreased libido, while 10 complained of difficulties in having children. A 22 percent increase in abnormal pregnancies was discovered after 62 of the workers' wives were administered the questionnaire, apparently due to husbands forgetting to include their wives' miscarriages.

Using these data, the perinatal mortality rate was found to be 13.3/100 conceptions (33/247) before beginning lead work, and increased to 19.1/100 conceptions (11/57), an increase of about 50 percent, after initial occupational lead exposure (Ex. 233).

The most important study indicating paternal reproductive effects presented during the hearings was a study by Dr. Ioana Lancranjan who demonstrated altered spermatogenesis with teratospermia (malformed sperm), asthenospermia (decreased motility) and hypospermia (decreased number of sperm) in male battery workers. The lowest blood lead level (mean) at which adverse effects were seen was 41 ±12 μg/100 ml. The result of altered spermatogenesis would be expected to lead to substantial decreases in these workers' fertility, and Dr. Lancranjan hypothesized that there may be teratogenic effects associated with teratospermia (Tr. 577).

Dr. Lancranjan, a neuroendocrinologist, reported initially in the Archives of Environmental Health (1975) (Ex. 23 (Lancranjan et al.)) and later at the OSHA hearings on Occupational Exposure to Lead, on her study in which she had examined 150 lead-exposed workers in a storage battery plant to determine the possible effect of lead exposure on male procreative abilities.

The workers were grouped on the basis of their complaints and on the basis of clinical and toxicological test results. In the first group the workers were further divided, based on the 1968 Amsterdam Meeting criteria, into subgroups: lead-poisoned workers (74.50 ±25 μg/100 ml mean blood lead level), lead workers with moderately increased absorption (52.80 ±21 μg/100 ml mean blood lead level), and lead workers with slightly increased absorption (41 ±12 μg/100 ml mean blood lead level). The second group (6 years mean occupational exposure) consisted of technicians and office workers with physiologic absorption of lead working in a polluted environment (23 ±14 μg/100 ml mean blood

lead level). A control group of 25 men without occupational exposure was also included.

Lancranjan found a significant increase in teratospermia, hypospermia and asthenospermia. Teratospermia was significantly increased among lead-poisoned workmen (blood lead mean 74.5 μg/100 ml and workmen with moderately increased absorption (blood lead mean 52.8 μg/100 ml (Table 1) Hypospermia and asthenospermia were increased not only in both preceding groups, but also those with only slightly increased absorption (blood lead mean 41 μg/100 ml.

Using a fertility criteria based on motility greater than 40 percent, sperm number greater than 20 million, and normal forms greater than 70 percent, the authors concluded that 50 percent of the lead poisoned subjects (blood lead concentration 74 ±26 μg/100 ml were infertile and 76 percent were hypofertile. (Ex. 23 (Lancranjan et al.), p. 399):

Ms. MILLER. I would like to tie together two ideas which I see from your presentation. One, I believe at, 75 percent were hypofertile? Had some decreased fertility?

Dr. LANCRANJAN. Yes.

Ms. MILLER. And 50 percent were infertile?

Dr. LANCRANJAN. Yes.

Ms. MILLER. What do you mean by infertile?

Dr. LANCRANJAN. That means that their chance to have, at that time, a child, was very reduced. That means around zero.

Ms. MILLER. All right. These are the kinds of people who might have to seek help to ever be able to conceive?

TABLE 1.

	N	PbB ug/100ml	N (Semen Analysis)	Alterations in Spermato-genesis	Asthenospermia	Hypospermia	Teratospermia
(a) Lead-poisoned workmen	23	74.5±26	16	15(93%)	8(50%)*	8(50%)*	14(86%)*
(b) Moderate increase lead absorption	42	52.8±21	29	22(68%)	15(51%)*	13(44%)*	17(58%)*
(c) Slight increase lead absorption	35	41±12	19	12(63%)	8(42%)**	8(42%)**	6(31%)
Physiological lead absorption in a polluted environment	50	23±14	25	7(28%)	6(24%)	7(28%)	4(16%)
Controls	50		50		6(12%)	5(10%)	7(14%)

* p<.001 from 12) Lancranjan et al.
** p<.01

Dr. Lancranjan. Yes. (Tr. 585)

The data suggests a dose-response relationship for altered spermatogenesis and teratospermia. The abnormal spermatozoa included binucleated, bicephalic, amorphous, and tapered forms. Reversibility of the lead-induced infertility was observed 3 months following removal of the male workers from exposure.

Lancranjan discovered no significant lead influence on the Leydig cell secretion of testosterone in the workers. The long-term exposure to increased lead levels was found to have produced a direct toxic effect on the germinal epithelium of the seminiferous tubules of the testes, and not an indirect effect through interference with the hypothalamopituitary system. Early in the report, Lancranjan noted that, in the endocrine system, the male testes are the most sensitive glands to a noxious environment. She further emphasized that past industrial practices disregarded this fact, by providing protection from lead exposure only to the female of childbearing age and not the male.

The study by Lancranjan, although unique in its purpose—to study adverse neuroendocrinological alterations produced by lead on male workers—engendered much criticism during the lead hearings. Most of the criticism was based on a review of Lancranjan's study by Dr. R. L. Zielhuis, professor of medicine at the University of Amsterdam:

In 1975 Lancranjan et al. published a study on the reproductive ability of lead exposed male workers. They compared three groups of workers (I average PbB 745 ppb, II 528 ppb, III 410 ppb) with a control group (PbB 230 ppb). The data suggested a dose response relationship for pathological erection, whereas decreased libido, pathological ejaculation and decreased orgasm was more prevalent in the lead workers; the data also suggested a dose response relationship for disturbed spermatogenesis and for teratospermia. However, the study as reported leaves many questions to be asked and comments to be made, e.g. no good matching of controls, overlapping of groups in regard to PbB and ALAU levels, PbB-levels may have been underestimated or ALAU overestimated, individual data not presented. This report may be regarded as indicative, but not as conclusive. (Ex. 24 (Zielhuis, Wibowo), p. 10)

The Lead Industries Association has also criticized the methodolgy of this work by Lancranjan. Dr. Lancranjan has responded to the critiques as follows:

Concerning the annex No. 11 of professor Zielhuis's review I have the following comments to make:

(a) We reported the mean values and their standard deviation (not a standard error!) of all toxicological data.

(b) I agree that as far as PbB is concerned, it is an overlapping between moderate absorption and slight absorption, but division of lead exposed subjects into IV categories was performed according to all toxicologic parameters.

(c) Subjects used and controls for measuring the fertile capacity had similar range of age and mean age.

(d) It was quite difficult to provide individual data of all 150 investigated lead-exposed subjects for a normal publication. Moreover, details concerning the exposure, as well as individual data, required by the Coronel Laboratory (after my departure from Romania) are considered as state secrets and will not be available. (Ex. 58, p. 6).

I write to confirm once again:

1. In my opinion the grouped data I have presented on blood lead levels allows us to fairly conclude that a dose-response relationship exists when we consider teratospermia. The other parameters measured may not show as strong a relationship but are certainly significantly altered over the controls.

2. The relatively high SD (standard deviation) in Pb blood levels represent expected biological variations that would present a scatter on a dose/response curve as calculated to the best fit. This is almost always the case in establishing a dose-response relationship, particularly in epidemiological studies.

3. The findings of lead in blood as against other biological parameters (ALA—urine, Pb urine, coproporhyrin/urine) in the subjects exposed in my study are factual, confirmed by reliable techniques. The absence of matching of the relative interrelations between different biological values is not unusual. The Amsterdam indicators of 68 on much interrelations were revised in their further conference of September 1976. (Ex. 318)

A second criticism by the LIA concerned Lancranjan's choice of the control groups:

Lancranjan's control subjects were mainly office workers and students—that is, people with sedentary occupations—whereas her lead-exposed group consisted largely of persons engaged in heavy manual labor. This difference may have influenced the results of her study. (Ex. 335, p. 33)

OSHA agrees that if this were a study dealing with physical exercise, and not lead exposure, the "defect" might be pertinent, particularly in reference to sexual dynamics. Fatigue may act, under certain circumstances, as a deterrent to sexual stimulus. However, what is pertinent, is the fact that Lancranjan's lead-exposed group was comprised of "50 technicians and office workers of this (storage battery) plant who worked in annex workrooms in a lead-polluted environment * * *." (Ex 23 (Lancranjan et al.), p. 396). Obviously, these individuals did not suffer from physical duress, but they did exhibit changes in spermatogenesis.

LIA, also argued that,

Although the most reliable method of determining the purported effects of lead on the fertility of workers would be to investigate the number and health of the children they had had, Lancranjan was not able to obtain that information. (Ex. 335, p. 33)

Lancranjan stated:

Dr. LANCRANJAN. I am sorry, again. This implies some political aspects, because I have to recognize that the standard of life is very low in Romania and many workers are happy not to conceive. It was not possible to publish such a declaration and to send such a letter from my country, but hoping that you are not relying on, you know, it is quite a danger for my family being now in Romania to declare such things, but you must imagine that in my country both partners are working and the law obliges each family to have at least four children and they haven't the possibility, the material possibility, to take care of so many children and they are happy not to have children. I am sorry to say all this (Tr. 606-07).

Further, the LIA stated:

Lancranjan was unable to determine whether her volunteers did in fact abstain from any sexual activity during the three-day period preceding the testing. Had some of the test subjects not abstained, this would have materially affected the data with respect to the number and motility of the sperm studied. (Ex. 355, p. 32).

Ms. Miller of the USWA questioned Dr. Lancranjan on this issue during the hearings:

Let us assume for a moment that they did not abstain. How might that have influenced your results?

Dr. LANCRANJAN. It was possible to obtain the decreased number of spermatozoa in their production, but not an influence on their morphology. That means teratospermia is teratospermia.

Ms. MILLER. So fertility might be affected in terms of having a decreased number of sperm?

Dr. LANCRANJAN. Of course. And when they came with their products, we again inquired if they followed the recommendation and I think that their standard of understanding was enough high to cooperate with us. It was not their interest because they didn't obtain anything from—They were not interested to give us.

Ms. MILLER. In addition, your results on teratospermia, here you saw the best correlation between the blood leads, you perhaps, would not have been influenced at all, is that correct?

Dr. LANCRANJAN. That is correct. (Tr. 588-89.)

After careful consideration of all the criticism offered concerning the Lancranjan study, OSHA agrees with the conclusions that she set forth:

Results showed a significant increase of spermatic alterations, asthenospermia, decreased motility, hypospermia, decreased number, and teratospermia, malformed sperm. Even in workers with moderate lead absorption, significant differences in asthenospermia and hypospermia were observed. The most frequent and significant alteration revealed by the semen analysis was teratospermia. (Tr. 1161)

To finally evaluate this findings in ths study, OSHA has carefully studied the research design, the experience and qualifications of the principal author, the history and dearth of this type of research, the context in which the work was carried out and the data

themselves. Dr. Lancranjan has been in the field of endocrinology for 10 years, during which time she has primarily focused her attention on studying the reproductive effects of various occupational exposures. During this time, she has studied the effect of carbon disulfide and organic solvents, particularly benzene, as well as lead. She is unquestionably qualified in this field. Second, OSHA believes this to be pioneering research of great significance. Investigations on human subjects have only recently attempted to determine the effect of lead on the male reproductive system. Studies in this area were faced with two major obstacles:

(1) Obtaining accurate information from a subject's memory concerning a very private subject—sexual performance; and

(2) Designing a study which is clearly interpretable or justifiably extrapolatable to the total organismal event.

This research on male reproductive effects, given its innovative character and profound implications, was bound to raise serious questions. OSHA believes reasonable issues have been raised and addressed by Dr. Lancranjan. It would be impossible without further research to eliminate all questions associated with the study. This would be true for any new work such as that described by Dr. Lancranjan. However, based on the evidence in this rulemaking record, OSHA recognizes that there are other studies, both human and animal, which have already been described that demonstrate lead's adverse effect on male fertility. That is, Lancranjan's study is not an isolated case. Therefore the agency has concluded that altered spermatogenesis, teratospermia, hypospermia, and asthenospermia did occur in lead exposed workers at blood lead levels heretofore unseen. It appears that alterations in sperm may have occurred in workers whose blood leads were as low as 30 to 40 μg/100 g. OSHA is in general agreement with Dr. Lancranjan when she states:

Ms. MILLER. You indicate that you feel OSHA's standard is a step in the right direction to protect workers?

Dr. LANCRANJAN. That is right. It is a step.

Ms. MILLER. At the same time you say that the mean the blood levels of 40 micrograms per 100 grams is obviously required to reduce the effects of the type that you saw in these males?

Dr. LANCRANJAN. When I wrote this draft, I was—I was concerned that it was not easy in Amsterdam to impose a lower level and for that reason, I thought that would be a step to be reached, but based on my data, I would have the courage now to recommend even a lower one, because in cases with 41 plus or minus 12, my programs, the lead in blood at 100 milliliters, I already found disturbances of spermatogenesis. To be sure it is possible to be sure in biology, I would recommend a lower level as a safety limit.

Ms. MILLER. What might such a level be?

Dr. LANCRANJAN. You see 23 plus or minus 14 in my table were subjects without significant disturbances of spermatogenesis. So a level between 40 and 20, let us say 30.

Ms. MILLER. 30 micrograms?

Dr. LANCRANJAN. Yes; 30 micrograms per 100 milliliters.

Ms. MILLER. That should be an outside limit, but you feel no one should exceed that level to preclude——

Dr. LANCRANJAN. To avoid an effect on male fertility. (Tr. 586-87.)

(C) *Genetic Effects.* There is evidence in the record that genetic damage from exposure to lead occurs in male and female germ cells. The result of this genetic damage may be (1) the death of the fetus by spontaneous abortion, miscarriage, or stillbirth, or (2) a birth defect or disease in a live born child.

As early as 1914, Oliver studied pregnancy outcome among the wives of males employed as house painters, many of whom suffered from lead colic. Of 467 deliveries, 23 percent (107/467) were stillborn as compared to a stillbirth rate of 8 percent in the entire town. (Ex. 23 (Lancranjan et al.), Ref. 21.)

Lewin also reviewed the reproductive histories of "healthy" women who were married to lead workers. Out of 32 pregnancies, there were 34.4 percent miscarriages and 3.1 percent stillbirths. Of those children live born, 40 percent died within the first year of life, and only 2 survived to adulthood (Ex. 27 (13), p. 6).

It is not clear from the older literature whether the fetal loss which was observed in the wives of workers was due to a mutational event in a sperm cell prior to conception, or due to the teratogenic effect of lead in the developing conceptus following exposure of the pregnant wife to lead-covered work clothes worn home by her husband. Similar results have been found in animal studies where there was no possibility for contamination, which suggests that the genetic damage is caused by lead. The paternal effect of lead on perinatal mortality was first demonstrated by Cole and Bachuber (Ex. 23 (Lancranjan et al.), Ref. 25). Two strains of rabbits were fed lead acetate and then mated with nonexposed females. The authors reported lower birth weights in the pregnancies from lead-exposed males, and higher mortality within the first 4 days after birth. These results were corroborated in guinea pigs.

The effects of lead on reproduction and growth of second generation rats was also investigated. Dalldorf and Williams (Ex. 23 (Lancranjan et al.), Ref. 23) reported that while the growth in the first generation was normal, there was stunted growth in the second generation. In addition there was a significant increase in mortality in the second generation as well as incidents of male and female sterility.

Stowe and Goyer (Ex. 27 (13), Ref. Stowe and Goyer) found a reduced birth rate, survival rate and litter size (i.e., number) in a study of first generation lead toxic male rats.

Nonetheless, these early reports in the literature prompted investigators to further study the mutagenic effect of lead as a possible cause of the increased rate of abortion and stillbirths which was observed (Ex. 233, Ref. 40; (Ex. 27 (13), Ref. DeKnudt et al.; (Ex. 27 (13), Ref. Forni et al.). Dr. Lancranjan has suggested that these genetically impaired cells result in pregnancy failings, but may well be transmitted in the form of gene mutations to the offspring (Tr. 577-578). Although studies have not been specifically undertaken to demonstrate the subcellular effects that lead accumulation might cause in germinal cells (Tr. 668), the studies conducted by Schwanitz, DeKnudt, and Forni on lymphocytes demonstrates that lead does induce human chromosomal changes in somatic cells.

The study by Schwanitz et al. (Ex. 233, Ref. 40) reported a highly significant increase in the rate of lymphocytic chromosome aberrations in eight factory workers exposed to lead oxide who had shown no symptoms of lead poisoning. These workers had a mean blood lead concentration of 74.4 μg/100 ml (range 62-89) and increased ALA-U excretion. The percentage of abnormal metaphases in the cultured cells was 18.75 percent in the exposed group, as compared to 5.13 percent in the 15 healthy controls. The increase was highly significant for chromatid and isochromatid gaps, for isochromatid breaks and for atypical chromosomes. No correlation was found between the blood lead level and the number of chromosomally abnormal cells. Lehnert, in Germany, found an increase in gap-break chromosomal changes in lead workers having a blood lead in the range of 62-89 μg/100 ml (Ex. 233, Ref. 43). He found a positive correlation between increased urinary ALA and the percent of abnormal mitoses seen.

A more extensive cytogenetic study was conducted by Forni and Secchi on workers with an occupational history of lead exposure, who had exhibited various degrees of symptoms (Ex. 6 (53)). Chromosome studies were carried out on 65 male workers occupationally exposed to lead and 65 unexposed controls, matched for age. The workers were divided into three groups: group I, 15 workers with preclinical intoxication; group II, 37 workers with clinical signs or symptoms of lead poisoning; group III, 13 workers with past lead poisoning, who had not

been exposed to lead for 18 months. Evaluation showed statistically significant increased rates of chromatid and unstable chromosomal changes both in group I and group II, when compared to the controls. The differences were not significant for group III.

A prospective study on 11 subjects before and during initial exposure to moderate concentrations of lead fumes in a storage battery plant was carried out on the same group (Ex. 23 (Forni et al.)). The rate of abnormal metaphases was approximately doubled after 1 month of work, remained in this range up to 7 months, and then tended to decrease somewhat. The ALA-D activity of the red blood cells was reduced to almost 50 percent of the initial value after 1 month, and decreased further in subsequent months. Pb-U and CPP increased sharply after 1 month, while ALA-U increased moderately. The authors concluded that the biochemical and cytogenetic data suggested that an adjustment mechanism may intervene after some months of lead exposure.

While many studies demonstrate that lead can cause chromosomal abnormalities, a study by Sperling (Ex. 72, appendix 3 (8)) found no increased chromatid or chromosamal aberrations in the lymphocytes of 5 workers exposed to lead oxide fumes compared to 10 controls. The blood lead level of the exposed group ranged from 50 to 100 μg/100 ml. O'Riordan and Evans (Ex. 27(13), Ref. O'Riordan and Evans) studied 62 shipbreaking yard workers, 35 of which were engaged as burners, directly exposed to lead oxide fumes. The authors concluded that there was only a small increase in the frequencies of chromatid breaks and in the number of cells with abnormal chromosomes in the lead fume-exposed group compared to the controls. However, blood lead concentrations in the range of 80-120 μg/100 ml were found in some of the controls. In other words, the fact that the controls were also exposed to lead is very likely the cause of the negative results. If one compares the rate of abnormal cells to blood lead level, a small steady increase in abnormalities is seen with rising blood lead level.

OSHA has reviewed these cytogenetic studies, and has determined that the preponderance of scientific evidence indicates that workers exposed to lead show an increased incidence of chromosomal abnormalities.

While these chromosomal abnormalities, particularly chromatid changes, may not have a clearly defined biological significance, the Agency has decided that such results must be seriously considered. Forni emphasized this point:

> Increased rates of chromosomal abnormalities are present in cultured lymphocytes not only of workers with clinical lead poisoning but also of subjects with preclinical lead intoxication with no clinical symptoms of signs of disease. Therefore, we can suppose that such alterations might be present in large populations of workers exposed to lead Ex. 6(53), p. 479).

However, since plumbism is not evident these chromosomal changes may go undetected. She stressed:

> On the other hand, it seems that the increased rate of chromosomal abnormalities in comparison to controls of the same age tend to reduce in a number of months or years when the worker leaves the dangerous occupation as suggested by the data obtained in other groups of subjects with past poisoning (Ex. 6(53), p. 479).

Drs. Lancranjan, Hunt, Hricko, and others have suggested that these observed cytogenetic effects result in pregnancy failings and may also be transmitted to the fetus as genetic defects. This is important as research has shown that upon cytogenetic examination, 50 percent of human spontaneous abortions have chromosomal abnormalities, as do 10 percent of all stillbirths.

(2) *During Pregnancy.* During the hearings, an abundance of information was supplied on the many cases of spontaneous abortions, miscarriages, stillbirths, early infant deaths, and premature births resulting from occupational exposure to lead (Ex. 24 (Zielhuis and Wibowo); Ex. 233; Ex. 95, Ref. 482; Ex. 27(13); Tr. 644-647; Ex. 60a.ii).

There are many reports in the early literature of fetal loss in women exposed to lead which were discussed by Rom (Ex. 233). Tardieu reported in 1905 that 608 out of 1,000 pregnancies in lead workers ended in abortion. Legge, who summarized the reports of 11 English factory inspectors, reported that of 212 pregnancies in 77 females working with lead, only 61 living children were produced; there were 21 stillbirths, 90 miscarriages, and of 101 children born, 40 died in their first year. Fifteen women had never become pregnant. Sir Thomas Oliver noted that females premaritally exposed to lead had twice as many miscarriages and stillbirths as female mill workers of similar ages, and that females exposed after marriage had a threefold increase. Nogaki studied the pregnancy outcome of 104 Japanese women, and discovered that there was a preexposure miscarriage rate of 45/1,000, which rose to a rate of 84/1,000 pregnancies following occupational lead exposure. The miscarriage rate for 75 comparable employees who were not exposed to lead was 59/1,000 pregnancies. The maternal blood leads were high, ranging from .110 to .317 mg percent. Pindborg found that of the 25 pregnancies he studied in women who had ingested lead oxide as an abortifacient, and had mild to moderately severe lead poisoning, 60 percent aborted in the first trimester. Rennert, Chyzzer, and Oliver all reported convulsions and macrocephaly in the offspring of women employed by a cottage industry using lead glazes, demonstrating the profound effect of lead on the development of the fetal nervous system. Lane (Ex. 95, Ref. 328) studied female lead workers exposed to air leads of 75 $\mu g/m^3$ to determine the effect of such levels on pregnancy. Out of 15 pregnancies, there were 7 times the expected number of stillbirths (3/15). Since the sample size was small, statistical significance cannot be demonstrated. However, it is important to note that all the women had been removed from lead work as soon as pregnancy was disclosed. Teitlebaum suggests that a sufficient period of time was not allowed to reduce lead body burdens, therefore causing fetal wastage.

> Fahim et al., compared lead values and the course of 249 pregnancies in Columbia, Mo., with 253 occurring in the center of America's lead belt at Rolla, Mo. At Columbia, greater than 96 percent delivered normally at term, 3 percent were preterm (defined as a neonate born before 37 weeks of gestation and weighing less than 2,500 grams), and less than 1 percent had premature rupture of the membrane. At Rolla, only 70 percent were term, 17 percent had premature membrane rupture (defined as spontaneous rupture of the membrane before the onset of labor and when labor does not begin within 12 hours), and 13 percent were preterm. The striking blood-lead findings from the Rolla lead belt area were a doubling of maternal blood lead in the premature membrane rupture and preterm groups, and a fourfold increase of fetal blood lead in these groups. Fahim has also noted an increase in molar pregnancies (which result from a blighted ovum) in the Rolla region. He also states that women in the lead belt have increased menstrual disturbances—amenorrhea, dysmenorrhea, irregularity of menstrual cycles, and menorrhagia (Ex. 233, p. 3).

Zielhuis and Wibowo (Ex. 24 (Zielhuis and Wibowo)) criticized this study primarily because 70 percent of the women placed in the "mean mining region" group had blood lead levels comparable to 96 percent of those placed in the "no lead mining region" group. While OSHA acknowledges that the two groups were not as distinct as their group titles of "near mining" and "no lead mining" implied, the fact remains that the women with higher blood lead levels demonstrated the effects described.

This early literature clearly notes cases of sterility and abortions resulting from female lead exposure. The mechanism by which lead interacts with the female reproductive system to cause these conditions, however, is imperfectly understood. The early literature suggests that such cases prob-

ably are caused by germ cell alterations.

Dr. Vilma Hunt has examined the literature documenting reductions in fertility and increased rates of abortions and stillbirths and has probed to determine what biological effects, if any, lead exerts on the cellular and subcellular level. Hunt presented testimony which indicated that impaired germ cells, paternal or maternal in origin, would cause pregnancy failings, as well as be transmitted in the form of gene mutations to the offspring (Tr. 577-578).

While the precise mechanism(s) by which lead effects spontaneous abortion, miscarriage, and stillbirth in women is unclear, there is no debate that such effects occur. Further research is required to determine whether genetic, teratogenic, fetotoxic or, embryotoxic mechanisms are active. Any, or all may be responsible for adverse effects in the fetus. OSHA believes that, whatever the mechanism, a standard must be promulgated which prevents these effects of lead from occuring.

There is conclusive evidence that lead crosses the placenta of pregnant women and enters the fetal tissues; lead levels in the mother's blood are comparable to concentrations of lead in the umbilical cord blood at birth. (Ex. 24 (Zielhuis, Wibowo); *Ibid.*, Ref. Fahim; *Ibid.*, Ref. Haas; *Ibid.*, Ref. Baglan; Ex. 95, Ref. 32; Ex. 60a.ii; Ex. 233; Ex. 335). Correlation coefficients between lead in the umbilical cord and maternal blood lead levels have been reported as high as 0.84 (Ex. 95(32)). Transplacental passage of lead becomes detectable at 12-14 weeks of gestation.

The lead, in this way, may directly impair fetal survival and development. Fetal tissues have the ability to store lead. For example, a study on lead transfer published by Barltrop in *Mineral Metabolism in Pediatrics* in 1969 (Ex. 95, Ref. 32), demonstrated that maternal mean blood lead values of 15.2 μg/100 ml (8-22 μg/100 ml) correspond to fetal mean blood lead values of 11.2 μg/100 ml (4-24 μg/100 ml). By analyzing tissue from 34 fetal specimens of 10 to 40 weeks gestation, Barltrop further established that "the distribution of lead between the (fetal) tissues was found to be similar in concentrations to that in later life."

Because the fetal specimens of 10 to 12 weeks maturity were so small, chemical analysis of all tissues was somewhat limited. Consequently, Barltrop concentrated on those fetuses reaching term (or longer). From an in-depth evaluation of the tissues from a 40-week-old fetus, he found that most of the total body lead was concentrated in the fetal skeleton. The next highest lead concentrations were located in the fetal liver, heart, kidneys, and brain, in decreasing order. The relatively high concentrations of lead contained in both blood and brain are a reflection of their mass in comparison to that of the entire fetus. An organ such as the brain, which has "a relatively low affinity for lead but large relative mass," may become a significant contributor to the total body burden. It is therefore established that lead does cross the placental barrier, and is capable of being stored in fetal tissue.

When discussing the effects of lead on the fetus, one of the major issues to be addressed is whether the "fetus is most susceptible to lead toxicity during the stage of most active growth, suggesting that the early pregnancy is most endangered and that the fetus is possibly more sensitive than the young child" (Ex. 59, p. 6).

Dr. Vilma Hunt reviewed the data which served as a basis for this view that the initial trimester represented the most lead sensitive fetal period. She stated her conclusions as follows:

"The evidence for first trimester loss is primarily from studies of acute severe lead poisoning in women using lead salts as an abortifacient or severe industrial poisoning cases. The cases came to attention because of the death of the mother, because of her severe poisoning. Abortions certainly occur under such conditions as the anecdotal reports attest. Teratogenic effects, per se, have not been observed in surviving fetuses and live borns who have experienced lead intoxication in utero throughout gestation including the first trimester. I would say that the weight of evidence points to toxic effects on maternal physiology as the prime cause for embryo loss in the first trimester, under conditions of high blood lead levels in the pregnant women, over 100 μg/100 ml. Bell proposed that excessive lead first injures the chorionic epithelium of the uterus and thus, indirectly injures the fetus leading to its expulsion. Hardy and Hamilton stated that expulsion of the fetus follows with, or without, a direct stimulating effect of lead on the uterine musculature. It is appropriate to note the considerable capacity for contraction and relaxation of the uterine musculature which is maintained throughout a woman's reproductive life (Masters and Johnson).

"A review of placental physiology shows that the nutrition of the early fetus is primarily due to trophoblastic digestion and absorption of nutrients from the endometrial decidua, diminishing in importance by the 12th week of gestation, by which time nutrients are then obtained from diffusion through the placental membrane.

"In the early months of development placental permeability is relatively slight for two reasons: First, the total surface area of the placental membrane is still small at that time, and second, the thickness of the membrane is great. However, as the placenta becomes older, the permeability increases progressively until the last month or so of pregnancy, when it begins to decrease again.

The placenta also has considerable storage capacity, and during the first few months of pregnancy, it grows tremendously in size while the fetus remains relatively small. Calcium along with other substrates is stored in the placenta to be used in the later months of pregnancy for growth by the fetus. It could be expected that lead would be similarly stored.

It is my opinion then, that the first trimester has not been shown to be the period of highest vulnerability for the fetus because:

1. The nutrition of the fetus for the first 10 to 12 weeks is primarily dependent on nutrients from the endometrial decidua and not directly from the blood nutrients passing through the placenta.
2. Specific human teratogenic effects have not been reported as a result of lead exposure. Deformities such as macrocephaly are likely to result from lead exposure throughout the gestation period of cell differentiation.
3. Probable susceptibility of uterine musculature to lead (above 100 μg/100 ml blood) could result in expulsive contractions (Ex. 59, p. 6-7).

On the other hand, Dr. Hunt would not exclude the potential for lead-induced effects on the initial trimester of pregnancy. She maintains that the presence of lead in fetal tissue does not necessarily indicate that the observed effects occurred during the second and third trimester; they may, in fact, be the result of earlier accumulations of lead in the first trimester of pregnancy (Tr. 634). She supported the testimony presented by Dr. Hricko (Ex. 60A) and agreed that lead does not pass from mother to fetus via the placenta until the 10th-12th week of gestation. This does not mean, however, that the fetus is not directly affected by lead which is absorbed during the first trimester of pregnancy. As the placenta is maturing, and the placental barrier is thinning, it is storing calcium necessary for later fetal skeletal production (Tr. 634). Concomitant with the first evidence of fetal skeletal calcification, lead is observed present in the fetus. Like calcium, lead may be stored in the placenta during the early stages of pregnancy to be released when the placenta becomes functional (Tr. 634).

Furthermore, Barltrop's study (Ex. 95, Ref. 32) verifies that the first trimester is not the greatest period of fetal susceptibility to lead, since lead did not become detectable in fetal tissues until sometime between 12-14 weeks. Thereafter, lead concentrations increase in these tissues until term.

Evidence presented at the hearing demonstrates that stillbirths, along with children who die shortly after birth, have significantly higher lead levels than normal neonates (EPA Criteria Document, Ch. 11, Ref. 359). This supports the position reiterated by Hunt and Hricko, that the accumulation of lead found in the fetus may have occurred in the earlier stages of

pregnancy and not necessarily only in the second and third trimester.

While Dr. Hunt maintains that specific teratogenic effects cannot be demonstrated to occur due to lead exposure during the period of cell differentiation in the first trimester, deformities are probably the result of exposure throughout the gestation period. OSHA agrees with the conclusions of Dr. Hunt and believes that the growing fetus is vulnerable whatever its stage of development.

d. *Fetal and neonatal effects.* Lead exposure has clearly been shown to have a serious effect on fertility and fetal survival. In addition, there have been extensive investigations on the effects of lead exposure on surviving offspring of lead workers.

Since studies concerned with the effect of lead on the fetus are scanty, witnesses discussed studies of children exposed to lead, and extrapolated their results to the developing fetus, as well as the newborn.

(1) *Heme synthesis inhibition.* Children are similar to adults, insofar as the earliest demonstrated effect of lead involves its ability to inhibit heme production. The implications of this inhibition are potentially profound, since the ultimate result is the reduction of the body's ability to efficiently produce the energy required for normal activity and maintenance. Lead, by interfering with heme synthesis, impairs the normal respiratory process. Transport of oxygen to the lungs requires muscle contraction. Heme proteins, myoglobin and the cytochromes are utilized in this step. The transport of oxygen from the lungs to all body tissues is facilitated in the hemoglobin of the red blood cells whose production is also limited by lead exposure. Far more significant is lead's potentially damaging effect on the individual cellular respiratory apparatus. Each cell in the body uses the cytochromes, as well as oxygen, in an electron transport process required for energy utilization.

In the past, indicators of lead-stimulated impairment have been based on the hematopoietic system. The indicators include alterations in: (1) the activity of the erythropoietic enzyme ALAD, and (2) the level of ZPP in the red blood cells, ALA in serum, and ALA and CPP in urine.

Inhibition of ALAD activity represents the earliest evidence of an adverse effect of environmental lead exposure. This occurs in both children (Ex. 95, Ref. 391) and adults (Ex. 6 (70)) at blood lead levels as low as 10–20 μg/100 ml. At such low levels of lead exposure the biological significance of this inhibition is unclear, since there is no accumulation of precursors or products. At slightly higher lead levels, between 30–50 μg/dl, the activity of ALAD is reduced to below 50 percent, causing a significant buildup of the precursor ALA in the urine (Ex. 95, Ref. 391; Ex. 96). Similar findings are seen in adults (Ex. 96). Nearly complete inhibition of the enzyme occurs around 50 μg/dl in children (Ex. 95, Ref. 391) and 70 μg/100 ml in adults (Ex. 6(70)).

Increased erythrocyte protoporphyrins also occur at blood lead levels above about 20 μg/100 ml (Ex. 32(15)). The Center for Disease Control (CDC) considers blood lead levels of 30 μg/100 ml in children, accompanied by increased erythrocyte protoporphyrins, to be indicative of lead poisoning (Ex. 32 (15)). Studies demonstrate a correlation between ALAD activity in human mothers and fetuses. Most recently, an inhibition of erythrocyte ALAD activity related to lead in both the pregnant woman and the fetus has been observed. Since ALAD is inhibited in both mother and fetus exposed to lead, fetal lead exposure should be no higher than a level associated with significant impairment to the ALAD system in the mother. Such impairment occurs as blood lead levels rise above 30 μg/100 ml.

Piomelli observed an exponential rise in ZPP with arithmetic blood lead level increases between 5 and 90 μg/100 ml in children (EPA Criteria Document, Ch. 11, Ref. 147). Others have confirmed this correlation (Ex. 6 (79), Ref. 23; EPA Criteria Document, Ch. 11, Ref. 153; EPA Criteria Document, Ch. 11, Ref. 156). Furthermore, the slope of the curve of ZPP versus blood lead was found to be steeper in children than adult males (Ex. 6 (79), Ref. 23), and comparable to that in adult females (EPA Criteria Document, Ch. 11, Ref. 159; Ex. 23 (Roels)). Chisholm (Ex. 99B) suggested that in children, ZPP represented a better indication of overexposure to lead than blood lead levels. This may be true only in children with a relatively constant blood lead level (EPA Criteria Document, Ch. 11, Ref. 150), and without iron deficiency. (EPA Criteria Document, Ch. 11, Ref. 164).

Since a rise in ZPP represents the first indication of lead impairment, the threshold blood lead level at which this occurs is crucial. A study by Roels on 143 schoolchildren with blood leads ranging from 5–40 μg/100 ml indicated a threshold effect at blood lead levels between 15–20 μg/100 ml blood lead. (EPA Criteria Document, Ch. 11, Ref. 159) Using probit analysis and segmental curve fitting on 1816 data points from children 2–12 years of age, Piomelli assessed the threshold for no rise in ZPP at 15.5 μg/100 ml blood lead. (EPA Criteria Document, Ch. 11, Ref. 169).

Even if ferrochelatase, has a reserve capacity similar to ALAD, the early accumulation of precursor implies that such safeguards are overpowered by lead at blood lead levels above 15–20 μg/100 ml. Furthermore, since the effect of lead on iron insertion is not limited to the blood-producing system, but extending to all cells, impairment relevant to human health occurs at blood lead levels above 15–20 μg/100 ml blood lead.

Anemia may result from impaired heme synthesis, as well as hemolysis. The blood lead threshold for childhood anemia is 40 μg/100 ml (Ex. 32 (67)), whereas such effects are noted in adults above 50 μg/100 ml (Ex. 5 (18)). Bradley et al. (EPA Criteria Document, Ch. 11, Ref. 209) reported that 39 percent of children with blood lead levels less than 40 μg/100 ml had hemoglobin values of 10 g or less. In comparison, Betts et al. (EPA Criteria Document, Ch. 11, Ref. 101) reported anemia in 36 percent of children with blood lead values between 37–60 μg/dl, and in 14 percent of children with blood lead concentrations less than 37 μg/dl. Signs of anemia include pallor, sallow complexion, and symptoms include fatigue and malaise. In young children unable to express themselves, anemia may be overlooked. (Ex. 5 (5).)

(2) *Neurological effects.* Lead is capable of damaging both the central and peripheral nervous system. Children, which in this context includes the newborn and the fetus, are most clearly the population at extreme risk. In children the central nervous system may be severely damaged resulting in frank encephalopathy, coma, convulsion and death, while anemia, and peripheral neuropathy are readily observable lead-related phenomena.

Less specific symptoms of childhood lead poisoning, antecedents of severe problems, may also go unreported. Irritability, restlessness, apathy, abdominal pain, headaches, vomiting, constipation, hallucinations, developmental delays and regression may be misinterpreted as indications of other childhood illnesses.

Psychological and other functional impairment in asymptomatic children may also be misinterpreted or overlooked. Therefore, recognition of these problems often occurs in retrospect after clear cases of encephalopathy or anemia have set in. Once acute encephalopathy has occurred, there is a high probability of permanent irreversible damage to the nervous system.

Early damage to the nervous system in children exposed to lead has been documented in numerous studies. These studies indicate that damage may have occurred in children only moderately exposed and in whom no demonstrated morbidity had been

shown to exist. These adverse effects associated with lead exposure include behavioral problems, difficulty in task performance, deficiency in IQ, and motor nerve conduction defects. Behavioral problems such as hyperactivity have been seen in children whose blood lead levels were 25 to 55 μg/100 ml. In general, neurologic effects, including both peripheral neuropathies and signs of CNS damage, are first encountered in children as blood lead levels reach 50 μg/100 ml and very rapidly intensify in severity as a function of increasing blood lead elevations. Neurologic damage at low blood lead levels in children formed the basis for CDC's recommendation that blood lead levels in children be maintained below 30 μg/100 ml (Ex. 32(15)). The Committee on Toxicology of the National Academy of Sciences concurred with this recommendation and also recommended that given the variation among individuals, the mean blood lead concentration for groups should not exceed 20 μg/100 ml (Ex. 86M, p. 9) OSHA believes the fetus is certainly no less susceptible than child to neurological damage from exposure to lead, and therefore should be similarly protected. In fact, there is evidence which suggests that long term neurobehavioral deficits may also be induced by exposures of human fetuses to lead in utero, as indicated by the apparent higher incidence of postnatal mental retardation among children born to mothers experiencing lead exposure before and during pregnancy.

A study by Beattie in 1975 (Ex. 6(6)) suggests that childhood mental retardation may be caused by maternal ingestion during pregnancy of tap water containing lead. An analysis of the lead content of tap water in homes occupied during the three trimesters of pregnancy and during the first year of life of 77 mentally retarded children (IQ less than 70) aged 2–7, and of 77 nonretarded matched controls, demonstrated the water lead content to be significantly higher in the retarded group. The probability of retardation was significantly higher when the water exceeded 80 μg/100 ml (The World Health Organization's acceptable lead level in tap water is 10 μg/100 ml). The blood leads of the retarded group were significantly greater (25±12 μg/100 ml) than matched pediatric patients (17.8±5 μ'g/100 ml). Of 64 matched pairs, no normal children came from homes with water lead levels greater than 30 μg/100 ml, while 11 mentally retarded children came from homes with such levels. The authors conclude that women exposed to a highly leaded water supply had an increased likelihood of producing a defective child, by a 1.7 factor.

In 1977 Moore (EPA Criteria Document, Ch. 11, Ref. 242) determined the blood lead levels of Beattie's original test subjects at 2 weeks after birth, and found the mean leads to be significantly different in the 41 retarded and 36 normal subjects (25±8.9 vs. 20.9±3, p. 05). Blood leads over 30 μg/100 ml at birth were observed in one third of the mentally retarded children, compared to 12.5 percent of the controls. Furthermore, for eleven mentally retarded children associated with high water lead, there was a highly significant relationship between neonatal blood lead and domestic lead concentration from the maternal home during pregnancy. These two studies suggest that lead exposure to the fetus, through maternal ingestion of leaded tap water, may cause disturbances in brain organization that result in mental retardation. Insufficient information exists to estimate the ingested lead level that might cause these future defects.

The effects of lead on the neurological system of children has been extensively reviewed (EPA Criteria Document, ch. 11, Ref. 92; Ex. 24 (Zielhuis, Wibowo); Ex. 86M), and OSHA believes there is littel value repeating those reviews in this final standard. In OSHA's view, there is conclusive evidence that lead passes through the placental membrane, and that there is an increased elimination of lead through breast milk. Zielhuis, in his review of reproductive effects, stated:

> Increased elemination of lead through breast milk in combination with previous intrauterine exposure is a reason for concern in regard to the health of the infant. (Ex. 24 (Zielhuis, Wibowo), p. 14.)

OSHA further finds there is conclusive evidence that exposure of the fetus and infant to lead induces neurological damage manifested by behavioral disorders, motor nerve conduction velocity decrements, deficiency in IQ, subjective CNS symptoms difficulty in task performance and mental retardation. These effects occur at blood lead levels below 30 μg/100 ml, but generally are manifest at 50 μg/100 ml. The following brief review discusses a few of the studies which demonstrate nervous system damage in children and presumably the fetus and newborn.

De la Burde (EPA Criteria Document, Ch. 11, Ref. 223) studied the latency of lead-related neurobehavioral symptoms in asymptomatic children. Seventy children, age 4, who had a history of plaster and paint eating between 1 and 3 years of age and blood lead levels above 30 μg (mean=59 μg/100 ml) were studied. The authors found significant differences in psychological tests performed at 4 years of age between the lead exposed children and controls. They observed dysfunctions of the CNS, fine motor dysfunction impaired concept formation and altered behavioral profile.

In a followup study on the same 67 children at age 7 and 8, de la Burde (Ex. 6 (25)) showed similar findings. Still observed were differences in behavior, visual motor and fine motor coordination and global IQ. The de la Burde results can be interpreted as demonstrating neurobehavioral deficits at blood lead levels of 40 to 70 μg/100 ml. Similar conclusions were also warranted on the basis of the results of a study by Perino and Ernhart. (EPA Criteria Document, Ch. 11, Ref. 225.) Hyperactivity was found to be associated with exposure to lead. (EPA Criteia Document, Ch. 11, Ref. 236.) Baloh (Ex. 6(4)) and Roberts et al. (Science 168:1120–1123, 1974) also found increased hyperactivity in "asymptomatic" children with chronic increased lead absorption.

Three studies were conducted on children living near a smelter in El Paso, Tex. As described by Carnow (Ex. 27 (7), p. 159), a large number of children were found with elevated blood lead values. Basophilic stippling, anemia, hyperexcitability, and fatigue were noted as well. The children were then hospitalized and chelated. In a followup study of 10 of the children, Carnow et al. (ibid.) reported significant numbers of abnormal EEG's and learning deficit. One year following treatment, Landrigan (Ex. 6 (99)) conducted a series of studies on the same population. His cohort consisted of 45 currently asymptomatic children ages 3–15, and 78 ethnically and socioeconomically matched controls. Mean blood lead concentrations of 48 μg/dl (range 10–68) and 27 μg/dl range less than 40) were recorded for the respective groups. He also found abnormalities in the test group. Demonstrated were nonverbal cognitive and perceptual motor skill decreases, as well as low grade motor neuropathy in children with blood lead levels of 40–60 μg/100 ml. Full scale IQ, verbal IQ, behavioral and hyperactivity ratings failed to display any differences in the two groups.

Landsdown (Ex. 6 (99), Ref. Landsdown) also investigated the relationship between blood lead, general intelligence, reading ability, and behavioral disorders in school age children living near lead polluting facilities. Distances from the facility were related to blood lead levels, but there was no relationship between blood level and any measurable mental functioning. However, less than 19 percent studied had blood leads over 40 μg/dl.

Another followup study on the same group (Ex. 6 (99), Ref. McNeil and Ptasnik) failed to demonstrate neurological and psychological abnormalities.

The evidence describing adverse effects from lead exposure to the fetus and newborn was uncontroverted during the hearings. There was, in fact, virtually no debate on the issue of whether the fetus and newborn are at risk from exposure to lead. LIA addressed the fetus in their posthearing brief as follows:

> The evidence submitted at the hearings, however, established that females themselves are not more susceptible than males. (See e.g., Needleman, 1116-17; Stellman, 1154-55; NOW 2478) (see also NIOSH 1321, 1802). The more serious but quite different question raised by "female employees of childbearing age" is the problem of potential health hazards to the fetus.
>
> The problem of protecting unborn children of female lead workers arises as a consequence of a confluence of several factors:
>
> (1) Lead in the mother's bloodstream crosses the placental membrane and can affect the unborn child.
>
> (2) Although the medical data and studies are not entirely consistent, it is possible—as the notice suggests—that "the statistical likelihood of clinical symptoms and permanent damage" to the fetus may increase once the blood lead level of the mother reaches 30 or 40 μg/100 g. (Exhibit 2, at 45936) (see also Lundquist 4509.) (Ex. 335, p. 38.)

The United Steel workers, in their posthearing brief, quote Hricko:

> * * * lead exposure can potentially affect one's ability to have normal healthy children in a variety of ways * * * prior to conception * * * there may be menstrual disorders, interference of sexual function, lower fertility, possible genetic damage (there) may be problems with sperm prior to conception which could result in miscarriage or stillbirth. At conception, there could be difficulties in conceiving a child, problems with implantation. During pregnancy there could be miscarriage or stillbirth as a result of substances crossing the placenta and reaching the developing fetus.
>
> On the newborn baby, there could be toxic effects as a result of chemicals transmitted to the child in the mother's breast milk.
>
> Or on the growing child there can be toxic effects of lead. When lead is inadvertently brought home on parent's work clothes. (Hricko, Tr. 677-8.) (Ex. 345, p. 38.)

What was not clear prior to the hearing, was precisely the blood lead level in the mother or father which would protect against lead-induced effects. Rom, for example, has suggested that there may be no threshold at which adverse effects would not occur in the course of development of the new fetus. (Ex. 233.)

Scientific theory openly admits the difficulties involved in the extrapolation of these data to precise standards, such as the relative hemoconcentration of the newborn, the relative hemodilution of the pregnant woman and the unproven possibility of mobilization of lead from the skeleton during pregnancy. Nonetheless, the body of evidence resulting from the hearings has convinced OSHA that blood lead levels must be kept below the 30 μg/100 ml range to adequately protect the developing fetus. (Tr. 6470, 71; 1770; 1958; 1151; 4509; Tr. 675; 587; Tr. 647.) OSHA is mindful of the statement of the Center for Disease Control which considers blood lead levels of 30 μg/100 ml to be elevated in children. (Ex. 32 (15).) OSHA will consider the blood lead level of a pregnant woman to be elevated if it exceeds 30 μg/100 ml.

With respect to the effect of lead on the male reproductive system, OSHA agrees with Dr. Hricko that there has been "appallingly little research on this problem (the precise blood lead levels at which reproductive damage occurs) by either U.S. industry or the Government in the last 30-40 years". (Tr. 675.) However, studies have been described which indicate that lead can cause decreased fertility, sperm abnormalities, impotence, and difficulties in erection in males and in animals. Several studies have demonstrated chromosomal changes in workers exposed to lead. Based on these studies, OSHA concludes that males exhibit lead-induced reproductive effects at 30 μg/100 ml and above. OSHA considers the effects on male fertility to be a matter of serious concern, and agrees with the conclusion of Infante and Wagoner that:

> In light of these findings, we must now transfer male employees from high exposure areas, or require proof of their inability to reproduce as has previously been the public health approach for females. (Ex. 27 (13), p. 10.)

Historically in many developed countries, "occupational exposure of adult females to lead is forbidden by law." (Ex. 24 (Zielhuis, Wibowo), p. 1.) This restriction has been based on reports of adverse effects on reproduction. There is, in fact, evidence in this rulemaking record that some firms in the United States have barred women from employment involving lead. (Tr. 6471; 678-79.) The evidence of mutagenic effect in both men and women, and of reduced fertility in males demonstrate that both men and women must be considered at risk from exposure to lead. Given the relative number of male and female employees in lead operations, this conclusion is even more valid.

> While only an estimated 500 babies per year are born to women lead workers, the number born to wives of male lead workers is several thousand (Tr. 631.)
>
> Working men are generally fertile and potential procreators throughout their working years—in 1980, 62.5 million men, versus 22 million women of childbearing age. (Tr. 631.)

OSHA concludes that there is no basis in the record for preferential hiring of men over women in the lead industry, nor will this final standard create a basis for exclusion from work of any person, male or female, who is capable of procreating.

In summary:

1. The evidence in this rulemaking record demonstrates conclusively that lead has severe effects on the reproductive capability of males and females.

2. Lead exerts genetic, gametotoxic, intrauterine, and extrauterine effects.

3. The fetus and newborn are sensitive to lead; the fetus is exposed to lead through transplacental passage from the mother, while the newborn is exposed to lead in the breast milk.

4. Maintenance of maternal blood lead level below 30 μg/100 ml is required to adequately protect the fetus.

5. Blood lead levels should be maintained below 30 μg/100 ml in both male and female workers who wish to plan pregnancies.

6. Altered spermatogenesis, teratospermia, asthenospermia, and hypospermia are evident in workers exposed to lead. Blood lead levels of these workers were apparently as low as 30-40 μg/100 ml.

6. *Mortality experience of lead workers.* The proposed lead standard discussed in some detail the mortality study by Cooper and Gaffey of lead smelter and battery plant workers in which there was a suggestion that prolonged exposure to lead may increase the risk of contracting a number of chronic diseases, such as nephritis and other hypertensive diseases.

> Apart from the effects of lead on enzyme systems and the possible appearance of mild clinical symptoms at blood lead levels in the range of 40-80 μg/100 g, there is concern that continued low level exposure to lead may increase the risk of developing chronic disease as well as contribute to the shortening of life. In a recent mortality study by Cooper and Gaffey of lead smelter and battery plant workers, evidence was produced suggesting that prolonged exposure to lead may increase the risk of contracting a number of chronic diseases, such as nephritis and other hypertensive diseases. Additionally, the standard mortality ratios (SMR's) observed by Cooper and Gaffey for all causes of death in the smelter and battery workers were 107 and 99, respectively. These SMR's were only slightly different from an SMR of 100 which represents that of the general population.
>
> The authors did not consider this small deviation of the workers' SMR's from that of the general population to be of any medical significance. It should be noted, however, that results of mortality studies are frequently subject to differing interpretations among scientists. In this regard, several important factors in evaluating the mortality experience of workers compared to the general population deserve mention. For example, it is generally concluded that when the mortality rate of the study population does not exceed that of the general population, no excess deaths rates were found in the study population. However, since the worker population tends to be healthier than the general population, the expected death

rates for workers should be lower than the expected death rates for the general population. Thus, SMR's for workers should be lower than 100 and perhaps should be in the range of 80 to 90. If this is so, the SMR's found by Cooper and Gaffey in lead workers are more significant. In any case, OSHA desires that the issue of appropriate SMR's for the working population, which involves evaluation of other toxic substances as well as lead, be addressed in comments submitted on this proposal.

Another critical factor in evaluating this mortality study is whether enough individuals were followed for an adequate period of time following onset of their exposure to lead to allow for the development of chronic diseases. Thus, the status (living or dead) of workers followed was ascertained at the end of 1970. Since nearly 1,400 of the 2,352 smelter workers started work in 1950 or later and nearly 1,700 of the 4,680 battery workers began work in 1950 or later, insufficient time may have passed following onset of exposure to permit the development of chronic diseases in a high proportion of those studied. As a result, although the latency period is unknown, these results may be somewhat diluted by the composition of the workers who were studied.

Of all the suggestive positive results from the Cooper and Gaffey study, the findings of a nearly two-to-threefold excess in nephritis among workers exposed to lead appears to be reasonably well supported by data from previous studies. For example, a number of earlier studies have observed an increase in nephritis in workers exposed to lead. In these observations, the possible interactive effects between lead and other renal toxic agents, such as cadmium, cannot be ruled out. (Ex. 2, p. 45937.)

The record contains rather limited discussion of this study and no new data was developed during the hearings. LIA did criticize the statement:

> There is concern that continued low level exposure to lead may increase the risk of developing chronic disease as well as contribute to the shortening of life. (Ex. 2, p. 45936.)
>
> The Association believes that this concern is without foundation and is refuted by the very Cooper and Gaffey study on which OSHA relied in raising the issue. (Ex. 335, p. 42.)

Based on the extensive evidence in this rulemaking record, OSHA believes there is little if any doubt of the accuracy of the statement in the proposal, but those issues are discussed elsewhere in this preamble and in this subsection OSHA will attempt to amplify the discussion on Cooper and Gaffey presented in the proposal.

Most of the data on mortality in the record relates to the mortality study of lead workers carried out by Cooper, Tabershaw, and Gaffey presented as a Followup Report to the International Lead and Zinc Research Organization by Tabershaw Cooper Associates and published subsequently by Cooper and Gaffey. (Ex. 5 (28).) The comments provided in the hearing largely do not illuminate the report by Tabershaw, Cooper, and Gaffey. OSHA will focus its attention on the report itself.

This study suffers from a number of shortcomings, most of which are recognized and addressed by the authors. These are:

(1) Although 10 battery plants are studied, one provides 60 percent of the battery population under study and 93 percent of the battery plant deaths. It is unfortunate that this plant alone was not evaluated in this report as the detailed information available through the company may have provided a good deal more, such as exposure levels, race distribution, et cetera.

(2) The battery plant workers are subdivided into those who were first employed before 1946 and those employed after 1946, and those employed less than and greater than 10 years. Ninety-four percent of the deaths occurred in workers employed first before 1946 and 84 percent of the deaths occurred in workers employed greater than 10 years. As a result, the evaluation of cause of specific mortality by these subdivisions is really not interpretable, since the numbers in the after 1946 employment group and less than 10 years employment group are too small to be usefully interpreted.

(3) The inability to identify race in the cohort except from death certificates is another important problem. Race specific rates cannot be calculated, and much hypothesizing had to take the place of specific calculations. Even hypothesizing is limited since 20–30 percent of the population has not even a current estimate of racial distribution.

(4) It is unclear why the authors did not analyze proportionate mortality ratios by race since this information was available on death certificates.

(5) An attempt was made to subdivide the population into exposure categories of high, medium, and low, based on estimates of industrial hygienists familiar with smelter and battery plant operations. Since 10 plants make up the battery total, and 6 plants make up the smelter total, the possible variations between these plants make some reinterpretation of job classification by exposure level next to impossible. This is true especially since no attempt is made to categorize, even crudely, a range of exposure defining the three levels. Furthermore, there is no indication of the potential variability between the plants, either currently or in the past. All data is presented only as average data for battery or smelter operations. It is more than likely that there is large variation between plants due to different processes, procedures, or control operations. Furthermore, review of the biological values shows that smelter workers' average levels decrease over time, while battery workers' average levels increase over time. Thus, subdividing the population into exposure categories is unlikely to be productive.

With these criticisms in mind, the study still deserves attention. A major point, however, regards how to look at such a study. The shortcomings in the study presented by the author and by OSHA in large part will tend to bias against the finding of work-related excess causing specific mortality. It is of interest, therefore, to look at what excesses are noted, and to make some estimate of their significance.

The information on mortality analysis is reasonably summarized for the malignancies. One is suspicious, in reviewing the results, that there might be some excess of respiratory or digestive cancer related to work in smelters or in battery plants, but the authors are reasonably cautious in drawing any conclusions. Much more data different from the available studies will be necessary before any more can be said.

Deaths from hypertensive disease and renal disease, however, are a different question. The authors, particularly in their final report to ILZRO, discuss, in reasonable detail, the evidence for exposure related excess renal disease and selected hypertensive disease in this population.

Reviewing first the evidence regarding smelter workers, "Vascular Lesions Affecting the CNS" appear to be elevated in total, and distributed according to duration: those employed before 1946, SMR=118 and those employed after 1946, SMR=76; those employed less than 10 years, SME=92; and those employed greater than 10 years, SMR=108. The fact that these SMR's do not also distribute according to estimated dose levels is of little concern given the problems with those estimates. As the authors point out, this excess is consistent with previous reports in the literature, specifically those by Dingwall-Fordyce and Lane in 1963 (Ex. 6 (40)), and Lane in 1964 (Ex. 5 (1)). In examining "Hypertensive Heart Disease," there is somewhat larger excess in smelter workers which is more strikingly distributed by employment history: those employed before 1946, SMR=161, and those employed after 1946, SMR=32; those employed less than 10 years, SMR=41, and those employed greater than 10 years, SMR=144. Impressive overall elevations are seen in the smelter workers for deaths due to "Other Hypertensive Disease" and deaths due to "Chronic and Unspecified Nephritis, Renal Sclerosis" (SMR=369 and 250, respectively) when these are distributed by date of first employment and duration of employment, both distribute in a direction that is consistent

with an association with work in smelters (see table 1).

TABLE 1

	First employment		Years employed	
	Before 1946	After 1946	>10	<10
Other Hypertensive...	475	238	314	400
Chronic and Unspecified and Nephritis, Renal Sclerosis	358	143	195	284

When the authors attempt to adjust results for possible racial differences, their efforts suggest that race alone does not account for the differences reported. In sum, then, there is good evidence in the smelter populations that mortailty due to CNS vascular disease and hypertensive cardiovascular-renal disease is excessive in smelter workers and probably has a work-related etiology. A reasonable hypothesis is that this is related to lead exposure. However, as the authors point out, there are other exposures in smelting environments, and these would have to be excluded before final acceptance of such a hypothesis is possible.

Reviewing these same cause-of-death categories for battery workers reveals somewhat different results. There is no suspicious excess of "Vascular Disease Related to CNS" (SMR=76), or "Hypertensive Heart Disease" (SMR=90). Battery workers, however, do show excess SMR's for "Other Hypertensive Disease" (SMR=207) and "Chronic and Unspecified Nephritis, Renal Sclerosis" (SMR=163). OSHA has chosen not to discuss the results distributed by date of first employment or length of employment because of the grossly uneven distribution of deaths in these subgroupings which were commented on earlier.

The authors appear to suggest that even though there is some evidence for excess mortality for "Other Hypertensive Disease" and "Chronic and Unspecified Nephritis, Renal Sclerosis," that this is not compelling in the face of the absence of clear evidence that excess risk is present for "Vascular Diseases of the CNS" and "Hypertensive Heart Disease." They cite several historical studies (five of the six unlikely to have been controlled for cigarette smoking history) in support of the lack of an association of hypertension in general with lead exposure. On the other hand, they do report literature supporting specific association of lead poisoning with chronic renal damage. The impression is that they believe that there is an association in their study population between exposure to lead and excess mortality from chronic renal disease, but that they think this reflects exposures much higher than the current permissible exposure limit.

The only evidence that is possible inconsistent is the absence of a general excess of disease associated with hypertension. There are, however, too many contributing causes to hypertension other than possible lead exposure to be surprised at the absence of a lead exposure associated risk in other hypertension categories. The most reasonable summary of this data is provided by the authors in the Final Report.

> Despite the uncertainties of diagnosis inherent in death certificates, the excess deaths in our studies from "Other Hypertensive Diseases" and "Chronic Nephritis or Other Renal Sclerosis" collectively support the view that lead may be associated with cronic renal disease. (Ex. 5 (28), p. 107.)

The authors go on to suggest that since this population had high lead exposure in the past, that evidence of chronic renal disease or hypertension associated with lead exposure cannot be used to support the permissible exposure limit below the existing one. This argument is based on the fact that the population under study did not consistently have exposures under the current exposure limit. OSHA reiterates, however, that mortality studies are notoriously insensitive measures of risk.

The overall impression of this study is that excess mortality from chronic renal disease and possibly excess hypertensive vascular disease are seen in the study populations. There appears to be an increased risk, in workers exposed to lead, of suffering from compromised renal function and hypertension disease as a result of their work exposures. It is unlikely that any mortality study will identify a level below which such risks do not occur unless both better measures of long term levels of exposure to lead and information about other causes of hypertension can be made available.

7. *Air to blood relationship.* The proposed lead standard reduced the permissible exposure limit from 200 $\mu g/m^3$ to an 8-hour time-weighted average concentration, based on a 40-hour workweek, of 100 micrograms of lead per cubic meter of air (100 $\mu g/m^3$). The Lead Industries Association (LIA) recommended that OSHA adopt a biological enforcement limit instead of using a specific air-lead number for all industries and operations. One of the key questions raised in justifying a biological standard was the purported lack of a relationship between air levels and blood lead measurements. The purpose of this section is to address the air lead level and blood lead level relationship in detail.

Based upon the evidence in the record, OSHA has concluded that a relationship between air lead levels and population-average blood lead levels unquestionably exists. OSHA is confident that a permissible exposure limit based upon measurement of air lead levels will accomplish the intended goal of protecting workers' health. In addition, OSHA has determined that the Center for Policy Alternatives' application of the Bernard Model accurately predicts the effects on blood leads over time produced by changes in air lead levels. OSHA believes that both the basic construction of the Bernard Model of physiological lead transport and the application of the Bernard Model for prediction of blood lead levels represents an accomplishment heretofore unseen in attempts to establish air level to blood level relationships. Insofar as this model takes into account particle size and job tenure it has avoided the weaknesses of earlier studies. The model does, however, incorporate the findings of the earlier studies and is therefore the best synthesis of theory and actual research to date.

No participants in the hearings argued that total reliance be placed upon air sampling or biological monitoring to the exclusion of the other and OSHA will require use of both measures to maximize protection of the lead worker population in general and the individual worker in particular. However, in the enforcement context OSHA will place primary reliance on air lead level measurements to determine compliance with the permissible exposure limit. Further discussion of the permissible exposure limit is found in that section.

The proposed lead standard established the following goals with respect to worker protection:

> Our present judgement is that in order to provide the appropriate margin of safety, as well as to provide significant protection against the effects; clinical or subclinical, and the mild symptoms which may occur at blood lead levels below 80 $\mu g/100$ g, it is necessary to set an airborne level which will limit blood lead levels to 60 $\mu g/100$ g. A maximum blood lead level of 60 $\mu g/100$ g corresponds to a mean blood level of about 40 $\mu g/100$ g, since a mean level of 40 $\mu g/100$ g will result in a range in workers of approximately 20 $\mu g/100$ g at the lower limits to 60 $\mu g/100$ g at the upper limits. Having determined the maximum blood lead level which the protection of employees and prudence permits, and the corresponding mean blood lead level, it is necessary to correlate these levels to the extent possible with air lead levels in order to establish the permissible exposure limit. (Ex. 2, p. 45938.)

In order to establish the correlation between air lead levels and the corresponding blood lead levels OSHA relied on the work of Williams et al. which was the most comprehensive reported study of its kind at that time. (Ex. 5(32)) OSHA, in this final standard, has evaluated the findings of a

series of subsequent studies which became available during the rulemaking process.

a. *Practical and theoretical difficulties in the use of blood lead-air lead correlation.* Almost all of the studies, whether based on observation of general or occupational populations, attempt to relate measurements of blood lead values to observed air lead values by means of linear regression techniques. Note that this does not mean that only linear relationships were developed. The least squared technique was also applied to transformation of the variables, such as the logarithms of blood lead and air lead, in a few studies. There are a number of practical and theoretical difficulties in the design and execution of experiments of this type which should be considered before attempting to discuss and compare the results of the various studies in question.

1. *Properties of linear regression models.* The linear regression technique makes use of complicated mathematical algorithms to determine the best linear "fit" between a number of observations and two or more quantities (such as individual blood lead and air lead values). The result is an equation, which relates the values of the dependent variable to that of the independent variable (simple regression) or variables (multiple regression). If x is the independent variable and y the dependent variable, a regression problem, as we shall consider it, is a problem in which, for a fixed value of x, y has some particular distribution of values. In other words, we are dealing with a series of populations, a different population of y values for each value of x. We say we are studying the regression of y on x.

Our analysis becomes simpler and our results more explicit if we make certain assumptions about the nature of the distribution of y (for fixed x). One assumption generally made is for any x, the distribution of y is normal. Most of the studies discussed below develop simple regression equations which relate blood lead value to air lead level. A few fit the observed blood lead levels to quadratic equations or use the logarithm of one or more of the variables to obtain a better fit to the data, but the principle is essentially the same. In either case an equation is derived in the following form:

$$\text{Blood lead} = F(\text{air lead}) + e \quad (1).$$

where e is an error term. In most cases, a simple linear relationship was fit to the data:

$$\text{Blood lead} = a + b(\text{air lead}) + e \quad (2).$$

where a and b are constants that minimize the sum of the squared deviations from the calculated straight line relationship. It can be shown that, under a number of conditions relating to simple distribution and measurement error, that the constants a and b will accurately estimate the true relationship between the independent and dependent variable. That is, under ideal conditions, the slope, coefficient b, will truly represent the effect of the independent variable on the dependent variable, all other conditions being held constant.

A common error made by many users of regression analysis is to confuse the observed regression coefficients, which describe the numerical properties of a data set, with constants describing the causal relationships between the variables. It is rarely true (outside of the physical sciences) that the causal relationships in a system are well enough defined so that their properties can be adequately defined by a small number of variables. This is especially true in the case of most of the studies reviewed here on air lead-blood lead relationships. The bulk of the data in the record makes it clear that the relationships between blood lead and air lead in occupational populations is not independent of factors such as job tenure and particle size distribution. It is one thing to say that a linear relationship was observed between the blood lead levels and air lead exposure at a given level of statistical significance, for a given sample of workers. It is another thing entirely to use the observed relationship to predict the effect of lowering air lead exposure on even that same sample of workers let alone to generalize to other samples. Generally, it is best to conservative when using cross-sectional data, obtained at a given point in time, to predict effects over time. Rarely will all other factors be held constant. OSHA has reviewed numerous studies including those describing a model which attempt to take such confounding variables as job tenure and particle size into account.

In order to generalize the results of a given study or to predict the nature of the causal relationships at work it is very helpful if one or more of the following types of information is available:

Other studies on similar populations, perferably using different methodologies, so that some degree of confirmation can be obtained.

Some idea as to the kinds of biases and inaccuracies inherent in the studies themselves, so that disagreement between studies can be at least partially resolved.

A plausible physical explanation of the observed relationship, based on other lines of investigation (in this case, for instance, laboratory studies of lead metabolism and transport).

The first two lines of inquiry will now be examined as they apply to the studies of the relationship between blood lead and air lead, while most of the detailed discussion of the physiological bases for predicted responses to particulate lead will be reserved for the following subsection of the Bernard model.

The vast majority of the studies of blood lead-air lead correlations fit linear relationships to the observed data, as in equation (2). It is most probable that while this mathematical relationship may be the best linear fit to the observed data for a given sample of workers, with their specific tenure and exposure backgrounds, it is not an accurate description of the true relationship between exposure and blood lead. Such an equation, used in this fashion, would implicitly assume that there is some kind of mechanism by which particulate lead exposure of any size distribution is quickly and linearly transformed in workers of any job tenure or exposure history into a given blood lead level. This is almost certainly not the case. Blood lead levels are known to depend not only on total current lead exposure but also on previous body burden (related to exposure history, job tenure), the size distribution of the particulates, and individual variability in response to lead exposure. Setting aside temporarily the exact nature of the dependencies of blood lead levels on the various factors, we can write the following equation which is likely to be a more accurate representation of the blood lead-air lead relationship:

$$\text{Blood Lead} = F(\text{Job Tenure, exposure history, particulate size, individual variability}) + e \quad (3)$$

If this expression more adequately reflects the true picture of blood lead-air correlation than equation (2), attempting to fit the data to equation (2) will have the following effects: First, it will systematically bias the values of the a and b, that is, it will make a and b inaccurate representations of the true relationship of blood lead to air lead. By ignoring the effects of job tenure and particulate size distribution, the resulting coefficients, a and b, and the observed relationship between air and blood leads will be only an approximation of the observed effects of air lead, particle size distribution and tenure as they are distributed among the particular population studied. Thus, even two methodologically perfect studies performed on populations with different job tenure, exposed to different particle size distributions would not agree as to the observed effect of the relationship between blood lead and air lead.

Another important effect of this specification error would be to affect the statistical significance of the coefficients a and b. Thus, not only might the observed coefficients be different,

but even for a more or less correct coefficient, its statistical significance could be affected such that no relationship might be detected with a reasonable level of confidence. Finally, the mis-specification could affect the distribution of the error term, e, in equation (2).

In the studies to be reviewed, clearly the most serious source of specification error comes from a failure to include the effect of previous lead body burden in determining current blood lead levels. As will be discussed in a later section, there is much experimental evidence to suggest that during continuous exposure to lead, the lead levels in various organs of the body increase slowly over time, and that blood lead levels probably never reach equilibrium. Thus, if we attempt to predict the blood lead levels for a sample of workmen with different tenures, studies which did not include a term for job tenure would overpredict blood leads given a certain exposure for workers with short employment history, and underpredict blood leads for workers with long job tenure. Regression lines, calculated from mixed-tenure population would then have lower slopes and larger intercepts at 0 exposure than would actually be observed if job tenure were taken into account.

Similarly, none of the studies included simultaneous analysis of the effects of particle size distribution. If, as is likely, most workers exposed to high lead particulate levels are exposed mainly to large particles which are not absorbed very efficiently in the lungs, while workers exposed to relatively low particulate levels are exposed to larger proportions of small particles which are absorbed quite efficiently, another source of bias is introduced, which would also tend to result in underestimation of the slope coefficient in the dependence of blood lead on exposures.

There is little doubt that this kind of specification error affects the accuracy of the prediction of air lead levels required to produce given blood lead levels. Several of the studies, if used to calculate these values, would indicate that even at an average exposure level of 0 $\mu g/m^3$ lead, an average blood lead level of greater then 40 $\mu g/100cc$ would be observed (Ex. 234(22)). Such a finding is clearly at odds with numerous observations of blood lead levels in populations without occupational lead exposures. It is clear that the true "b" intercept is certainly under 25 $\mu g/100g$ and is very probably under 20 $\mu g/100g$ for most areas. Studies which predict behaviors of this kind cannot be used to make accurate predictions of the incremental benefits of exposure reduction at low exposure levels. In those studies where these sources of bias seem smaller the results do not differ very much with predictions based upon a model to be discussed below. This should not be surprising, since the better studies presumably do give a fairly accurate picture of the effects of particulate exposure and size and job tenure as they are distributed across the particular populations studied. These differences in particle size distribution and job tenures between factories and industries is probably one reason why many of the studies generate apparent relationships between blood lead and air lead that disagree. The application of the Bernard model developed during hearings on Medical Removal Protection and discussed below is simply an attempt to generalize the results of these studies, and, thus, to generate a better approximation of the true blood lead-air lead relationship. (Note that the model generates an infinite number of blood lead relationships, corresponding to all possible job tenures. This set of relationships can also be expressed in one equation, similar to a multiple regression result, which includes a term for job tenure.)

Blood lead=a+[b(f(exposure, particle size))]xf(tenure)+e. Hopefully in this manner much of the specification error can be avoided.

It is well known that errors in measuring either the dependent or independent variable can adversely affect regression results. Generally, errors in measuring the dependent variable (i.e., blood lead level) only affect the statistical significance of the slope coefficient, but do not bias it. By contrast, errors in measurement of the independent variable will result in biasing the slope coefficient toward zero. (In general, for a normally distributed measurement error, V, in the dependent variable, the predicted slope coefficient, will differ from the true coefficient, in the following manner:

$$\hat{\beta} = \frac{\beta}{1+\sigma_v^2/\mathrm{var}(x)},$$

σ_v^2 = the variance of the measurement error

var(x) = the observed variance in the independent variable

if all the other assumptions required to make the least squares method unbiased and efficient are fulfilled.)

In most of the studies of blood lead-air lead relationship, the mean of many different air lead measurements was taken, thus minimizing the contribution of measurement error to the total variance of the dependent variable. Only in the case of the Delco-Remy study, where single measurements of air lead measurements air lead and blood lead levels were paired, would the inaccuracies in air lead measurement likely have been a sizable problem (Ex. 285). Of course, to the extent that any of the studies were conducted over short periods of time during which particulate levels were not typical of the average values, measurement error would be a problem.

Another type of measurement error, distinct from the classical "errors in variables," results if the present average lead levels are not typical of past exposure, or if there is some trend in exposure over time. This is so because previous body burden is an important factor in determining present blood lead levels. There is considerable evidence in the record that air lead levels have fallen significantly in the lead industry within recent years. If one were to study a population that previously had been exposed to high lead levels and were at present being exposed to lower levels, the resulting relationship would be biased upward since some of the workers would have elevated blood leads due to previously acquired body burdens.

To summarize, it is probable that a number of sources of error may significantly affect the accuracy of any incremental benefit prediction conducted using any one study of air lead-blood lead correlations. The major sources of error are:

The unjustified causal interpretation of coefficients of simple regression results based on observational data relating to specific populations at one point in time. This causal interpretation is unjustified because of specification errors inherent in the design of these studies. Important variables such as job tenure are omitted from consideration.

The omission of considerations of job tenure distributions and particle size also suggest that none of these results individually is appropriate to use in predicting the effect of air lead reduction throughout the entire industry. (This does not mean that it is not possible to derive a generally applicable model, however.)

Most of these studies include no explicit theoretical justification for the

use of any particular fit to the data, whether linear or nonlinear.

Errors in the measurement of air lead levels would, on average, further bias the results of these analyses.

These studies are helpful, however, in obtaining a general idea of the apparent effect of particulate lead exposure on blood lead levels in existing occupational situations. They represent a necessary point of departure from which a more complete general model can be developed.

2. *Studies of air lead-blood lead correlation.* There are in the record a number of reviews summarizing the findings of epidemiological and clinical studies of the relationships between exposure to particulate lead and observed blood lead levels. The majority of these studies deal with nonoccupational exposures to lead particulate, usually at much lower levels than could be expected to be encountered during occupational exposure, and are thus of limited value in determining the response of blood lead levels to the relatively high particulate levels encountered in the workplace (Ex. 86E). These studies are useful, however, in providing a baseline set of normal blood lead levels against which occupational levels can be compared. Many also indicate urban-rural differences in blood levels.

One general population study (the Azar study) was discussed by Dr. Paul Hammond at the lead hearings (Ex. 54), with regard to its usefulness in predicting blood lead levels in the industrial situation. The Azar study was based on data gathered on the blood lead levels and air lead exposures for 150 subjects in California, none of whom had any history of occupational exposure. The subjects had been exposed to average airborne-lead concentrations of 0.2-9 $\mu g/m^3$. Air lead levels were measured three times each. The resulting data was fit to a logarithmic relationship, which indicated that as air lead exposures increased, the corresponding increase in blood levels became smaller for a given increment of air level. Hammond criticized the study as follows:

The limitations of the study so far as its utility for assessing the contribution of air lead to PbB in industrial exposure were threefold. First, the air was general ambient air, not industrial air. These are probably quite different as to aerodynamic characteristics and as to chemical composition. Second, the upper limit of air lead concentration was only 9 $\mu g/m^3$. Third, the variability of contributions of lead from sources other than air was so great that the confidence limits for the regression line were broad. This last limitation is inherent in any cross-sectional study. (Ex. 54; p. 5.)

He did conclude, however, that "* * * the Azar regression equation is quite consistent with the limited experimental data available concerning industrial exposure." (Ex. 54, appendix A.) To support this, he showed that blood level predictions made using the AZAR regression agreed well with the findings of several other studies, most notably the Williams study.

In light of more recent studies conducted in occupational settings it is no longer likely that such a claim could be supported by the bulk of the evidence in the record. Most of the other studies to be reviewed here disagree strongly with the AZAR finding that blood lead levels would practically level out (with blood lead levels in the low 40's) at increasing air lead levels above 100 $\mu g/m^3$. The extrapolation of the relationship based on observation at blood lead levels between 0.2 and 9 $\mu g/m^3$ to levels as high as 300 $\mu g/m^3$ could not be expected to give accurate results. It is likely that Hammond's other criticisms also apply.

There have been only a very limited number of clinical studies concerning the relationship between particulate lead exposure and blood lead levels. Probably the best of these, and the most relevant to the occupational situation, were performed by Kehoe, in the 1940's and 1950's. (Ex. 5 (33)).

In the first study, two subjects were exposed to airborne lead particulate in an environmentally controlled experimental chamber for about 8 hours per day, 5 days per week for 88 and 92 weeks, respectively. The lead particulate was produced by burning tetraethyl lead in the flame of a propane-fueled bunsen burner. The first subject was exposed to an average particulate concentration of 75 $\mu g/m^3$, and the other to a particulate concentration of 150 $\mu g/m^3$. In both cases, the blood levels of the subjects appeared to rise rather rapidly after the initiation of the exposure and then stabilize at new levels, and fall slowly after the end of the exposure period. These studies will be discussed in more detail later in this section but are mentioned here because these two subjects provide data points for a number of blood lead-air correlation studies.

Recently, a number of studies have been performed in the workplace, of workers' responses to particulate lead exposure, the results of which are summarized in table 1. The best known of these studies was performed by Williams et al. (Ex. 5 (32)), who observed 39 workers in a battery factory. Particulate exposure was measured, using personal samplers, for 10 consecutive work days; blood samples were taken daily during the second work week. It was assumed in analysis that within the occupational range, the relationship between blood lead and air lead levels was linear.

The use of this study for rulemaking has been attacked on several grounds, which center around Williams' inclusion in his experiment sample a number of very lightly exposed workers, who have very low blood levels. The Lead Industry Association claims that if this group of workers is omitted, then the observed slope of the blood lead—air lead relationship is much smaller, which is true. Globe Union has developed a log—log relationship which they claim fits all of the data much better than Williams regressions. (Ex. 466). Williams himself reported that he detected a large systematic error in all lead measurement just after the study was published (Ex. 234(8)). All of these critics caution against the use of this regression alone to predict the effect of imposing blood lead standards.

Despite the possible shortcomings in this study, the general feature of the results obtained do not disagree widely from those of the other studies to be discussed. Even if this result is fortuitous, the general care with which this study was executed seems to justify including it in developing a general model of blood lead-air lead response. For example, even if the controls are removed the regression equation is $Y=46.07+.12X$ which is not inconsistent with other studies. Note however, the magnitude of the Y intercept. This is consistent with OSHA's examination of error sources, i.e., Y intercept biased high.

In 1976, Buncher *et al.* (Ex. 285) analyzed a large body of data on particulate air lead and blood lead levels gathered by Delco-Remy as part of its monitoring and medical removal program in their Muncie, Ind. plant. Particulate levels were measured using stationary and personal samplers. Using paired single observations of blood lead and air lead measurements, a simple linear regression model was developed. While a positive and statistically significant correlation between blood lead and air lead was observed, the dependence was much weaker than observed by Williams for a similar population of workers. It is unfortunate that this study used only single paired air lead and blood lead measurements, taken as much as 30 days apart, to calculate the relationship between blood lead and air lead. It is likely that the measurement error (in air lead levels) in this study, which resulted in one of the smallest slope coefficients relating blood lead to air lead of any study in the record, were sizable. This would result in the slope coefficient being biased toward 0.

Even if the actual measurement error were small, measuring blood leads and air leads at points this far removed in time would produce inaccuracies produced by real fluctuations in both blood and air lead levels. At best, this study thus measures a fairly

close approximation between current blood lead levels and recent exposure. Another analysis of the Delco-Remy data has been carried out by NIOSH (Ex. 86D), which contains some exploratory data analysis dealing with blood lead-exposure and blood-lead tenure relationships. It is unfortunate that further analyses were not performed on this data, since it is probably the largest, most complete body of information on a working population exposed to lead particulate that is available anywhere, covering observations on about 700 employees of varying employment tenures, at varying exposure levels, for 3 consecutive years.

Globe Union, Inc. has also conducted research on blood lead-air lead correlations among its employees. (Ex. 235) The blood lead levels of 15 workers were observed over a 6-month period, during which frequent blood lead determinations were performed and personal exposures were monitored with back-pack samplers. Again, a linear dependence of blood level on particulate exposure was fit to the data. The relationship was similar to that observed by Williams *et al.*, with a somewhat smaller slope.

King *et al.*, (Ex. 335, p. 63–66), have studied blood lead-air lead relationships in workers at three battery and pigment plants in Britain. Their study is unusual among the studies in the record in that it is one of the few which explicitly incorporates considerations of particle size and solubility differences, an important factor in determining blood lead levels. Unfortunately, they did not analyze the simultaneous effect of particle size and total particulate lead on blood lead, so that their observed blood lead-air lead correlation suffers from the same inherent specification errors common to all the occupational studies.

The effect of not including job tenure in the analysis is particularly apparent in this study, in which the majority of the relationships derived indicate that workers with no occupational exposure would have blood lead levels greater than 40 μg/100cc. This is clearly at odds with observations on unexposed populations. King himself has indicated that virtually all the subjects of the study had been employed at least 2 years. (Ex. 234 (22)). This implies that virtually all of the subjects had developed an appreciable body burden of lead prior to the initiation of the study. This would result in the biasing of the intercept at zero exposure upwards, just as has been observed.

In addition, King's studies include large number of workers exposed to particulate lead levels much greater than 200 μg/m³. This could produce two effects. First, it is possible that only particularly hearty workers with weak response to lead exposure could tolerate such exposures for long. The experimental sample could be biased toward workers with lower than average sensitivity to lead exposure. King specifically denies this, but it seems that it would be rather difficult to determine whether or not sensitive workers had been selected out or not.

Another problem with the King study, including as it does large numbers of subjects exposed to very high levels of lead particulate, is that it might not be a very good predictor of blood lead levels in the region of interest, 0 to 200 μg/m³. This is so because particle size characteristics or physiological responses might change at extremely high exposures.

Two studies of air lead-blood lead relationships in the primary smelting industry were performed by ASARCO. The "El Paso Study" dealt with workers at one plant in Texas. It was an extremely well-controlled study in which total particulate levels were measured for each subject for 10 consecutive working days with backpack samplers, and three blood samples were taken during the second week of the study for each worker. Again, a large, statistically significant linear relationship was observed to exist between total particulate lead exposure and blood lead levels.

The final study often quoted in the record was performed by Sakurai (Ex. 5(9)) in an automobile parts factory in Japan. It is difficult to draw any conclusions about the form of the blood lead—air lead relationship, since the primary thrust of the article was directed at other measures of biological response, and, in fact, only one data point relating air lead levels to blood lead levels was given. Workers in one department who had been exposed to an average particulate lead level of 59.7 μg/m³ had an average blood lead level of 51.8 μg/100 g.

TABLE 1.—SUGGESTED AIR-LEAD BLOOD LEAD RELATIONSHIPS

Linear Relationships

Blood Lead = a(Air Lead) + b

Source of Relationship	b	a	Nonlinear
King:			
Smelting (3)	52	0.053	
Battery (1)	45	.032	
Pigments (2a).	30	.07	
Pigments (quadratic fit.)			Blood lead = 26 + 0.12 (Air lead) + 0.000098 (Air lead)².
Globe-Union	39.7	.1229	
ASARCO (El Paso).	32	.185	
Williams	30.1	.201	
Delco-Remy (Buncher).	37.45	.0628	
Azar/Hammond			Log (blood lead) = 1.3771 + 0.153 log [40 (Air) + 128]/168.
Job Tenure (years):			
0.95	25.80	.1521	
3.4	28.30	.2062	
9.0	29.80	.2404	
16.0	30.64	.2604	
28.5	131.46	.2778	

The results of these studies provide data necessary for the development of a comprehensive model of blood lead response to occupational particulate exposure. These studies in and of themselves do not comprise such a model. They do not measure the dynamic response of blood lead over time to a particular exposure level. All that they do is provide a "snap shot" of how past and present exposures have combined to produce given distributions of blood lead levels in more or less typical working populations at one point in time. Even if they were all executed perfectly in the absence of any measurement error, we would not expect them to agree perfectly, owing to differences in tenure distribution in the various populations studied, possible differences in particle size, and other factors including average physical work demands (and hence, total respiration). The potential individual variability arising from this last factor is very large; the respiratory intake of a standard 70 kg man varies from 3.6 m³ during 8 hours of rest to 9.6 m³ during 8 hours of light work or normal nonoccupational activity. Even larger amounts of air are taken in during heavy work. Six subjects performing heavy work (600-800 kgm-min.) on a bicycle ergometer had total ventilation averaging five times their resting rates. The differences between average total respiration in the various working populations studied for air lead-blood lead correlations are undocumented.

Earlier in this section, possible sources of bias in studies of this kind were examined, that could either produce results that would not adequately reflect the character of the raw data, or would produce results that differ from the true response of blood lead to air lead. It is probable that the unusually low slope in the Delco-Remy study can be at least partially attributed to measurement errors in the air lead values. King's results seem at least in part due to his studying a sample of workers all of which had been employed long enough prior to

the study to have developed significant body burdens of lead, which probably biased the calculated intercept values upward and the calculated slope parameters downward. King's sample also included many workers at very high (300-900 $\mu g/m^3$) air lead exposures.

The remaining studies agree reasonably well, considering the difference in location, methodology, and difference between industries. The results and predictions of all of these studies were used in adapting the Bernard model for use in predicting the response of blood lead levels to occupational particulate exposures. The Bernard model therefore represents the most accurate model to date.

b. *Physiological Models of Blood Lead Response.* In order to accurately predict the effects on blood lead levels over time produced by changes in air lead levels, it is necessary to construct a model that takes into account as many of the important factors as possible which affect blood lead levels. The adaptation of the physiological model originally developed by S. R. Bernard by the Center for Policy Alternatives (Ex. 439), is an attempt to combine experimentally observed properties of mammalian lead transport and metabolism, including consideration of the dynamics of blood lead transport and metabolism, and consideration of the dynamics of blood lead response to long term exposure, with observed physical properties of airborne particulates encountered in the workplace, in order to produce as complete and accurate a picture of the response of blood lead levels to particulate lead exposure as is possible with current information.

The CPA study also included specific consideration of individual variability in response to air lead, which is necessary in predicting the responses of large populations of workers to changes in air lead exposures. One of the guides used in constructing the model was the series of air lead—blood lead relationships observed in specific working populations, subject to the reservations previously discussed concerning the inherent limitations of such studies. The CPA report was an attempt to develop a generally applicable set of relationships which accurately described the response of blood lead levels to air lead levels for a working population with a known job tenure distribution, exposed to a physically defined particulate exposure.

(1) *The Bernard Model.* The Bernard model is an example of one of the most common types of pharmacokinetic models used to describe the transport and metabolism of drugs or foreign substances in the body. It is a multi-compartment mammilary model. Such models postulate that the substance in question first appears in the blood, and then is transported or diffused into a number of different compartments from the blood, corresponding to the different organ systems in the body. Transfer is assumed to occur only between the blood and the organ compartments, not between organ compartments. The rate of transfer into and out of the blood stream from the various compartments depends upon a number of factors, such as whether or not that particular organ specifically takes up or metabolizes the substance in question. In general, especially in the case of substances which are not metabolized, the rate of transfer between compartments is linearly related to the concentration of the substances in the compartments. This is consistent with the basic physical principals of chemical kinetics that would govern the transfer of a substance across an inert membrane in the absence of any other driving force. The relatively few exceptions to the linear transfer principle tend to occur only in cases where an organ specifically sequesters or metabolizes the substance in question.

In the course of the rulemaking process, representatives of the lead industry introduced a number of studies which they claim demonstrate nonlinear response to lead exposure in animals. These studies were subsequently extensively reviewed by Dr. Dale Hattis in his letter to Richard Gross of January 13, 1978 (Ex. 458A). He concluded that none of these studies supported the position that the lead transfer rates in humans were appreciably nonlinear at air lead and blood lead level ranges relevant to standard setting.

In designing a model and calculating the rate of transfer between compartments, the experimenter has many guidelines as to how to proceed. First he/she can simply follow total body excretion to ascertain the number of compartments that are individually taking up and excreting lead after an initial dose. The more exponential terms required to fit the data, the more compartments. Second, the investigator can actually follow the rate of uptake and release of the substance from the various tissues by autopsy or biopsy, and measure the rate of release. This latter approach is impossible, of course, in the study of human subjects. After observing the rates of release of the substance in question from the whole body and/or tissues, the investigator is left with a series of exponential retention equations which relate amount of lead left in each compartment after a given time to initial dose. Using well-developed mathematical techniques, this set of equations can be solved subject to the constraint that all of the ingested substance is accounted for, to yield the rate constants for transfer between compartments.

There seem to be two important considerations which could affect the accuracy of the Bernard model in predicting the behaviors of lead pools in the bodies of workers exposed to lead. Bernard's estimate of the turnover rate of lead in bone is based on measurements made in the skull, where turnover of unmetabolized trace elements is known to be slower than in most other bones of the body. This could result in the underestimation by the Bernard model of blood leads for a given exposure, and underestimation of the time required for recovery below a given level. This problem is probably offset by the observations that after long periods of exposure, lead deposited in skeletal bone tends to become *irreversibly* bound. Any permanent sequestration would cause the Bernard model to overpredict blood lead levels for a given exposure/job tenure combination, especially for long-tenured workers, and an overestimation of the amount of time required for blood leads to drop after exposure. What the overall effect of these two considerations would have on predictions of the model is difficult to say, other than that they would tend to offset each other.

In any event, it is better to make explicit analytical assumptions, based on experimental observations, as the Bernard model does, than to make the implicit assumptions about particulate size and lead transport and metabolism, that are made in simply fitting a simple straight line to blood lead-air lead correlation. The Bernard model as applied to the occupational situation by the CPA report also predicts a linear relationship for a given tenure.

As was discussed above, studies of blood lead-air lead correlations that fit straight lines of blood lead-total particulate exposure implicitly assume tenure to be unimportant in determining blood lead levels, but also assume that all lead particulate exposure, no matter what its size distribution, is absorbed and metabolized with the same efficiency. This is clearly not the case. In the first place, the proportion of particles which are deposited in the respiratory tract, rather than exhaled, varies considerably with particle size. Further, different size particles are deposited in different areas of the lung upon inhalation. In general, most particles less than 1 micron in diameter are deposited in the alveoli, whereas particles between 1 and 10 microns in diameter usually end up in the bronchi, and larger particles end up in the upper respiratory tract. The location of deposition is of some importance, since particles in the alveoli are not

likely to be swept from the lung by the vigorous ciliar activity in the bronchi. On the other hand, particles deposited in the bronchi and naso-pharynx generally are swept into the alimentary tract. Thus, there are two distinct modes of absorption: by dissolution in the alveoli and by absorption through the digestive tract. Two studies, one of them Kehoe's, suggest that small particles are deposited and absorbed with an efficiency of about 37 percent. On the other hand, absorption of dietary lead tends to be much less efficient, on the order of 6 to 10 percent. (Ex. 95).

One of the major uncertainties concerning the use of the Bernard model in predicting costs of medical removal protection is "Assumption C", an attempt to incorporate these aspects of lead absorption into the cost calculating methodology. Assumption C states that all of the first 12.5 $\mu g/m^3$ of particulate encountered by a worker will be small, and thus absorbed with an efficiency of 37 percent, and the rest will be large, and absorbed with an efficiency of 8 percent.

Assumption C has two parts: the first states that, in general, at low particulate exposure levels, most of the lead is present in small particles, and that as the total lead particulate level increases, the increase is made up primarily of larger lead particles. The second part of assumption C is that particle absorption efficiencies differ with particle size as described above.

The latter portion of assumption C is consistent with the bulk of the data in the literature. The theoretical basis for the first portion of assumption C is quite straightforward, as stated in the CPA report:

> Basically, we expect that there will be some tendency for workers with greater total air lead exposures to be located physically closer to sources of lead emission into the workplace atmosphere than their fellow workers with smaller air lead exposures. Because the larger lead particulates will tend to settle out from the atmosphere faster than smaller particulates, workers which are farther from a given lead particulate emission source will tend to be exposed to relatively less large particulates than workers which are closer to that emission source. If the distant workers also tend to be those with smaller total lead particulate exposures, then there will in general be a tendency for workers with smaller total lead exposures to be exposed to greater proportions of small-size particulates. Of course, in real workplaces where there are multiple lead particulate sources which may be expected to give rise to emissions of different particle size distributions we do not expect that there will be a perfect correlation between total lead exposure level and proportion of "large" lead particulate exposure. (Ex. 439B)

There is some data in the record to support the general features of particulate exposure postulated by assumption C. It has been shown that, for one population of mill workers, there is a strong inverse correlation between total exposure and small particulate exposure (see Addenda to CPA report, Ex. 439B). Further, data from the AMAX Buick smelter (Ex. 247) show that for six locations, whose average total particulate exposure is about 1,000 $\mu g/m^3$, the average amount of particulate of diameter less than 1.1 micron is only 21 μg, with only two locations (Dross refinery) having small particulate levels greater than 12.5 μg. It thus appears that, at least in the smelting industry, the vast majority of particulate above 12.5 $\mu g/m^3$ is large.

A single study of the particle-size distribution in the battery and pigment industries is in the record. (Ex. 234 (22)). This data is somewhat difficult to interpret, since the sampling size ranges do not correspond easily to alveolar-bronchial deposition. It does, however, appear that in the factories in question which had relatively high average particulate levels, only a very small amount of the particles were less than 1.0 micron in diameter (or measured as such by the methodology used). In the first factory, which has an average total particulate level of 360.6 $\mu g/m^3$, only 5.6 percent, or 20.2 $\mu g/m^3$, measured as being smaller than 0.7 μg. Extrapolation through the cutoff points of the next sampling plate, which had an upper size cutoff of 5 microns, suggests that in total, only about 21.6 $\mu g/m^3$ of the total particulate was smaller than 1 micron. Results obtained in a similar fashion for the other two factories studied indicate that in one of them, which had a total particulate level of 294.2 $\mu g/m^3$, about 18 $\mu g/m^3$ was smaller than 1 micron, and the other, which had a total particulate level of 121.3 $\mu g/m^3$, only 12.5 $\mu g/m^3$ consisted of particles smaller than 1.0 micron. It is difficult to draw any firm conclusions from these data, but it does tend to indicate that 12.5 $\mu g/m^3$ is a reasonable estimate of the maximum amount of small particulate occurring in occupationally relevant particulate exposures.

Assumption C has been criticized in that it is simply a convenient set of arbitrary assumptions, designed to make the Bernard model fit observed patterns of blood lead-air lead correlation. (Ex. 466). In OSHA's opinion, the reasonableness of both the theoretical and observational bases of assumption C are quite convincing. The fact that assumption C agrees fairly well with most air lead-blood lead correlations, but agrees exactly with none should not be disturbing since these studies do present a reasonably good representation of the effects of particulate level, job tenure, and particle size distribution as averaged over the particular working population studied.

The marginal benefit calculations conducted using the Bernard model and assumption C (See PEL Section.) argue fairly well with those conducted using all of the previously observed blood lead-air level correlations except those with very low slopes. These low slopes are probably the result of specification errors in the blood-air correlation model produced by the presence of many long-termed workers in the sample.

A final criticism which has been made of the application of the Benard model to predict occupational blood lead levels is that the model predictions do not correspond to the classic clinical obsevations of Kehoe on the blood lead level response to controlled exposures to lead particulates. (Ex. 5(33)). The major discrepancies were:

> One of the subjects exposed to particulate lead (F.C.) exhibited blood levels that rose and then began to decline during exposure to 150 $\mu g/m^3$ particulate exposure, while the model would predict that his blood level would rise continuously.
>
> The other subject (M.O.B.) exhibited blood lead levels that reached an equilibrium level quite rapidly after exposure to particulate lead was begun, in contrast to the model prediction of ever increasing blood lead levels.
>
> The blood lead levels for both subjects exposed to particulate lead rose less rapidly than the model would predict.
>
> Subject F.C. exhibited increasing urinary lead levels while his blood lead level was decreasing, contrary to model predictions.

Before examining these objections, a brief review of the experimental conditions of the Kehoe study would be in order.

Both subjects were exposed to particulate lead consisting of very fine particles of lead oxide, with a mean diameter of 0.05 micron, produced by the combustion of tetraethyl lead in a small propane burner. Subjects were exposed to lead 8 hours/day, 5 days/week, for periods up to 92 weeks, in a small, cubic experimental chamber 10 feet on a side. The dietary intake of lead was monitored closely, and precautions were taken so that none of the particulate lead in the chamber was ingested, rather than inhaled. The experimental subjects were given strict hygiene instructions and the experimental chamber, especially designed to avoid dust buildup, was cleaned each day prior to the exposure period.

Several of these features of the experimental design make this study a poor simulation of the occupational situation. The size of the lead particulate, which had no particles larger than 0.17 micron, was much smaller than the particulate exposures generally encountered in industry. The bulk of most particulate exposures is larger

than 1 micron in diameter, which means that while most industrial exposure will probably be deposited in the bronchi or naso-pharynx and be absorbed in the alimentary tract with about an 8 percent efficiency. All of the rather high levels of particulate exposure in the Kehoe studies would be absorbed with an efficiency of about 37 percent after deposition in the alveoli. Also, by confining the subjects into the very small experimental chamber and supplying them with only light bookkeeping or laboratory tasks to perform, it is likely that the experimental subjects were not nearly as active as the typical lead worker. Thus, they probably required less oxygen, and hence breathed much less lead particulate, than they would have in an industrial situation. And finally, of course, the laboratory cleanliness of the experimental chamber is hardly typical of the workplace.

These considerations not only make these experiments poor predictors of the response of blood lead to particulate exposure in the occupational context, but they also make the experiments themselves difficult to model accurately.

It must be assumed, that since the particulate is all less than 0.17 micron in diameter, that it will all be absorbed with an efficiency of 37 percent. If one then simply runs the models, assuming the experimental subjects breathe 9.6 m^3 air per day, then the model predictions for at least one subject approaches the observed result.

In regard to the specific claim that the blood levels of subject F.C. begin to fall during the exposure period, it can be said that it is difficult to tell, although that may be the case. Similarly, it is difficult due to large fluctuations in the blood lead levels to decide whether the blood lead levels of subject M.O.B. really reach an equilibrium value or do not actually continue to rise somewhat slowly as the model would predict. That the model would not predict rising urinary lead levels while blood levels were decreasing is true. It thus seems that the model does only moderately well in predicting the results of Kehoe's experiments on airborne lead exposure.

The model does do quite well, however, in predicting the response of blood lead to dietary intake of lead as studied by Kehoe. In these experiments, one subject each was given 0.2, 1, or 2 mg lead/day orally in solution, for periods of up to 4 years. To run this simulation, it was assumed that each subject was given the indicated amount of lead, in addition to normal dietary lead, every day, and that it was absorbed with an efficiency of 8 percent. All subjects were assumed to start at a blood lead level of 23 $\mu g/100$ g, with corresponding pool levels.

The model predictions parallel the observed data quite well for the two subjects who were given the larger daily doses of lead (E.B. and M.R.), but not so well for the subject (S.W.) who was given the lowest dose. It should be noted that due to large fluctuations in his blood lead prior to exposure, it was difficult to decide what value of blood lead to start the simulation with. At any starting value between 23 and 35, the model would predict that this small an oral dose of lead would produce a more or less stable blood lead level (in reality, actually slowly increasing or decreasing) for the duration of the experiment. It should also be noted that Kehoe could detect no significant difference between the blood lead levels of this subject during the control period and during the period exposure.

To summarize: The Bernard model seems to do reasonably well in simulating the data on three out of five of the experimental subjects studied by Kehoe. The data on another subject is somewhat equivocal. Only for one subject (F.C.), exposed to a relatively high particulate level, does the model fail to accurately predict the general features of the response of blood lead levels to lead exposure. Since there are numerous possible sources of variation in individual responses to lead exposures, failure of the model to predict well for one subject out of five is acceptable.

c. *Variability of individual blood lead levels.* This section will attempt to identify and analyze some factors which contribute to the observed variability in blood lead levels. This is necessary because, in order to predict the number of workers that would be expected to be above 60 $\mu g/100$ g a number of probabilistic assumptions about the variability of individual blood lead levels must be made. Original assumptions made have been the subject of some criticism.

One assumption made by CPA was that all of the variability in blood lead levels in a population of workers who had similar job tenures and exposure histories would be due to individual variations in physiological response to particulate exposure. A second assumption, supported by the Delco-Remy and Williams data is that the average standard deviation for a population of workers produced by this individual variability is ± 9.5 $\mu g/100$ g blood. It was implicitly assumed that blood lead variations are normally distributed about the average value for a given tenure-exposure population:

$$(\text{Blood Level})_i = \text{average blood lead} + e_i \quad (1)$$

The blood lead level for the worker is equal to the average blood lead level for that tenure-exposure combination plus a normally distributed error term with an expected value of 0 and standard deviation of 9.5 $\mu g/100$ g.

Originally the 9.5 figure was adopted as a conservatively high figure, based on summary statistics from the studies of American and British battery workers cited previously. A number of criticisms have been leveled at the use of these probabilistic assumptions (Ex. 451B). It has been claimed, first, that not all variations in observed blood lead levels is due to individual variations in physiological response, but rather there is also a large contribution from errors inherent in measuring blood lead levels. Second, it has been claimed that the observed blood lead distribution tends to be log normal, rather than normal, which would result in a higher proportion of a given population having high blood lead levels than in the normally distributed case. A third criticism is that, even if the observed distribution of blood leads is normal, the observed standard deviation of blood lead levels is greater than 9.5, owing to the abovementioned measurement errors and contribution due to short term, rather than long term, fluctuations in individual responses to lead exposure. Claims have been made that a figure as high as 15.5 $\mu g/100$ g would be a more accurate estimation for the standard deviation in blood lead levels.

In order to study more fully the basis for these criticisms, a few brief analyses of some of the data on the record were performed. Attempts were made, using data from several sources representative of typical industrial populations, to decide whether or not blood lead measurements for populations with similar exposure-tenure histories were normally distributed, and if so, how large the variation was likely to be. Analyses were also performed to decide how much of the variability in blood lead levels could be attributed to long term variability in individual response to lead exposure, how much could be attributed to real short term variability in blood lead levels and how much could be attributed to errors in the measurement of blood lead levels.

If long term variation in individuals' susceptibility are not the only source of variability in blood lead levels, then, e_i in equation 1 in reality becomes the sum of two or more kinds of variations:

$$e_i = e_1 + e_2 \quad (2)$$

In this case, e_1 is the error due to long term variation in individual response to lead exposure, and e_2 is the error term due to other sources, such as short term fluctuations in individual levels (weekly, cyclic, etc.) and measurement error.

Using raw data from the Delco-Remy biological monitoring program, attempts were made to answer the following questions: Is either e_1 or e_2 not normally distributed? (In particular, is either e_1 or e_2 log-normally distributed?) If either e_1 or e_2 is log-normally distributed, then it is unlikely that e_1 is normally distributed. Second, what are the magnitudes of e_1 and e_2? We know from basic statistics that, if e_1 and e_2 are independent of each other, then: Variance=(Deviations from mean value).[2] The standard deviation is the square root of the variance.

$$Var(e_1) = Var(e_1) + Var(e_2) \quad (3)$$

Thus, we can estimate the standard deviation of e_1, the observed variation in blood lead levels, from the variances of e_1 and e_2.

An attempt to estimate e_1, the error due to long term variations between individuals in response to lead exposure, and to decide whether these variations are distributed normally or not about the mean value was made in the following manner:

From the summary statistics in exhibit 86D, departments at the Delco-Remy plant were divided, according to the mean blood lead values for all of the male employees, into groups of departments with average blood lead levels that were equal (group 1=departments with average blood level between 40-41.9; group 2=departments with average blood lead levels between 42.0-43.9; etc.). It was assumed that departments with similar average blood lead levels consisted of workers with similar exposure-tenure histories. To the extent that this is not true, values calculated for e_1 will be overestimated. Since the values for each department are the mean of the yearly average blood values for the workers in the department, they contain little variation that could be attributed to short term variation or measurement error. Since the values are also assumed to come from populations with similar exposure-tenure histories, all of the variations observed can be attributed to long term individual variations in response to lead exposure, that is, to be equal to e_1 in equation 4. The observed standard deviation for each of these groups of departments is given in the third column of table 2. The average observed standard deviation for e_1 in all the departments is 5.46 μg/100 g.

In order to determine whether the blood lead levels for the individuals in the various departments were normally distributed, the distribution of the individual blood lead levels for each department within the five groups were pooled, and subjected to a X^2 goodness-of-fit test with the appropriate number of degrees of freedom. The results are also given in table 2.

TABLE 2.—*Long-term variability of blood leads for departments with similar average blood lead levels*

Departmental average blood leads	N (workers)	Average std. dev.	X^2(i)* (for normality)	Prob. (H_o=false)
40-41.9	307	5.48	9.85 (7)	~0.60
42-43.9	212	6.67	6.07 (8)	~0.5
44-45.9	315	5.21	7.06 (7)	~0.5
46-47.9	249	4.87	14.46 (6)	~0.95
48-51.3	135	4.91	1.76 (6)	~0.05
		=5.46		

*Figures in parentheses—number of degrees of freedom.

Only in the case of one group (average blood lead=46-47.9) does the goodness-of-fit test suggest that the observed long term variability is not normally distributed. In all of the other cases, with this sample size, the goodness-of-fit test cannot distinguish any of the distributions from normal.

In order to estimate e_2, the variations due to measurement error and short-term individual fluctuations, 27 workers whose yearly average blood lead level did not change more than 2 μg/100 g for the period of observation (1974-75) were selected. Since their yearly average blood lead levels did not change from year to year, it was assumed that the contribution to the total variation from long-term variability was zero. Thus, all of the observed variation must have been due to short-term variability in blood lead levels and measurement error.

For each of these workers, all of whom had had their blood lead measured at least four times in each year, mean blood leads and standard deviation were calculated. There were 300 observations in all. The average standard deviation for a single worker, whose blood lead level was stable in the long run (e_1=0), was 7.32 μg/100 g (variance=53.6). The X^2 value (10 d.f.), for this distribution, tested against a normal distribution with σ=7.32, is 6.52; the hypothesis that the distribution is normal cannot be discarded at any more than a 30 percent level of confidence. The same distribution, tested against the log-normal distribution generated from the same data (σ^2=0.004812), gave a X^2 value (9 d.f.) of 13.19; the hypothesis that the distribution is log normal can be discarded at a level just under 90 percent. This sample more closely resembles a normal distribution than a log normal distribution. The distribution is skewed somewhat to the right, however. But still, in as much as these techniques can determine, the upper outliers are explained at least as well by the normal distribution as by the log normal (judging by overall contribution by X^2).

As far as this limited analysis is concerned however, there seems to be only slight justification for claiming that either the long-term variation in blood lead level (e_1) or short-term variations (e_2), including measurement errors, are not normally distributed, at least for this set of data.

e_1, the observed variations in individual blood lead containing both short-term, individual fluctuations and long-term differences in physiological response, as well as measurement error can now be calculated:

$$Var(e_1) = Var(e_1) + Var(e_2) = 29.8 + 53.6 = 83.4 \quad (4)$$

The standard deviation of e_1 is thus 9.14 μg/100 g. This is just slightly less than the value used in the CPA analysis, and suggests that the latter is an appropriate value to use for cost calculations. Also, there are other instances in the record where a similar short-term variability in blood lead measurements was obtained. In the ASARCO El Paso lead study, (Ex. 4(5)), three blood lead measurements were taken on 42 workers (two on another) in 1 week. The average variance for a single worker was 79.3. Using this value of calculate e_1 (e_1=5.45), we get an observed standard deviation of 10.4 μg/100 g for individual blood lead determination.

In one study of the accuracy and precision of blood lead determination, 15 laboratories were given five portions of a single blood sample to measure the repeatability of the blood lead measurements. The average standard deviation for a single laboratory was 3.7 μg/100 g. Two laboratories did extremely poorly, with standard deviations of 10.0 and 15.5 μg/100 g. All the rest had standard deviations less than 3.5. The standard deviation for the 13 best laboratories ranged from

1.4 to 3.4, with an average standard deviation of 2.3 μg/100 g lead. No single analytic technique seemed to consistently do better than any other. (Roettgers, PbB Reference Control Program, West Allis Memorial Hospital, March 21, 1975.)

These results suggest that, in most cases, measurement errors do not contribute a large amount to the observed variability. This suggests that the observed number of blood lead values greater than 60 depends to a large extent on the nature and magnitude of true short-term fluctuations in blood lead level which appear not only in the Kehoe study, but in the Delco-Remy data as well, are real variations, not primarily due to measurement errors. This means that even if an individual worker has a long-term average blood lead level less than 60 μg/100 g, he may spend a significant fraction of the time above this level due to real short-term variations in his blood lead level.

In trying to make a final decision about what a reasonable estimate of variablity of blood lead levels would be, the answer would depend upon several factors. A lower bound estimate would involve using an e_l (σ=5.46), since it probably is a reasonable estimate of the observed long-term variability of individual blood lead levels. If one wished to include considerations of short-term variation, then e_l=e_l+e_s(σ=9.1-10.4) would be a good estimate, assuming reasonably low measurement errors. If one were to include conservatively high estimates of measurement error, then an upper bound estimate for the standard deviation of observed blood lead variability of σ=15.0 would be in order.

d. *Air Lead-Blood Lead Relationship.* In criticism of the CPA model and of the standard in general, the issue of the interpretation and usefulness of studies of blood lead-air lead relationships has frequently arisen. Some critics have claimed that no meaningful correlation exists between blood lead and air lead levels, or that the relationships are so weak that few health benefits would be attained by reducing exposures below 200 μg/m³. Others claim that there is so much variability between individuals and between studies that attempts to predict blood lead levels on the basis of exposure are useless. Much of this criticism is based on fundamental misunderstandings about the nature and meaning of regression analyses and the uses to which they can be put.

One of the Lead Industries Association contentions is that observed correlations between blood lead and air lead are too weak and variable to justify the imposition of standards for air lead levels. They cite the King and Delco-Remy studies to claim:

Air lead exposure is useless in predicting individual blood lead levels.

The data on blood lead-air lead correlation is so weak that they do not constitute an adequate basis for estimation of health benefits produced by decrease in exposure.

Even if the best studies are to be believed (they claim the King and Delco-Remy studies to be superior), they indicate that the incremental benefit of imposing an air standard below 200 μg/m³ would be minimal (Ex. 335).

The question may be asked do blood lead-air correlations exist? The answer is clearly yes. Despite the list of witnesses to the contrary quoted by the LIA, these are a long list of observational studies in the record which indicate that highly statistically significant correlations can almost always be found between airborne lead exposure and blood lead, where care is taken to measure accurately enough, and the appropriate data analytic methods are used. Many of these correlations appear to be small in magnitude, but there are at least two good reasons why this should be so. Most studies of blood lead-air lead correlation do not take into account differences in job tenure between workers. The inclusion of long-tenured workers with higher body burdens of lead, but low current exposure, biases the observed regression slope coefficient toward zero. Similarly, the presence of newly-hired workers with heavy current exposure but no previous body burden would also bias the slope of the air lead-blood lead dependence downward. That this effect is important is borne out by the results of several of the studies (including the King study) which indicate that even in the absence of any occupational exposure, the average worker could be expected to have a blood lead level well above 40 μg/100 cc. Another factor which would make blood lead-air lead correlations weaker than they actually are is error in measurement of air lead levels. Errors in measurement of the values of the independent variable will almost invariably result in a decrease in an observed slope of a regression line. It was shown that, owing to the known large errors in air lead measurements, this magnitude of this effect could well be significant. It is interesting to notice that the Delco-Remy study (Ex. 285), which had one of the lowest slope coefficients of almost any study, was based on single air lead measurements for each individual worker. All of the other occupational studies made use of at least 10 measurements/individual to minimize the errors in measurement of the air lead value. It is likely that the results of the Delco-Remy study were significantly affected by measurement error.

It is true that any one of these studies individually provides a poor basis for predicting individual blood levels. They certainly do better, however, in forming statistical predictions about average blood lead values and about the frequency of occurrence of blood lead levels far from the mean.

Can the air lead-blood lead correlations in the record be used to provide adequate predictions about health benefits from airborne exposure reduction?

In the strictest sense, no they cannot. The air lead-blood lead studies in the record provide measurements of the observed relationship between blood lead and air lead levels in specific populations at one point in time. They do not take into account variation in job tenure and previous body burden, and they do not furnish adequate approximations of the response of blood lead to air lead exposure over time. They do reflect a fair approximation of the effects of distribution in the various industries and firms, and thus provide rough estimates of the effect of changes in air lead levels and blood lead levels for that particular population, subject to the inherent limitations discussed above.

Owing to differences in job tenure distribution, measurement errors, and other factors, it would not be expected that any two studies would agree exactly. Owing to the specification and measurement errors, it could also not be expected that one of the studies would provide an accurate picture of the true effect of air lead exposure on blood lead, even for any particular job tenure, except fortuitously.

Thus, arguments about which study of blood lead-air lead correlation most accurately predict the magnitude of health benefits which would accrue from the imposition of a given standard are not very meaningful.

In summary, one could not expect to make accurate calculations of the effect on blood lead of reducing air lead exposure on the basis of any single short term cross-sectional study. To the extent that the individual studies agree, they help confirm the finding that a more or less linear relationship seems to exist between blood lead levels and current air lead exposure in the lead industry. To the extent that they disagree, they indicate the need for a more comprehensive model of blood lead response to airborne particulate lead exposure over time which the Bernard model accomplishes.

No cross-sectional study of air lead-blood lead relationships is likely to be an accurate predictor of blood lead response to airborne particulate lead exposure. A "snap shot" observation, taken of the blood lead levels and current exposures of a number of workers does not measure, except indirectly, the effects of lead exposure on blood lead levels over time. Cross-sectional

studies will always include large numbers of workers whose blood lead levels are determined primarily, not by present exposure, but by lead body burdens accumulated over their entire job history.

This inclusion of long-tenured workers in studies of this kind will result in predictions of unreasonably high blood levels at low exposure, and produce unreasonably low estimates of the slope of the dependence of blood lead on air lead.

Most of the relationships generated by the King study predict that the average blood lead levels observed in individuals with no occupational exposure to lead would be greater than 40 μg/100 cc. These unreasonably high values suggest that the results of King's studies have been affected by the inclusion of many workers with high initial body burdens. In addition, King's studies include large numbers of workers exposed to particulate lead levels much greater than 200 $\mu g/m^3$. This would produce two effects. First, it is possible that only particularly hearty workers with weak response to lead exposure could tolerate such exposures for long. The experimental sample could be biased toward workers with lower than average sensitivity to lead exposure. King specifically denies this, but it seems that it would be rather difficult to determine whether or not sensitive workers had been selected out or not. Another problem in King's study, including as it does large numbers of subjects exposed to very high blood levels of lead particulate, might not be a very good predictor of blood lead levels in the region of interest, 0 to 200 $\mu g/m^3$. This is so because particle size characteristics or physiological responses might change at extremely high exposures. Also, for two of the factories King studied, the results of the analysis depend rather heavily on relatively few outlying points.

In addition, there are methodological problems that might well result in studies of current air lead-blood lead correlations producing results that were not even accurate representations of the existing blood lead-air lead distributions. The most obvious of these is measurement error. If the average error in measurement of the independent variable in a regression is significant, compared to the actual variation in the data, the result will be that the observed slope coefficient will be biased toward zero, as long as the error is not correlated from observation to observation. As was previously discussed all of the studies of blood lead-air lead relationships in the record, except the Delco-Remy study, took this into account, and measured air lead levels many times and averaged the results to minimize measurement error. The Delco-Remy study conducted by Buncher, was constrained by the data available to pair single observations of blood lead and air lead for the analysis. It is highly probable that the pure measurement errors included in this will be that the very small slope observed in this analysis is due at least in part to errors in air lead measurement.

In summary, no study that relates cross-sectional data on present air lead and blood lead levels without taking tenure specifically into account is likely to be an accurate predictor of the effect of particulate exposure on blood lead. Specification errors (the exclusion of tenure) and measurement errors in air lead levels are both likely to bias the observed slope coefficients downward, and produce unrealistically high predictions of blood lead levels at low exposure. There is no one simple relationship between air lead and blood lead. There are, in reality, a set of them, one for each tenure and work load combination. If one were forced to choose one particular blood lead-air lead study to predict changes in blood lead levels after a change in exposure levels, there is no good reason why one should choose either the King study or the Delco-Remy study over any other, and a number of reasons why one might not choose them.

There were several criticisms of the design of the Bernard model itself, as distinct from the CPA adaptation of it. Most centered on one of two areas: The use of animal studies in developing a model for human lead metabolism, or the accuracy of the assumptions of linear transfer rates between compartments. LIA argues that "The Bernard model was based on limited data from a single experiment on baboons, each of which had received but one injection of radioactive lead." Thus its application to the prediction of blood lead levels in humans was unjustified. (Ex. 453 (19).)

The Bernard model was based on data from a number of experiments on baboons, humans, dogs, and rodents, not from "a single experiment on baboons." OSHA is confident that the quality of data obtained from the animal studies, particularly the baboon study is scientifically sound and that the general design of the Bernard model is without serious weakness.

The second major criticism of the Bernard model was that it postulated transfer rates between compartments ("linear transfer rates"). There seemed to be a good deal of confusion about what "linear transfer rate" meant. A few critics felt that it meant that blood lead was linearly related to exposure, which is one result of the model, given a constant tenure, but not necessarily dependent on linear transfer rates. Dr. Cole, the representative of the Lead Industries Association, cited seven studies to this effect. (Ex. 446.) Dr. Hattis of the CPA has responded in his letter of January 13, 1978 (Ex. 458A), to these studies, indicating why he believes that none of these studies provides convincing evidence that the assumption that the physiological response to lead exposure in animals is nonlinear over appropriate ranges of lead exposure.

The Lead Industries Association also claims that the CPA use of the Bernard model is inappropriate, since the amount of lead (0.419 mg.) in the R1 pool, supposedly corresponding to blood lead, is not large enough to produce a blood lead of 19 μg/100 g in an individual with a normal blood volume of 5 liters. (The concentration would be 7.9 μg/100 g instead.)

This approach misunderstands that the definition of the compartments as used in the Bernard model applies to kinetically defined pools of lead, not lead located in any one particular physical location or organ. In this case it is possible that there are two pools of lead which correspond more or less to that level circulating in the bloodstream at any one time. For instance, if the contents of the other rapidly-exchanging pool, are added to the contents of R1, the resulting total would produce a concentration of approximately 22 μg/100 g lead in 5 liters of blood. This would be consistent with a situation in which not all of the lead in the blood exchanged rapidly.

In fact, lead is known to be concentrated primarily in the red blood cells rather than in the plasma, and may well be too strongly bound to exchange quickly. Also, contrary to the LIA claims (Ex. 453), the validity of the Bernard model does not rely on a dietary content of 440 μg lead/day. It was assumed that the total intake of lead from all sources, including airborne lead as well, was 35.2 μg/day. This 35.2 μg of lead actually absorbed into the body to produce the equilibrium pool levels in equivalent to a dietary exposure of about 150 μg/day plus an airborne intake equivalent to 16 hours light activity and 8 hours rest at an ambient air lead level of about 2.5 $\mu g/m^3$ (a reasonable urban exposure).

"Assumption C" was used to describe the intake of lead that could be expected to result from different total particulate exposures. Assumption C states that, on the average, occupational particulate exposures totaling less than 12.5 $\mu g/m^3$ are composed primarily of small particles, less than 1 micron in diameter which are deposited mainly in the alveoli and absorbed with an efficiency of 37 percent. For exposures greater than 12.5 $\mu g/m^3$, the first 12.5 $\mu g/m^3$ is assumed to be made

up of small particles and the remainder composed primarily of particles larger than one micron, which are absorbed in the alimentary tract after being carried from the bronchi and nasopharynx by ciliar action.

Most of these criticisms argued that the intake-exposure assumptions incorporated into assumption C were not valid (Ex. 466). Generally, it was maintained that since the majority of particulate exposures were "respirable", that assumption C could not be correct. Very little quantitative data was used to support this position. Dr. Hattis has already demonstrated that at least in one smelting plant, the amount of small particulate is inversely correlated with total particulate levels. Also, in the three factories in the King study (a battery plant, a pigments plant, and a smelter) with total average particulate lead levels of 120–360 $\mu g/m^3$ the average amount of particulate smaller than 1 micron was about 18 $\mu g/m^3$. A similar picture was found from a number of sampling locations in one AMAX smelting plant.

Marjorie Lundquist also states (Ex. 466, p. 5) that only 20 percent of the lead particulate generated by the Globe Union pasting operation is less than 2 microns in diameter. This implies that at most 10 percent is smaller than the 1 micron size above which little alveolar deposition occurs. The physical justification for this part of assumption C has been discussed.

Basically, we expect that there will be some tendency for workers with greater total air lead exposures to be located physically closer to sources of lead emission into the workplace atmosphere than their fellow workers with smaller air lead exposures. Because the larger lead particulates will tend to settle out from the atmosphere faster than smaller particulates, workers which are farther from a given lead particulate emission source will tend to be esposed to relatively less large particulates than workers which are closer to that emission source. If the distant workers also tend to be those with smaller total lead particulate exposures, then there will in general be a tendency for workers with smaller total lead exposures to be exposed to greater proportions of small-sized particulates. Of course, in real workplaces where there are multiple lead particulate sources which may be expected to give rise to emissions of different particle size distributions, we do not expect that there will be a perfect correlation between total lead exposure level and proportion of "large" lead particulate exposure.

OSHA concludes that most occupational particulate exposures include at most 15–20$\mu g/m^3$ material small enough to be absorbed at high efficiency, and that assumption C is reasonable.

Lundquist also argues that assumption C is a rather arbitrary set of assumptions which were used only because they had to be used to make the model fit the observed data. The theoretical and observational bases for the various parts of assumption C have been discussed at length and OSHA believes they are all reasonable.

OSHA has no doubt that Lundquist has developed equations that fit the Williams' data (even with the plastic workers included) and her own data better than the model and assumption C. But since neither of those studies included specific treatments of job tenures or particle size distribution, which the model does, those findings by no means invalidate the applicability of the model to the occupational situation.

Predictions of the health benefits vary, depending upon the nature and magnitude of the variability that can be expected to occur, in a population of workers around the predicted average blood lead level. The original CPA report's treatment of this variability has been extensively criticized.

St. Joe Lead maintained that blood lead levels are not normally distributed but log-normally distributed (Ex. 451B). Analyses of the Delco-Remy data seemed to indicate that, at least for these data, such was not the case, and that bood lead levels were more or less normally distributed about the mean. The long term variation about the mean between workers for all of the samples tested except one could not be distinguished from a normal distribution by a X^2 goodness-of-fit test, with any level of certainty. It is possible that distribution of blood lead levels in samples of workers with widely varying tenure and exposure histories might well be best described by a log-normal distribution of exposure levels; it would not imply that workers with similar tenure-exposure histories would have blood lead levels that were log-normally distributed.

St. Joe additionally claims that if short term individual variation in blood lead levels and measurement errors, as well as long term individual variability, are taken into account, the total variability of blood lead levels is greater than the CPA model estimates(σ 15 rather than 9.5 $\mu g/100$ g). In a brief analysis of the Delco-Remy data it found that while measurement error and short term variability do contribute to the observed variability, the total observed variability still was similar in magnitude to that used in the original CPA report as an upper bound estimate. As stated above, the long term variability in blood lead levels between individual standard deviations of about 5.46 $\mu g/100$ g, while the short term variability for the average worker had a standard deviation of about 7.3 $\mu g/100$ g, with measurement errors usually accounting for only a small portion of the short term variability.

Basically, OSHA agrees that there are many other sources of variability other than long term differences between individual response to lead exposure that account for large portions of the observed variability in blood lead levels. Aside from short and long term variation in air lead exposure, a number of other factors are at work. Among them are:

Individual differences in size, body composition (relative sizes of potential lead storage pools).

Individual differences in lead absorption (e.g., from short term fluctuations and long term differences in dietary habits, gastrointestinal function).

Individual differences in lead excretion (e.g., from short term fluctuations and long term differences in water and salt elimination, kidney function).

Individual differences in work demands producing differences in the volume of air breathed.

Differences in work habits (e.g., hygiene, smoking) affecting the relative levels of inhalation and noninhalation routes of lead exposure.

Miscellaneous environmental conditions affecting physiological processes (heat, humidity, other chemical and physical stressors).

Variation in the workweek (overtime, etc.).

The CPA adaptation of the Bernard model does not specifically take these forms of variation into account. It is likely, however, that the measure of variability used in the analysis (σ=9.5 g/100 g), coming as it does from observational data, contains contributions from all of those factors. The CPA model probably still presents an accurate picture of long term average response to air lead exposure.

ATTACHMENT B. PERMISSIBLE EXPOSURE LIMIT (PEL)

The final standard establishes a permissible exposure limit (PEL) of 50 $\mu g/m^3$ averaged over an 8-hour period. The decision to establish this PEL was based on consideration of the health effects associated with exposure to lead, feasibility issues and the correlation of airborne concentrations of lead with blood lead levels which have been associated with adverse effects and symptoms of lead exposure. In the proposed lead standard OSHA stated:

Establishing the permissible exposure limit requires first a determination of the blood lead levels associated with adverse effects and symptoms of lead exposure and then correlating these blood lead levels with airborne concentrations of lead. In setting a permissible exposure level, consideration must also be given to providing an adequate margin of safety. Thus, while it is clear that we cannot permit employees to be exposed to levels of toxic substances which are known to be harmful, prudence dictates that we consider setting maximum exposure

levels which provide a certain margin of safety below the known harmful levels. (Ex. 2, p. 45938.)

The issue of whether the PEL for lead should be lower than the proposed 100 $\mu g/m^3$ level was originally raised in the preamble to the proposed standard (Ex. 2, p. 45934). It is addressed as a "major" issue and incorporates the subissues of: (1) whether 100 $\mu g/m^3$ incorporates an appropriate margin of safety; (2) whether subclinical effects should be considered in establishing the PEL (implying that if that question is answered in the affirmative then the PEL would clearly be lower than 100 $\mu g/m^3$; i.e., as low as air lead levels corresponding to 30–40 μg Pb/100 g of blood); and (3) whether the PEL should be low enough to protect "susceptible groups," such as pregnant women.

These issues were reiterated in the public notice of hearing (Ex. 21, pp. 809–10) along with discussion of new studies on the effects of lead on male and female reproductive functions. The notice also referred to comments received from several groups claiming that 100 $\mu g/m^3$ was not protective of these reproductive functions. A reading of the cited comments and studies indicate that adequate protection could require an air level as low as 20–50 $\mu g/m^3$.

These issues were thoroughly debated by all parties to the rulemaking in oral testimony and written comments. This in itself should show that the public had actual notice of OSHA's intention to consider alternative PEL's, possibly as low as 20 $\mu g/m^3$, upon resolution of the subissues indicated above.

At the time the proposal was issued OSHA stated:

> Our present judgment is that in order to provide the appropriate margin of safety, as well as to provide significant protection against the effects, clinical or subclinical, and the mild symptoms which may occur at blood lead levels below 80 $\mu g/100$ g, it is necessary to set an airborne level which will limit blood lead levels to 60 $\mu g/100$ g. A maximum blood lead level of 60 $\mu g/100$ g corresponds to a mean blood lead level of about 40 $\mu g/100$ g. (Ex. 2, p. 45938.)

Based upon the extensive evidence of adverse health effects associated with exposure to lead in the record OSHA has determined that in order to provide on appropriate margin of safety as well as provide maximum protection against the effects of lead exposure the blood lead level of lead workers must be kept below 40 $\mu g/100$ g. For purposes of definition blood lead levels up to 40 $\mu g/100$ g will be considered "normal" although the record indicates the prevalance of health effects below 40 $\mu g/100$ g. This is particularly true for reproductive effects of both male and females who want to plan pregnancies. Blood lead levels above 40 are unacceptably elevated. In establishing 40 $\mu g/100$ g as the maximum blood lead level which the protection of employees and prudence permits OSHA is mindful the requirement of the Act that "no employee will suffer material impairment of health or functional capacity for the period of his working life." OSHA has concluded that maintenance of blood lead levels below 40 $\mu g/100$ g by engineering and work practice control of airborne lead will provide protection of workers throughout their working lifetimes. There is substantial evidence in the record which indicates that the blood lead level of both men and women who wish to plan pregnancies should be maintained at less than 30 $\mu g/100$ g during this period, and this evidence forms the basis for the action level of 30 $\mu g/m^3$ established in this final standard and for other provisions which will be discussed below in the medical surveillance, medical removal protection, and education and training sections.

OSHA recognizes that a PEL of 50 $\mu g/m^3$ will not achieve the goal of maintaining the blood lead levels in occupationally exposed workers below 40 $\mu g/100$ g. Based on the calculations using the CPA application of the Bernard model (SD 9.5 $\mu g/100$ g) OSHA predicts that at equilibrium, 0.5 percent of workers blood leads will exceed 60 mg/100 g, 5.5 percent will have PbB between 50–60 $\mu g/100$ g; 23.3 percent will be between 40–50 $\mu g/100$ g; and overall 29.3 percent of lead exposed workers will have PbB above 40 $\mu g/100$ g at any one time when compliance with 50 $\mu g/m^3$ PEL is achieved. This blood lead level distribution represents a marked improvement over the current levels in the industry and forms the basis for the calculation of incremental benefits obtained from the reduction of the PEL described later in this section.

In establishing 40 $\mu g/100$ g as a maximum desirable blood lead level, OSHA is conscious of the fact that the Act mandates that OSHA set a standard which meets the test of feasibility. OSHA has determined that 50 $\mu g/m^3$ represents the lowest level for which there exists record evidence on feasibility for primary and secondary smelting, SLI battery manufacturing, pigment manufacturing, and brass/bronze foundries. OSHA has concluded that the 50 $\mu g/m^3$ exposure limit is the level which properly balances the need to minimize deleterious health effects and meets the test of feasibility. Compliance with this level will provide a dramatic reduction in the number of workers whose blood lead levels are greater than 40 $\mu g/100$ g and will virtually eliminate all blood lead levels above 60 $\mu g/100$ g.

During the hearings there was testimony which argued for establishing a reasonable margin of safety (Tr. 1066–68, 1073, 1073–74, Ex. 335, p. 79). For example, Dr. Epstein testified that the AFL–CIO proposal of 40 $\mu g/m^3$ provided no more than a twofold margin of safety against clinical manifestations of lead toxicity. OSHA recognizes a more conservative PEL than 50 $\mu g/m^3$ would provide a greater margin of safety and reduce the extent of certain physiological changes whose significance is currently unknown. However, the constraint of the record on feasibility has limited the agency's ability to establish a margin of safety beyond that anticipated by the PEL of 50 $\mu g/m^3$.

A PEL of 50 $\mu g/m^3$ is achievable almost entirely through engineering and work practice controls, the preferable control strategy. The exposure limit is based upon what can be achieved by the affected industries taken as a whole using available technology or technology looming on the near horizon. OSHA has determined that the industries which will face the greatest difficulties in implementation of engineering controls will be primary and secondary smelters, pigment manufacturing, brass and bronze foundries and SLI battery manufacturers, and for this reason the PEL will be phased in with extended periods of time allotted for compliance (see methods of compliance) in these industries. The issue of feasibility is addressed in attachment D and will not be discussed here. Suffice it to say that OSHA has determined that the standard is feasible and that the PEL of 50 $\mu g/m^3$ represents the intersection between maximization of health benefits and feasibility.

The permissible exposure limit is based to a large part on the evidence of adverse health effects from exposure to lead previously described in the health effects section. OSHA has followed the logic of the proposal where first a determination of the blood lead levels associated with adverse effects and symptoms is made followed by correlation of these PbB levels with airborne concentrations of lead. The health effects section will be divided into three parts.

A. Clinical versus subclinical effects. A discussion of whether early health effects resulting from exposure to lead at low levels should be considered in establishing the PEL.

B. Health effects and the PEL. A discussion of the conclusions derived from evidence presented in the health effects section.

C. Clinical effects below 80 $\mu g/100$ g. A discussion of whether clinical effect occur at blood lead levels below 80 $\mu g/100$ g.

The remaining sections in the PEL which follow health effects are benefits of the PEL and alternatives to the PEL.

1. *Health effects.*

a. *Clinical versus subclinical effects.* In deciding upon the PEL it was necessary for OSHA to address this major issue raised in the proposal, namely:

> Whether subclinical effects of exposure should be considered in establishing a standard for occupational exposure to any substance, in this case lead. (Ex. 2, p. 45934.)

The proposal approached the latter issue as follows:

> Despite decades of research, the complex relationship between chemical exposures and human responses is still imperfectly understood. Incapacitating illness and death represent one extreme of a spectrum of responses, but other serious biological effects include physiological or metabolic changes that may be precursors or sentinels of disease. Boundaries between these categories overlap due to the variation of individual susceptibilities and exposures in the working population.
>
> It is customary to term "clinical" those biological changes that are known to directly indicate disease. Those changes of subtler significance which may not be symptoms of presently known or detectable disease are called subclinical. For example, as pointed out below, when lead in the blood exceeds 40 μg/100 g, will begin to excrete increased quantities of ALA into the urine, reflecting an enzyme inhibition caused by lead. If the amounts of ALA to reach a certain level in the urine, it could cause anemia and otherwise adversely affect the human body. However, it is not known with certainty at what level this enzyme inhibition becomes clinically important. What we do know is that such excretion is not physiologically desirable.
>
> As we point out below, the proposal is designed to provide a permissible exposure limit for the working population that should protect against known clinical effects of lead exposure. In addition, subclinical effects in workers would be substantially reduced. In any event, the question of both clinical and subclinical effects should be fully discussed in comments submitted, as well as at the hearing, if one is held, and might necessitate a different permissible exposure limit in the final standard than that proposed. (Ex. 2, p. 45935.)

It should be remembered that the proposed lead standard was drafted approximately 3 years ago when the data on the early stages of lead-induced disease was less well understood. Today OSHA believes that the original terms "clinical" and "subclinical" represent vast oversimplifications of a disease process and for this reason has avoided their use in this final standard. The use of the terms creates a false dichotomy which is neither accurate nor useful in describing adverse health consequences from exposure to lead. OSHA contends subclinical effects are in reality early stages in a continuum of disease. It is axiomatic that the chronic, irreversible stage is preceded at the opposite end of the disease by an early, relatively mild apparently reversible stage of disease. This earliest stage is characterized by varying subjective and/or objective symptoms that may not at first unduly alarm the victim or present a physician with clear-cut diagnosis. Nevertheless, this early developmental stage of disease is a pathological state and is potentially irreversible in some cases even at early stages. OSHA finds persuasive the arguments for adopting a lead regulation which protects workers from the early consequences of lead exposure. OSHA has concluded that reduction in motor nerve conduction velocities, elevation of enzyme inhibition products from heme impairment, decrease in hemoglobin levels, CNS symptoms, neurobehavioral effects, and reduced kidney function represent manifestations of a disease process and are, in themselves, important health effects which may be characterized as material impairment of health. (See health effects for an in depth discussion of effects.) Any standard for lead must prevent the onset of these changes since this will have the ultimate effect of preventing the development of more severe manifestations of disease later in life.

OSHA must promulgate a standard which prevents occupational disease resulting from both acute and prolonged or chronic exposure to lead in order to guard against the onset, progression, and severity of chronic degenerative diseases of aging workers. The degree of protection to be provided must extend over the full span of working life and must cover the more susceptible, as well as the more robust, members of the exposed group. Since the objective must be to limit exposures over an extended period of time to prevent future trouble, as well as immediate illness, the mere absence of illness or lack of clinical signs will not constitute sufficient evidence of adequate health protection. There should be no implications of immediate ill health in case the PEL is exceeded. The usual medical signs for disturbance, are wholly inadequate to provide employee protection. Simply to prevent overt manifestations of disease is not sufficient to prevent material impairment of health for the period of a working life since many of the disorders associated with lead are either irreversible (neurological disease and reproductive effects) or are only manifested when severe damage has occurred (kidney). Rather the PEL must seek to prevent the earliest indications or onset of disease and to the degree feasible establish a safety margin to allow for the remaining years of exposure.

Fortunately, the record indicates that there are now available many methods for detection and measurement of the degree of impairment caused by lead as expressed in terms of a variety of biochemical, physiological, and psychological disturbances. Some of these tests function at relatively gross levels which are immediately below morbidity. For example BUN S-creatinine, and hemoglobin serve as inadequate measures of ill health, whereas others reveal earlier changes that are highly sensitive, e.g. ALAD inhibition. OSHA recognizes that an uncritical assumption which interprets any demonstrated biological response as evidence of ill health or impending loss of health is fraught with uncertainty and borders on oversimplification of the disease process. For example, OSHA has not established a PEL which will prevent enzyme inhibition although it would be reasonable to do so in order to maximize the margin of safety. Rather the PEL is designed to prevent the effects of enzyme inhibition especially given the exponential changes which occurs above 40 μg/100 g. For example the National Academy of Sciences concluded:

> Arithmetic increases in blood lead content above approximately 40 μg/100 g of whole blood are correlated with a continuing exponential decrease in ALAD activity in hemolysates of peripheral blood, an exponential increase in urinary ALA excretion, and an exponential increase in "chelatable" lead. When all the available data are considered together, they are consistent with the hypothesis that the inhibition of ALAD activity in vivo in intact man becomes physiologically significant as blood lead content rises above approximately 40 μg/100 g of whole blood and that the partial inhibition observed is reflected by an increasing rate of excretion of its substrate (ALS) in urine. (Ex. 95, p. 171.)

To reiterate the policy stated above, prevention of disease implies protection at early, presumably reversible stages of disease as well as prevention of overt signs of illness. The need to approach lead on this basis was recognized by the National Academy of Sciences as early as 1972:

> Biochemical changes occur at blood lead concentrations well below those defining industrial toxicity and are perhaps the correlates of insidious changes. For example, interference with heme biosynthesis is the earliest evidence detected as the blood lead content rises above 40 μg/100 g of blood. Lane was pointed out that only the lead worker undergoing some toxic episode comes to medical attention. The worker who has become slowly and insidiously poisoned, who is "below par" but without acute manifestations, appears to be well, because he presents no overt health problems. However, he may be subject later to chronic nephritis and cerebral hemorrhage. As Hardy points out, "nonspecificity of sign and symptom, delayed diagnosable damage because of the body's incredible margin of safety, and more than one insult acting like lead or with lead require sophisticated attention to

the potential effect of low doses of lead-in much the same manner as low levels of ionizing radiation have been studied since the use of atomic energy for military purposes in 1945."

If the notion of "insidious poisoning" is valid, one might expect that workers exposed to lead concentrations below those which produce overt symptoms of toxicity would also undergo behavioral changes similar to the sensory, motor, and other alterations characteristic of frank lead poisoning, but to a lesser degree. However, no investigations of this have been reported. Nonetheless, a responsible company physician in sufficient contact with his workers is in a position to evaluate the early behavioral changes resulting from low-level poisoning. Given a familiarity with the base-line behavior of a worker, the physician can be alerted by the frequency of changes in some symptom categories that are otherwise difficult to interpret—irritability, lassitude, constipation, headaches, insomnia, abdominal cramps, and other diffuse complaints—as well as any increase in accident rates.

The symtoms of lead poisoning are, initially at least, rather vague; irritability and other mood changes predominate in the early stages, frank psychosis and encephalopathy later. The long biologic half-life results in so slow a buildup of toxic levels in the body that no connection may seem evident between the beginning of exposure to a chronically noxious environment and the development and progression of the symptoms of lead poisoning. (Ex. 95, p. 158-59.)

In 1972 there had been no investigations which had reported the behavioral changes described above in workers exposed to low levels of lead. The record in these proceedings demonstrates in numerous studies that the insidious poisoning does indeed occur in workers at low levels of exposure, and in order to prevent further development and progression of these signs and symptoms of lead poisoning, a conservative PEL must be established.

Dr. Bridbord of NIOSH developed an overview of the effects of lead which OSHA believes is an accurate representation of the disease process associated with exposure to lead and will repeat it in its entirety:

Mr. KUCHENBECKER. We've heard words like abnormal, damage, disease or subclinical disease, toxic poisoning, and even, as in the last discussion, we're talking about people dying with lead poisoning.

My concern is that, as a physician, could you give me your own feelings as to when you feel that we have sufficient dysfunction of those organ systems to be concerned in the sense of this is dysfunction, it's disease, it's illness, it's something that we have to control in the work environment, for the three systems, neurological, hemotological, and renal.

Dr. BRIDBORD. I think I'd like to present a conceptual framework first and then go back to each of the organ systems and give you some of my opinions as to how the various changes fit in. If one could envision a triangle for a moment and then draw horizontal lines within that triangle, draw one, two, three, four, so that we have five spaces. Have the triangle's lower base be parallel to the bottom of the paper, the first space there developed, the largest length on the bottom let's call normal. The next box, somewhat smaller, let's call physiological change of uncertain significance. The next line let's call pathophysiological change. In other words, something that we think is a change that is very closely associated with disease but may not, in and of itself, be called disease. The next box let's call morbidity which would represent fairly severe disease and finally, the tip of the triangle or the tip of the iceberg so to speak, would be mortality.

Now, within this, one point that I would like to emphasize is that there is no really sharp distinction. You are probably dealing with a continuum of a spectrum of response that one can find. One way to look at this triangle concept is that a group of people exposed to a harmful agent might express a spectrum of response. In other words, those most susceptible individuals might actually die. Those people still susceptible but not the most susceptible, might suffer severe disease. Conversely, there will be a broader number of people who may not be effected to the best of our knowledge.

I think in this particular triangle scheme, it would be important to keep a sharp line drawn between the concept of physiological change of uncertain significance and pathophysiological change. While there may not really be a clear cut distinction, conceptually there is. Because that's where we begin to see changes that are somewhat indicative of a precursor of possible real important health effect.

Let's take the hematologic system, blood forming elements. The change in ALAD activity might fall either into the normal or the physiologic change of uncertain significance. I personally would put it in the physiologic change of uncertain significance. Increase in zinc protoporphyrin or free erythrocyte protoporphyrin before it begins to increase exponentially indicating a backup in the metabolite in the body that quite likely is significant even though we may not fully understand, I would still put it in the physiologic change of uncertain significance.

Once the ZPP or the FEP begin to increase exponentially or the ALA in the urine begins to increase exponentially, at blood lead levels of about 40, although you could find some studies which suggest, particularly the ZPP and FEP might really start to go up somewhat under 40. I begin to view that as a pathophysiologic change. We may not completely undestand what it means yet but that it's not strictly speaking, normal and that's an indicator of a pathologic process or a disease process.

Morbidity in this case, I would define as anemia let's also call pathophysiologic changes going up toward the morbidity category as a decreased hemoglobin even though that decreased hemoglobin still might not put that person clearly in an abnormal clinical state but we still have some evidence that hemoglobin is going down. Once the hemoglobin actually was reduced below the normal limits of clinical acceptability, I would say that that would represent morbidity or some clear cut disease process.

I have already indicated that I felt that blood leads of about 40 begin to move into the pathophysiologic range. I think that there's fairly clear-cut evidence that as blood leads get to 60 and maybe a little below that, we begin to get into the range of morbidity. I think the range of morbidity in terms of blood lead levels and hemoglobin response is probably a pretty great range because, as I said, I don't think you could really move into the mortality category with the hemoglobin that clearly, just because of anemia.

I think the earliest sign that I would consider adverse, would be the decreased nerve conduction velocities in which case, in adults we begin to see this as blood lead levels rise about 50. One reason why I think that is clearly a pathophysiologic response or should be categorized as such, is that the ability of the nervous system to repair itself is fairly limited. That's not to say that there couldn't be any reversibility in some of these indicators but clearly there is very limited capacity to repair damage once such damage has occurred.

I think another point on the nerve conduction velocities is we're still measuring a fairly simple function and that to perform complex functions requires some integation of a number of circuits, maybe an electrical analogue might be a good example and that has to involve a certain amount of feedback and any decrease that one might find in a simple straight path I would think would tend to be accentuated to some degree as you get into more complex task and integration of many switching points, etc.

But I would clearly put the nerve conduction velocity in the pathophysiologic change. I think, in terms of dose response relationships in adults, I'm not sure we have a great deal of evidence to find where the pathophysiologic change clearly becomes a morbidity change. Again, it's probably a continuum. It's probably that triangle break is going to vary from individual to individual in terms of when the pathophysiologic change begins to be considered morbidity.

Certainly, once someone has had wrist drop, unequivocally that's morbidity and that's a very distinct disease entity. In the case of damage to the nervous system, it is quite well established that at fairly high levels of exposure, that can be the cause of death even in adults.

As far as the kidneys go, we've already heard evidence today and spoke somewhat yesterday of the fact that our clinical indicators of disease, early damage to the kidney, are not very good. At least the routine indicators that we have. As far as I'm concerned, when we have elevated B.U.N.'s, particularly when accompanied by an additional test of abnormal renal function, I would call that morbidity. Fairly severe disease. Damage of at least two-thirds of the kidney. I'm not sure that the data available allow on the precisely defined, exactly what blood level it does or doesn't occur. I think the chronicity of exposure is probably as important as a specific blood level.

My personal opinion, and I think I stated this yesterday, is that a blood lead level of 60 and a chronic exposure basis I don't believe provide a margin of safety to protect against this severe disease. I personally would say a blood lead level of 40 would be more appropriate. (Tr. 1796-1802.)

During his testimony Dr. Teitelbaum echoed many of Dr. Bridbord's conclusions:

Physicians have had little difficulty identifying advanced lead intoxication in any of these societies. The problem always has been how to recognize early lead intoxication at a time when lead disease was still reversible. If any single question is common to all physicians who have observed and treated lead intoxication throughout history, it

has been how to prevent the occurrence of lead intoxication and how could one recognize it early enough to prevent death or pemanent injury when it occurred.

On this basis, it is a national disgrace that in 1977, when the tools for recognition of early lead intoxication or asymptomatic lead effect exists, and when the engineering controls for the prevention of lead intoxication exist, that we should still be in a quandry as whether it is, possible to prevent lead poisoning, to make an early diagnosis of lead effect of lead poisoning and to treat those persons who have suffered lead intoxication.

In an era in which routine monitoring of esoteric industrial toxins in parts per billion is a daily reality, and at a time when the prevention of exceptionally rare industrial disease is commonplace, it is unacceptable that lead poisoning, a well-recognized, well-described, entirely preventable disease continues to affect American workers.

I believe that all of the activities necessary to erradicate this industial disease can be accomplished on a cost-effective basis.

Our present technologic sophistication permits us to recognize two categories of patients who have abnormal lead absorption. One group has overt lead intoxication, lead poisoning. These patients have absorbed so much lead that a clinical diagnosis of lead intoxication can be made on the basis of history and physical examination alone. The patient's illness requires laboratory confirmation, not toxic diagnosis.

Such patients have long since passed the point at which preventive medicine is an issue. The remaining issue in their cases is one of therapeutic intervention for lead poisoning. Certainly, we cannot tolerate the promulgation of abnormally high lead accumulation that a patient had to develop overt lead intoxication that would be obvious to any physician on a clinical basis before any action was taken to protect the worker. Rather, we must focus our attention on prevention of lead poisoning. The standard must focus on the more important group of individuals with excessive lead absorption, those who have no disease, but have lead effect demonstrated by metabolic abnormalities which are the stalking horses of future lead intoxication.

This group of patients is not lead poisoned in the traditional sense. No physician could, on the clinical basis alone, make the diagnosis of lead intoxication in them. Without sophisticated laboratory studies, these individuals would not be recognized as poisoned because they have no obvious clinical findings. However, they have obvious laboratory evidence of excessive lead absorption. They show evidence of interference with normal red blood cell manufacture and interference with normal nerve conduction time, and interference with other enzyme systems which are intimately involved with the maintenance of human homeostasis. If these abnormalities are ignored in a planned attempt to wait for overt disease, surely no preventive medicine is being practiced. These individuals with lead effect, but no lead disease by the classic definition of lead intoxication, are the group of workers who the standard must identify if the more obvious disease is prevented. On the basis of all present knowledge, these workers still have reversible findings; they are not yet seriously intoxicated and there is every reason to hope for their complete recovery.

The proposed standard falls short of the absolute prevention of lead effects, even in the context of our present imperfect knowledge of the disease. To achieve this end, no lead exposure would be permitted. However, as a realistic concession to human frailty, it permits exposures of 100 micrograms per meter cubed, an air level at which levels of body lead and metabolic markers of lead absorption are effected in some workers to a degree which is not subtle. In fact, as you will see from a series of cases which I will shortly present, it is possible for an individual to have quite severe lead poisoning, far beyond asymptomatic lead effect with the levels of blood and urine lead or urinary delta ALA which would be required by the proposed standard in order to activate monitoring and medical surveillance. (Tr. 374-378.)

These comments on blood lead distribution are consistent with the recommended guidelines for PbB based on health criteria of the Second International Workshop on Permissible Levels for Occupational Exposure to Inorganic Lead, 1976.

It was agreed that for male workers individual blood leads should not exceed 60 μg/100 ml in the light of present knowledge available to this group. It is, however, desirable to reduce individual exposure below this level, taking into account the effects on the hematopoietic system at concentrations above 45 to 50 μg/100 ml and on nerve conduction velocity at concentrations between 50 and 60 μg/100 ml. (Ex. 262.)

The health effects section of this final standard described the adverse effects associated with lead exposure. It is apparent from the record that material impairment to various organ systems occurs at lower blood lead levels than were previously thought to be harmful. The vast majority of the physicians who testified supported the view that blood lead levels should be maintained at or below 40 μg/100 g in order to protect against the onset of early manifestations of disease previously described as subclinical effects.

Testimony by Teitelbaum and Bridbord have already been cited. Similar testimony was presented by other physicians at the hearings. Dr. Lilis (Tr. 2700-01), Dr. Needleman (Tr. 1085-86; 1106-07); Dr. Epstein (Tr. 1051-52, 1058-65, 1067-68), 1072, 1073-74, 1104-05); Dr. Lancranjan (Tr. 1771), Dr. Wolfe (Tr. 4140), and Dr. Piomelli (Tr. 467). In addition to testimony by numerous scientists and physicians OSHA has given significant weight to the submissions of noted scientific bodies such as the National Academy of Sciences (Ex. 95, Ex. 86M) and other Government agencies, EPA (FEIS (92)) and the Center for Disease Control (Ex. 2, (15)).

The subject of subclinical effects was indeed discussed at great length during the rulemaking hearings and was the source of some controversy. The lead industries' arguments were summarized in the Post-hearing brief of the LIA:

> Much, if not most, of the record pertaining to the medical issues raised by the proposed standard relates to the problem of trying to determine "the point at which subclinical changes become sufficiently serious to represent a threat to health * * *." (Exhibit 2, at 45935.) "Subclinical" effects involve biochemical and physiological parameters which occur at blood lead levels lower than those usually associated with overt "clinical effects." Ibid. As Hammond explained when testifying for OSHA, "subclinical" or "subcritical" is the usual terminology * * * for an effect that does not appear to have an effect on health, per se. (Hammond 300-01.)
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> Before considering the significance of the various biophysical changes which are said to occur at different blood-lead concentrations, two preliminary observations are in order.
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> First, "subclinical effects" almost by definition are outside the scope of the Secretary's authority, since he is permitted to set standards only with respect to "material impairment of health or functional capacity." 29 U.S.C. § 655(b)(5). He is not authorized, as Senator Dominick pointed out, "to eliminate all risks to safety and health." And, as Dr. Williams explained, "if 'clinical' directly indicates disease, 'subclinical' can only mean 'does not directly indicate disease', and circumlocution should not have it otherwise." (Ex. 3 (65); Ex. 234 (8).)
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> Second, it is important to remember that although exposure to lead may cause biological changes, not every biological change which occurs in response to an external stimulus is harmful. Most of those who believe that the biological action level proposed by OSHA is too high proceed on the assumption that virtually any detectable change is automatically deleterious to health and is therefore intolerable (e.g., Piomelli 466-67; Seppalainen 118; Lilis 2701). That assumption is incorrect. Our bodies respond to innumerable stimuli— temperature, Light, physical substances, exertion, and a myraid of others. The fact that a biological change has occurred does not necessarily signal physical injury or even the threat of injury. This is true despite the fact that the biological change is characterized as a "subclinical effect," for as Dr. Bridbord of NIOSH noted, no one "has all of the answers to at what point (subclinical changes) * * * become significant." (Bridbord 1454.) The question, therefore, is not whether subclinical effects result from lead exposure, but rather whether those effects have health implications which justify a particular exposure standard. As indicated by the analyses below, LIA submits that they do not. (Ex. 3353, p. 20-22.)

The Lead Industries Association has argued that workers will not suffer material impairment of health if blood leads are below 80 μg/100 g. In setting forth their arguments they quote from Senator Dominick during the original debate of the OSHA Act in the Senate in October 1970 in order to set a legal and statutory background that OSHA must consider in determining the final standard. In setting standards regulating toxic materials or harmful agents, the Secretary is under

the stricture to adequately assure "to the extent feasible and on the basis of the best available evidence, that no employee will suffer material impairment of health or functional capacity" 29 U.S.C. 3665 (6)(5). As first proposed, the statute would have read "no employee will suffer any impairment." (Emphasis in original.) During the debate on the bill in the Senate in October 1970, Senator Dominick proposed amendment No. 1054 to effectuate the substitution of the word "material" in lieu of "any" in the original draft. In explaining the reason for the modification, he stated that:

> This requirement is inherently confusing and unrealistic. It could be read to require the Secretary to ban all occupations in which there remains *some* risk of injury, impaired health, or life expectancy. In the case of all occupations, it will be impossible to eliminate all risks to safety and health. Thus, the present criteria could, if literally applied, close every business in this nation. *Ibid.* (Emphasis in original.)

When the amendment reached the floor, Senator Dominick elaborated on his concerns and the need for the amendment.

> No job can be rendered perfectly safe, and no employee can be made perfectly secure from injury. Hence, it is impossible to fashion criteria which would assure these unattainable goals * * *. It is unrealistic to attempt, as this section apparently does, to establish a utopia free from any hazards. Absolute safety is an impossibility and it will only create confusion in the administration of this act for the Congress to set clearly unattainable goals * * *. The difficulty of the language I am dealing with here and that I am trying to delete is the requirement, that the Secretary, in establishing standards, must assure that there will not be any risk at all. Legislative History, at 480–81.

The statute, which in its original form would have required the Secretary to establish standards to assure that there would not be any impairment at all, whether or not that impairment was due to the employee's negligence or conditions within the employer's control, was deemed to be an unenforceable requirement.

The statute in its final state does not seek to require standards that regulate risks beyond the employer's control such as employee negligence or outside conditions. Rather, the statute's purpose in relation to toxic or physical agents as established by Senator Dominick was to assure the provision of "such steps as are feasible and practical to provide an atmosphere within which a person's health and safety would not be affected." (Legislative History, at 502.) Senator Williams emphasized that that type of protection was due all employees, including those who might have continuous exposure to the hazard for the full period of their working life. (Legislative History, at 503.) Senator Dominick's comments in the legislative history fully support this reasoning.

> * * * The Secretary has got to use his best efforts to promulgate the best available standards * * * so that we can get at something which might not be toxic now if he works in it a short time but if he works in it the rest of his life might be very dangerous; and we want to make sure that such things are taken into consideration in establishing the standard. (Legislative History, at 503.)

Support for OSHA's requirement that a worker be protected from long term health effects from exposure to toxic substances throughout his working life is also found elsewhere in the legislative history. Senator Williams, in discussing the need for standards dealing with warning labels for toxic materials, stated that workers are often unaware of their exposure to harmful agents or toxic materials. In some cases, consequences of overexposure may be severe and immediate, in other cases, effects may be delayed or latent. Williams affirmed that "In all these situations (whether the consequences of overexposure be immediate or latent) it is important that the worker be adequately protected against excessive exposure * * *" (Legislative History, at 415.) Senator Williams intended that protection be provided to workers both before and after experiencing the overt effects of overexposure.

Further support can be gained from language of the Act which incorporates congressional findings and the general purposes of the Act. Section 2(h)(7) affirms that it is part of the congressional policy to provide medical criteria which will assure that "no employee will suffer *diminished* health * * * as a result of his work experience." (Emphasis added.) Section 2(b)(6) grants the Secretary the authority to explore ways to discover latent disease acknowledging that the problems of occupational health standards are often quite different from those involved in occupational safety. Both these sections indicate congressional recognition of the need to protect the worker during all stages of the development of an occupational disease.

The Secretary has been vested with authority to establish standards which protect the employee from material impairment. In the promulgation of any standard, however, the Secretary may neither exceed that authority nor may he base the standard on arbitrary assumptions. In promulgating the lead standard, the Agency has acted within the scope of his authority.

In order to carry out the congressional mandate that no employee suffer material impairment, the standard accords a margin of safety. The legislative history also justifies promulgation of a standard to prevent against the long-term effects of lead. Senator Williams explained that the statute requires the Secretary to protect both those employees who show severe and immediate effects and those in whom the effects may be delayed or latent. (Legislative History at 481.) By promulgating the present standard, the Secretary has assured that both groups will be afforded protection.

The PEL OSHA has established to effectuate the protection of workers mandated by the statute was chosen after careful consideration of the best available evidence in the record and the latest scientific data available in the lead field as required by the statute. (29 U.S.C. § 655(b)(5).) A full analysis of that evidence is given elsewhere in the preamble demonstrating that long-term blood lead levels in excess of 40 μg/100 g must be avoided. OSHA therefore disagrees with the arguments set forth by LIA in which they claim it was not congressional intent to include "subclinical" effects in the development of standards.

b. *Health effects and the PEL.* The record demonstrates that lead has profoundly adverse effects on the health of workers in the lead industry. Inhalation, the most important source of lead intake, and ingestion results in damage to the nervous, urinary, and reproductive systems and inhibits synthesis of the molecule, heme, which is responsible for oxygen transport in living systems.

The signs and symptoms of severe lead intoxiction which occur at blood lead levels of 80 μg/100 g and above are well documented. The symptoms of severe lead intoxication are known from studies carried out many years ago and include loss of appetite, metallic taste in the mouth, constipation, nausea, pallor, excessive tiredness, weakness, insomnia, headache, nervous irritability, muscle and joint pains, fine tremors, numbness, dizziness, hyperactivity, and colic.

Damage to the central nervous system in general and the brain (encephalopathy) in particular is the most severe clincial form of lead intoxication. The most severe often fatal form of encephalopathy may be preceded by vomiting, apathy progressing to drowsiness and stupor, poor memory, restlessness, irritability, tremor, and convulsions. It may arise precipitously with the onset of intractable seizures, followed by coma, cardiorespiratroy arrest and death. There is a tendency toward the occurrence of weakness of extensor muscle groups; i.e. motor impairment. This weakness may progress to palsy, often observed as a characteristic "wrist drop" of "foot drop" and is a manifestion of a disease to the peripheral nervous system (peripheral

neuropathy). Lead intoxication also results in kidney damage with few, if any, symptoms appearing until extensive and most likely permanent kidney damage has occurred. NIOSH testified that:

> Of considerable concern are the effects resulting from long-term lead exposure. there is evidence that prolonged exposure can increase the risk of nephritis, mental deficiency, premature aging, and high blood pressure. (Ex. 84, p. 6.)

Exposure to lead results in decreased libido, impotence, and sterility in men and decreased fertility, abnormal menstrual and ovarian cycles in women. The course of pregnancy is adversely affected by exposure to lead. There is conclusive evidence for miscarriage and stillbirth in women who were exposed to lead or whose husbands were exposed. Children born of parents either of whom were exposed to lead are more likely to have birth defects, mental retardation, behavioral disorders, or die during the first year.

During the past 10 years there have been many new observations and research on the health effects of lead at levels heretofore thought to be inconsequential. The main research topics which have been addressed are early biochemical changes in the synthesis of the respiratory pigment heme; and early effects on the nervous system including behavioral and peripheral nerve effects. Studies on the involvement of lead in kidney disease and effects on reproductive capacity of male and female workers, and effects on the fetus have also been conducted as have studies on and the relation between exposure to lead in air and resulting blood lead concentration.

The disease process associated with lead exposure can be subdivided according to Bridbord (Tr. 1976-02) into five stages: Normal, physiological change of uncertain significance, pathophysiological change, overt symptons (morbidity), and mortality. Within this process there is no sharp distinction, but rather there is a continuum of effects. Boundaries between categories overlap due to the variation of individual susceptibilities and exposures in the working population. OSHA believes that the standard adopted must prevent pathophysiologic changes from exposure to lead. Pathophysiologic changes indicate the occurence of important health effects. The basis for this decision is twofold—first, pathophysiologic changes are early stages in the disease process which would grow worse with continued exposure and which may include early effects which even at early stages may be irreversible, and therefore represent material impairment themselves. Second, prevention of pathophysiologic changes will prevent the onset of the more serious, irreversible and debilitating manifestations of disease.

The evidence in this record demonstrates that prevention of adverse health effects from exposure to lead throughout a working lifetime requires that blood lead levels be maintained at or below 40 μg/100 g. Feasibility constraints prevent OSHA from establishing a standard which would eliminate all physiological changes, reproductive effects or mild signs and symptoms but the agency believes the vast majority of workers will be protected by it. The remainder to this summary will address the health effects evidence in each system: heme synthesis inhibition, and damage to the nervous, urinary, and reproductive systems.

(1) *Heme synthesis inhibition.* Heme is a complex molecule which has two functions in the body. First, heme is a constituent of hemoglobin, the protein present in red blood cells. A primary function of hemoglobin is to transport oxygen to the tissues. Interference with the formation of heme, if sufficient, results in decreased hemoglobin and ultimately anemia. Anemia is characterized by weakness, pallor and fatigability as a result of decreased oxygen carrying capacity in the blood.

Heme is also a constituent of another group of extremely important proteins, the cytochromes, which are present in every cell of the body. The function of heme in the cytochromes is to allow the cell to utilize oxygen. Heme may therefore be described as the "respiratory pigment" for the entire body. Interference with heme formation leads to interference in the respiration of every cell in the body. This is the most important effect of heme synthesis impairment. Piomelli has suggested that heme impairment in the cells would lead to a condition in each cell similar to that which would occur if the lungs of an individual did not function well. The central nervous system is particularly sensitive to the lack of oxygen and neurological damage could conceivably occur prior to anemia as a result of heme synthesis impairment in the brain. For example, Piomelli testified that: "It is very well known that the human being cannot stop breathing for more than 2 or 3 minutes without developing irreversible brain damage." (Tr. 460.) This effect would be expected to occur from impaired respiration resulting from impaired heme synthesis. In other words, heme synthesis impairment could potentially affect every cell through reduced respiration.

The effects of lead exposure on heme synthesis have been studied extensively by the scientific community. Nevertheless, there is considerable debate over certain issues concerning the health effects of lead on this system. The Agency found three major issues particularly important in evaluating the health effects of lead in reference to heme synthesis.

(1) What is the meaning of the enzyme inhibition and physiological changes known to occur in this system at low lead levels, and should these effects be considered as per se impairment of health in the establishment of a permissible level of worker exposure to lead. (2) At what PbB level does a lowering of hemoglobin leading to anemia begin to occur? (3) To what extent are lead effects on heme synthesis in the blood forming system indicative of changes in heme synthesis in other tissues?

The earliest demonstrated effect of lead involves its ability to inhibit the formation of heme. Scientific evidence has established that lead inhibits at least two enzymes of the heme synthesis pathway at very low PbB levels. Inhibition of delta aminolevulinic acid dehydrogenase (ALAD), an enzyme responsible for the synthesis of a precursor to heme, is observed at PbB levels below 20 μg/100 g. At a PbB level of 40 μg/100 g more than 20 percent of the population would have 70 percent inhibition of ALA-D. In the human body when an enzyme system is inhibited two effects are often seen: First, the molecule upon which the enzyme would act accumulates because it cannot undergo chemical reaction to produce the desired product and second, the desired product therefore decreases. Significant urinary excretion of the products of ALAD inhibition, such as delta aminolevulinic acid (ALA), occurs at this PbB level; 11 percent of adult males are excreting more than 10 mg/l.

The buildup of another product of impairment indicating inhibition of another enzyme ferrochelatase also occurs at low PbB levels. At a PbB level of 50 μg/100 g a larger proportion of the population would suffer these effects and the effects would be more extreme. At a PbB level of 50 μg/100 g, 70 percent of the population would have 70 percent inhibition of ALA-D, 37 percent would have ALA-U values larger than 10 mg/l and 80 percent of men and 100 percent of women would have increased free erythrocyte protoporphyrin (FEP), which is the product at inhibition of ferrochelatase. (Ex. 294 E). Industry representatives augued that these effects are the manifestation of the body attempting to maintain a stable internal environment to lead. OSHA believes that it is inappropriate and simplistic to describe these changes as biochemical adjustments. The depression of heme synthesis in all cells of the body is an effect of potentially far reaching proportion and prevention of enzyme effects is the key to prevention of more

serious clinical effects of lead toxicity, which become more obvious as the exposure continues. These measurable effects are a direct result of lead exposure and are considered by the agency to indicate the occurrence of disruptions of a fundamental and vital subcellular process, heme synthesis. These processes are not only essential to the process of hemoglobin synthesis, they are also vital to the function of all cells since heme is ubiquitous in the human.

OSHA believes the preponderance of the evidence indicates a progression of health effects of lead exposure starting with inhibition of enzymes, continuing through effects indicating measurable disruption of subcellular processes, such as the buildup of the products of impaired heme synthesis and eventually developing into the overt symptoms of lead poisioning as manifested in disorders in the nervous, renal, and blood forming systems. Biological variability among individuals will alter the PbB level at which a particular person will move through each stage in this disease continuum. Therefore, at each higher PbB level a greater proportion of the population will manifest each given effect. Given this understanding of the progressive stages of lead effects, OSHA has concluded that enzyme effects indicative of the disruption of heme synthesis are early stages of a disease process which eventually results in the clinical symptoms of lead poisoning. OSHA agrees with Piomelli who concluded "It is the responsibility of preventive medicine to detect those alternations (in heme synthesis) which may proceed frank symptomatology and to prevent the occurrence of these symptoms" (Tr. 456).

OSHA believes that health is not limited to the narrow definition of "absence of clinical symptoms." The early steps of the progression to disease cannot be considered as an attempt by the body to merely adjust and stabilize the internal environment to exposure to lead: they are early indications of significant physiological disruption. Whether or not the effects have proceeded to the later stages of clinical disease, disruption of these processes over a working lifetime must be considered as material impairment of health. As was previously discussed, at a PbB level of 40 μg/100 g and above, a significant proportion of the population would manifest extensive inhibition of ALA-D, elevations of ALA-U and of protoporphyrin levels. The agency believes that PbB levels should ideally be kept below 40 μg/100 g to minimize these effects.

Anemia is one of the established symptoms of lead poisoning. The symptoms of anemia are weakness, tiredness, pallor, waxy sallow complexion, headache, irritability, and other symptoms characteristic of the increased load on the cardiac system. The clinical symptoms of anemia due to lead are often indistinguishable from those of chronic anemias with a variety of other causes. Anemia due to lead is often seen in association with acute abdominal colic. The occurrence of anemia, as a result of lead exposure, is known to occur above PbB levels of 80 μg/100 g. The occurrence of this symptom at PbB levels below 80 was debated during the hearings.

OSHA believes that the debate concerning the occurrence of this symptom can only be comprehended within the context of an understanding of the full disease process which eventually results in anemia. The evidence concerning the mechanisms of this disease process indicates that the effect of lead on the hematopoietic system is subtle and complex. In evaluating the disease mechanisms of anemia, it was found that lead is an insidious poison which attacks, not one, but many of the physiological processes within the cell.

Because anemia is the result of a complex of different lead effects, there is considerable room for individual variability in the PbB level at which anemia will occur. Hemoglobin level is a continuous variable which may cause individuals to have a problem to a greater or lesser degree at any particular blood lead level. Anemia should be viewed as a late step in a complicated progression of lead effects.

Since anemia is a consequence of lowered hemoglobin (the protein in red cells responsible for respiration) OSHA has carefully analyzed those studies which reported reduced hemoglobin. Studies have associated PbB levels as low as 50 μg/100 g with lowered hemoglobin (Hb) levels (Ex. 6(37); 146-A; 5-9). In particular, Tola's study, which showed a lowering of Hb over time during lead exposure of 50 μg/100 g, is considered by OSHA as an example of lead affecting Hb levels at this low PbB range. In addition studies by the Mt. Sinai group (Ex. 24 (10), and Wolfe (Ex. 146(A)) also demonstrated anemia in lead exposed workers.

Based on evidence that indicates decreases in Hb levels with bloods leads above 50 μg/100 g, OSHA has concluded that a lowering of Hb level to a measurable degree will occur at PbB levels as low as 50 μg/100 g. The degree to which Hb is lowered at this PbB range may occur undetected since symptoms may be mild and are not likely to be so large as to require treatment for anemia. However, these changes must not be evaluated only as short-term effects alone but rather as changes that would occur over prolonged times. This implies that with reduced hemoglobin in an asymptomatic or mildly symptomatic individual there is a lifetime alteration in the oxygen carrying capacity of the blood, in the blood viscosity and in particular, the cardiac work load, which is distinct from the frank symptoms of anemia but far more insidious and which may be deleterious to the worker over the long term. Lastly, the data cited does support the view that lead induced anemia is clinically apparent at PbB's as low as 50 μg/100 g.

In evaluating the effects of lead on heme synthesis, Piomelli suggested that hematapoetic effects such as anemia are not the most significant clinical effect of heme synthesis disruption * * * a much more important fact is that the alteration of the mechanism of heme synthesis reflects the general toxicity of lead in the entire body. (Tr. 458.)

Evidence indicates that there is disruption of heme synthesis in other tissues of the body besides blood, and that this disruption results in alteration of the oxygen transport into the cells of the body. Enzyme (ALA-D) inhibition due to lead exposure had been found in the liver at PbB levels below 40 μg/100 g (Ex. 5(22)). Electron microscope studies have revealed mitochondrial changes. The mitochondria is that portion of the cell responsible for extracting nutrients and oxygen and in turn providing the energy needed elsewhere in the cell for performing cellular functions, associated with lead exposure such as lead granules in rat liver mitochondria (Tr. 459, ref. Walton in Nature 243, 1973) and broken distorted mitochondria in the renal cells of a lead-exposed workers. (Cramer et al Brit. J. Ind. Med. 1974). Some of these studies related changes in heme synthesis in the blood forming to changes in other tissues. Secchi (Ex. 5(22)) found a direct correlation of levels of ALA-D inhibition in the blood and in the liver. Millar found parallel decreases in ALA-D activity in the blood and in the brain at PbB levels above 30. (Ex. 23(68), ref. Millar.) This evidence supports Piomelli's suggestions that changes in heme synthesis in the blood forming (hematopoietic) system reflect changes that occur in other tissues. The work of Fishcbein et al. related levels of products of enzyme inhibition, a measure of heme synthesis disruption in the hematopoietic system, to various signs and symptoms of lead exposure including central nervous system symptoms, muscle and joint pain, weight loss, and lead colic at blood lead levels well below 80 μg/100 ml (mean PbB was approximately 60 μg/100 g) (Ex. 105D). Fishcbein also noted anemia in 37 percent of these same workers.

While the evidence relating lead effects of heme synthesis to symptoms throughout the body is not complete, the evidence is extensive enough and the issue is important enough to warrant very serious consideration with reference to the establishment of the standard. OSHA believes this evidence demonstrates that one early stage of lead disease in various tissues is the disruption of heme synthesis and that these effects in other lead-sensitive tissues parallel the measurable effects of heme synthesis disruption in the hematopoietic system and occur at comparably low PbB levels (below 40 μg/100 g). The heme effect is clearly not the only mechanism by which lead exerts its toxicolgical effect but it is one mechanism which we have substantial understanding of, can measure, and therefore must utilize in an effort to prevent the more severe symptoms in the individual.

In reference to the hematopoietic system, OSHA believes that the effects of lead are a complex progression from various biochemical changes through to the onset of clinical symptoms. At increasingly higher PbB levels an increasing proportion of the population will suffer more extreme effects. At a PbB level of 40 μg/100 g and above, a sizable proportion of the population would show measurable effects of the disruption of heme synthesis. A comparable degree of disruption of heme synthesis impairment would most likely occur in other cells in the body.

Piomelli gave an excellent summary of the importance of lead effects on heme synthesis stating:

> It is my understanding that regulations have the purpose of preventing material impairment of health. Alterations in heme synthesis do not produce subjective evidence of impairment of health, unless they reach the extreme depression in severe lead intoxication, when marked anemia occurs and the individual feels weak. However, it is not any longer possible to restrict the concept of health to the individuals subjective lack of feeling adverse effects. This is because we know that individuals may get adjusted to suboptimal health, if changes occur slowly enough and also because we now have the ability to detect functional impairments by appropriate tests, much before the individual can perceive any adverse effect. In fact, it is the responsibility of preventive medicine to detect those alterations which may precede frank symptomatology, and to prevent its occurrence. The alterations in heme synthesis caused by lead fulfill, in my opinion, the criteria for material adverse effects on health and can be used to forecast further damage. The depression of heme synthesis in all cells of the body is an effect of far-reaching proportion and it is the key to the multiple clinical effects of lead toxicity, which become obvious as the exposure continues. (Ex. 57, p. 21.)

This does not in any way suggest that the lead effect on heme is the only mechanism of lead disease, but it does suggest that this effect is at least one of the important mechanisms in lead disease. An understanding of this spectrum of effects from subcellular to clinical symptoms is relevant not only to the occurrence of anemia but will also be the expected pattern in lead induced neurological and renal disease.

OSHA believes that there is evidence demonstrating the impairment of heme synthesis and mitochondrial disruption in tissues throughout the body, and that these effects are the early stages of lead disease in these various tissues. The disruption of heme synthesis measured at low PbB levels is not only a measure of an early hematopoietic effect, it is also a measure which indicates early disease in other tissues. The Agency believes that such a pervasive physiological disruption must be considered as a material impairment of health and must be prevented. PbB levels greater than 40 μg/100 g should, therefore, be prevented to the extent feasible.

(2) *Neurological effects.* There is extensive evidence accumulated in both adults and children which indicates that toxic effects of lead have both central and peripheral nervous system manifestations. The effects of lead on the nervous system range from acute intoxication coma, cardiorespiratory arrest and fatal brain damage to mild symptoms, subtle behavioral and electrophysiologic changes associated with lower level exposures. Although the severe effects of lead have been known for some time, only in the last several years has evidence accumulated which demonstrates neurologic damage at low blood lead levels. All of this data reinforces a disturbing clinical impression that nervous system damage from increased lead absorption occurs early in a workers tenure, at low blood lead levels and is only partially reversible if at all. It is now understood that the location and degree of neurological damage depends on dose and duration of exposure.

The record in this rulemaking demonstrated that damage occurs in both the central and peripheral nervous systems at blood lead levels lower than prviously recognized. In particular, Lillis et al. (Ex. 24, (10)) has demonstrated central nervous system symptoms (tiredness, fatique, nervousness, sleeplessness or somnolence, or anxiety) in 56 percent of workers with blood lead levels below 80 μg/100 ml. The mean blood lead level was approximately 60 μg/100 ml. This same study reported symptoms of muscle and joint pain and/or soreness in 39 percent of the workers. It is extremely important to note that many of these subjects had been exposed less than a year. They also were able to demonstrate behavioral changes which were correlated with enzyme inhibition products from heme synthesis. Given this data, the authors cautioned that blood lead levels should not be allowed to exceed 60 μg/100 ml and should be maintained around 40 μg/100 g. Lillis testified that above 60 μg/100 g "one may expect florid lead poisoning, full blown lead poisoning." (Tr. 2700.) She proceeded to state: "Since ZPP starts to go up at around levels of 40 or 45, that means that at those levels you already find something going wrong in the body." (Tr. 2702.) Repko has carried out behavioral tests and demonstrated adverse effects in visual reaction time, as well as deficits in hearing among workers having a mean blood lead level of 46 μg/100 g. Valciukas et al. and Haenninen et. al. have also demonstrated impaired psychological performance among workers with low exposure to lead. Haenninen's work is particularly significant insofar as no single blood lead concentration had ever exceeded 70 μg/100 ml.

Based on the rulemaking record, OSHA has concluded that the earliest stages of lead-induced central nervous system disease first manifest themselves in the form of behavioral disorders and CNS symptoms. These disorders have been documented in numerous sound scientific studies and these behavioral disorders have been confirmed in workers whose blood lead levels are below 80 μg/100 g. Given the severity and potential nonreversibility of central nervous system disease, OSHA must pursue a conservative course of action. OSHA concludes that a blood lead level of 40 μg/100 g must be considered to be a threshold level for behavioral changes and mild CNS symptoms in adults, and to protect against long-term neurological effects, blood levels should never exceed 60 μg/100 g.

Some of the most extensive evidence in the rulemaking record is the data presented which confirms the existence of the early stages of lead induced damage to the peripheral nervous system in workers exposed to lead levels below 70 μg/100 g. Damage to the peripheral nervous system is named peripheral neuropathy and the distinguishing feature of it is the predominance of motor involvement as opposed to sensory damage. Three forms are noted. In the first, patients may complain of very severe pain and tenderness in the trunk muscles, as well as pain in the muscles of the extremity. As the pain and tenderness subside, weakness may emerge, with very slow recovery over the ensuing several months. In the second, more common form of peripheral neuropahthy due to lead poisoning, the neuropathy is described as painless, peripheral weakness occurring either after termination of excessive exposure or after long, moderately in-

creased exposure. This suggests that neuropathy of sufficient severity may cause irreversible impairment of peripheral nerve function.

The third form is seen in subjects with no obvious clinical signs of lead poisoning and is manifested by a slowing of motor nerve conduction velocity. The latter effects represent the earliest sign of neurological disease of the peripheral nerves. OSHA believes prevention of this stage is necessary to prevent further development of the disease and its associated forms which are likely to be irreversible.

The work of Catton, Oh, Landigran, Feldman, Behse, Mostafa et al., Gerald et al., Guadriglic et al., Araki, W. R. Lee, Repko, Lilis, Fischbein et al., and Seppalainen all demonstrate statistically significant loss of motor nerve conduction velocity in lead-exposed workers. Seppalainen was able to determine a dose-response relationship for the slowing of NCV compared with blood lead levels. It is apparent that slowing occurs in workers whose PbB levels are 50 μg/100 g and above but, whether there are effects as low as 40 μg/100 g is, as yet, undetermined. The 38 lead experts who participated in the Second International Workshop on Permissible Exposure Levels for Occupational Exposure to Inorganic Lead also reached this conclusion in their final report:

> It is not known whether the maximum blood lead concentration or the integrated average concentration is the determining factor in the development of changes in nerve conduction velocity. However, the Group concluded from the data presented by Seppalainen et al. and the data reported in the literature that changes in nerve conduction velocity occur in some lead workers at blood levels exceeding 50 μg/100 ml. It was thought that no conclusion could be drawn from the one case in the blood lead range 40–49 μg/100 ml.
>
> It is not possible to decide what any given measured small deficit means in terms of specific nervous damage. However, it is generally recognized that a clear deficit in the nerve conduction velocity of more than one nerve is an early stage in the development of clinically manifest neuropathy. There is no evidence that these changes progress. Reversibility should be studied. Although slight changes may be measured in persons experiencing no symptoms, it was the consensus of the group that such changes should be regarded as a critical effect. (Ex. 262, p. 64) (Critical effect is a defined point in the relationship between dose and effect in the individual, namely the point at which an adverse effect occurs in cellular function of the critical organ.)

These conclusions by recognized experts in the field were based largely on the work of Seppalainen and her coworkers. This work has been described by an industry spokesman, Dr. Malcolm, as being "immaculate." (Tr. 2073) Based on the extensive evidence in the record from Seppalainen and others, OSHA has concluded that exposure to lead at low levels causes peripheral neuropathy at exposure levels previously thought to be of relatively little consequence. Seppalainen has stated:

> Of course, in terms of health, the importance of slight subclinical neuropathy can be questioned, too, and we did not find any evidence that the well-being of these workers was influenced by the neuropathy, apart from a few complaints of numbness of the arms. Thus, the term poisoning, in its orthodox sense, cannot be applied to these disorders. But neuropathy, no matter how slight, must be regarded as a more serious effect than the quite reversible alterations in heme synthesis, because the nervous system has a poor regenerative capacity, and the acceptability of such a response must be judged from that point of view. Since the entire question belongs to the diffuse "gray area" between health and disease, it is more than probable that opinions will diverge. We think, however, that no damage to the nervous system should be accepted, and that, therefore, present concepts of safe and unsafe PbB levels must be reconsidered. (Ex. 5 (12), p. 183.)

Recovery from the effects of chronic lead poisoning may be feasible in some cases, if the worker is removed from the source of exposure and therapy is initiated immediately. There are instances, however, when complete recovery is impossible and the pathology is fixed. Even if the worker is removed from the source and therapy initiated, the worker may still experience impairment. In a recent paper describing his results Dr. R. Baloh, a neurologist at UCLA, questioned the reversibility of nervous system damage:

> Although there are isolated reports of significant improvement in lead induced motor neuron disease and peripheral neuropathy after treatment with chelation therapy, most studies have not been encouraging, and in the case of motor neuron disease, death has occurred despite adequate chelation therapy.
>
> All of this data reinforces a disturbing clinical impression that nervous system damage from increased lead absorption is only partially reversible, if at all, with chelation therapy and/or removal from further exposure. This is not particularly surprising, however, since experience with other heavy metal intoxication has been similar. Nervous system damage from arsenic and mercury responds minimally to chelation therapy. Apparently, irreversible changes occur once the heavy metal is bound by nervous tissue. Although further study is clearly needed, the major point I would like to make this morning is that there is strong evidence to suggest the only reliable way to treat nervous system damage from increased lead absorption is to prevent its occurrence in the first place. (Ex. 27 (7), p. 55.)

OSHA agrees with these concerns regarding irreversibility of neurological disease expressed by Dr. Baloh and therefore must establish a standard which will prevent the development of nervous system pathology at its earliest stages.

In order to prevent peripheral neuropathy as evidenced by slowing in NCV's, Seppalainen testified that "to be safe, I would say 50 μg/100 g blood" is the necessary level. (Tr. 147.) Dr. Seppalainen further recommended that studies be performed to determine "the safety at the level of 50 μg/100 ml." (Tr. 153.) OSHA agrees that the current evidence demonstrates that nerve conduction velocity reduction occurs at PbB levels of 50 μg/100 g and above. Therefore, a necessary goal of a standard for occupational lead exposure must be to assure that blood lead levels are maintained below 50 μg/100 g in order to provide an adequate margin of safety.

(3) *Renal System.* One of the most important contributions to the understanding of adverse health effects associated with exposure to inorganic lead was the elucidation of evidence on kidney disease during the hearings. It is apparent that kidney disease from exposure to lead is far more prevalent than previously believed. In the past, the number of lead workers with kidney disease in the United States was thought to be negligible, but the record indicates that a substantial number of workers may be afflicted with this disease. Wedeen, a nephrologist (kidney specialist) who testified at the hearings for OSHA stated that a minimal estimate of the incidence of this disease (nephropathy) would be 10 percent of lead workers. "According to this estimate, there may be 100,000 cases of preventable renal disease in this country. * * * If only 10 percent of these hundred thousand workers with occupational nephropathy came to chronic hemodialysis (kidney machines) the cost to medicare alone would be about $200 million per year. (Tr. 1741–42.)

The hazard here is compounded by the fact that, unlike the hematopoietic system, routine screening is ineffective in early diagnosis. Renal disease may be detected through routine screening only after about two-thirds of kidney function is lost or upon manifestation of symptoms of renal failure are present. By the time lead nephropathy can be detected by usual clinical procedures, irreparable damage has most likely been sustained. When symptoms of renal failure are present, it is simply too late to correct or prevent the disease and "progression to death or dialysis is likely." (Tr. 1732.) The research of Wedeen and his coworkers, the health hazard evaluation by NIOSH at Eagle Picher Industries, Inc., and the research in secondary smelters by Lilis, Fishbein et al. demonstrated that lead exposure is a key etiologic agent in the development of kidney disease among occupationally exposed workers. Clearly, too little attention has been given

to lead-induced renal disease in recent years, and while OSHA recognizes that further research is required to understand fully the disease mechanism, it is also necessary to protect the thousands of workers who are potentially in danger of developing renal disease. The record indicates that blood lead is an inadequate indicator of renal disease development. Dr. Bridbord questioned Dr. Wedeen on the issue of chronicity of exposure and blood lead levels.

Dr. BRIDBORD. Well, looking at a group of workers, currently employed, having a blood lead level on that worker and having some information, that to the best of our knowledge there were no major changes in that particular plant during the past number of years. Would that not be a somewhat better index of what the blood lead levels might have been in the past. Considering too, that these workers are currently employed.

Dr. WEDEEN. Sure I think that the blood level measured close to the time of exposure is probably more reflective. I worry very much, that this may occur after a few months of exposure and the blood lead level may remain the same for the next 20 years, despite the fact that the individual is continually accumulating lead in the body.

Dr. BRIDBORD. Would you think that the chronicity of lead exposure, apart from precisely whether the blood lead was above or below 80 or above or below 60 for example, might be an important factor in determining the eventual development of renal disease in lead workers?

Dr. WEDEEN. Yes. That is just what I meant, that the accumulative effects and the cumulative body burden may be very different from the blood lead level at any moment in time.

In other words, one could certainly imagine that a blood lead level of 80, for 2 years, may be very similar to a blood lead level of 40, for 4 years. I don't have that data, but something like that may well exist in terms of the danger of the different levels of exposure.

Dr. BRIDBORD. Alright.

Particularly, in view of that, and given the requirements of the Occupational Safety and Health Act, that sets standards which protect during the working lifetime, would you have some reservations about a blood lead maximum standard, even at 60?

Dr. WEDEEN. I certainly would. And I think I just expressed the basis for it. You will note that in my recording of these patients, very very few of them had blood lead levels over 60. I just feel that while the blood lead level is maybe better than nothing, it may be very practical. It probably doesn't do the job we are trying to do and certainly not from the physician's point of view, who has seen the individual patient, who may or may not be a current exposure at the level that got his disease (Tr. 1765-1766.)

The lead standard must therefore be directed towards limiting exposure so that occupational lead nephropathy is prevented. The Agency agrees with the views of Wedeen:

I have reported today 19 lead workers who have lost 30 to 50 percent of their kidney function. Since they showed no symptoms and had no routine laboratory evidence of kidney disease, it may be asked why this kidney function loss should be viewed as material damage. Lead nephropathy is important because the worker has lost the functional reserve, the safety, provided by two normal kidneys. If one kidney becomes damaged, the normal person has another to rely upon. The lead worker with 50 percent loss of kidney function has no such security. Future loss of kidney function will normally occur with increasing age, and may be accelerated by hypertension or infection. The usual life processes will bring the lead worker to the point of uremia, while the normal individual still has considerable renal functional reserve. Loss of a kidney is therefore more serious than loss of an arm, for example. Loss of an arm leads to obvious limitations in activity. Loss of a kidney or an equivalent loss of kidney function means the lead worker's ability to survive the biologic events of life is severely reduced. By the time lead nephropathy can be detected by usual clinical procedures, enormous and irreparable damage has been sustained. The lead standard must be directed towards limiting exposure so that occupational lead nephropathy does not occur. (Tr. 1747-1750.)

And OSHA agrees with Dr. Richard Wedeen, that "40 μg/100 ml is the upper acceptable limit" (Tr. 1771) and with Dr. Bridbord who stated "I personally think that a blood lead of 60 is too high to give me assurances that we are really going to protect against these effects." (kidney) (Tr. 1375). That is, while PbB levels are an inadequate measure of occupational exposure (though most agree the best available single measurement) they nonetheless provide a basis for determining body burden when measured over an extended period of time. OSHA believes that maintenance of PbB levels at or below 40 μg/100 ml will reduce the overall dose to the worker, decrease the body burden of lead and prevent sufficient buildup of lead in the kidney to effect renal damage.

(4) *Reproductive effects.* Exposure to lead has profoundly adverse effects on the course of reproduction in both males and females. In male workers exposed to lead there is evidence of decreased sexual drive, degeneration of the testes, impotence, decreased ability to produce healthy sperm, and sterility. During the hearings there was considerable discussion of the evidence submitted by Lancranjan et al. which demonstrated that the reproductive ability of men occupationally exposed to lead is interfered with. Lancranjan reported a significant increase in malformed sperm (teratospermia) among lead-poisoned workmen (blood lead mean 74.5 μg/100 ml) and workmen with moderately increased absorption (blood lead mean 52.8 μg/100 ml). Decreased number of sperm (hypospermia) and decreased motility (athenospermia) were observed not only in the preceding groups but also in those with only slightly increased absorption (blood lead mean 41 μg/100 ml). The authors concluded that these alterations were produced by a direct toxic effect on the male gonads, and that a dose-response relationship exists with respect to teratospermia. The other parameters measured do not show as strong a relationship but are significantly altered over controls. This work is consistent with other earlier literature quoted by Lancranjan.

Epidemiologic studies have pointed out previously both the reduction of number of offsprings in families of workers occupationally exposed to lead and increase of the miscarriage rate in women whose husbands were exposed to lead. Experimental investigations have also shown both a reduction in the number of offspring of laboratory animals and reduced birthweight and survival of progenies of animals fed with diets containing lead. (Ex. 23 (38), p. 400.)

The Lancranjan study is strongly indicative of adverse effects on male reproductive ability at low lead levels, and there is conclusive evidence for a dose-response relationship with respect to teratospermia in these lead exposed workers. In OSHA's view teratospermia represents material impairment of health to the male. OSHA believes that this evidence and other studies support the conclusion that lead exerts markedly adverse effects on the reproductive ability of males.

Germ cells can be affected by lead which causes genetic damage in the egg or sperm cells before conception and which can be passed on to the developing fetus. The record indicates that genetic damage from lead occurs prior to conception in either father or mother. The result of genetic damage could be failure to implant, miscarriage, stillbirth or birth defects.

The record indicates that exposure of women to lead is associated with ovarian cycles, premature birth, menstrual disorders, abnormal sterility, spontaneous miscarriage, and stillbirths. Infants of mothers with lead poisoning have suffered from lowered birth weights, slower growth, and nervous system disorders and death was more likely in the first year of life.

There is conclusive evidence in the record that lead passes the placental barrier. Multiple studies have established that the fetus is exposed to lead because of the passage of lead through the placental membrane. This evidence was uncontroverted during the hearings. The lead levels in the mother's blood are comparable to concentrations of lead in the umbilical cord blood at birth. Transplacental passage becomes detectable at 12-14 weeks of gestation and increases from that point until birth.

Numerous parties to the hearings raised the issue of whether the fetus is the most sensitive organism requiring protection from exposure to lead.

Bridbord, for example, argued that the immaturity of the blood brain barrier in the newborn raises additional concern about the presence of lead in fetal tissues.

The proposed lead standard raised the possibility that "the risk to the fetus from intrauterine exposure to high levels of lead in the mother's blood is maximal in the first trimester of pregnancy when the condition of pregnancy may not be known with certainty" (Ex. 2, p. 45936; Ex. 95.) OSHA agrees with Dr. Vilma Hunt who testified that "the first trimester has not been shown to be the period of highest vulnerability for the fetus." (Ex. 59.) OSHA has concluded that the fetus is at risk from exposure to lead throughout the gestation period, and therefore protection must be afforded throughout pregnancy.

There is little direct data on damage to the fetus from exposure to lead but there are extensive studies which demonstrate neurobehavioral effect in children. OSHA believes that the fetus would be at least as susceptible to neurological damage and heme inhibition as would older children and therefore data on children is relevant to the fetus.

Exposure to lead would be expected to adversely effect heme biosynthesis and the nervous system earliest and most profoundly in the fetus and newborn. Early enzyme inhibition in the heme forming system has been well documented, and the central nervous system has its most significant growth during gestation and the first two years following birth.

Lead is capable of damaging both the central and peripheral nervous system. At high exposures to lead (80 μg/100 ml and above) the central nervous system of children may be severely damaged resulting in coma, cardiorespiratory arrest and death. Symptoms of acute encephalopathy similar to those in adults have been reported in infants and young children with a markedly higher incidence of severe symptoms and deaths occurring in them than in adults. In children once acute encephalopathy occurs there is a high probability of permanent, irreversible damage to the CNS.

There is data which demonstrates that permanent damage to the CNS has occurred in children exposed at low lead levels and in whom no overt symptoms were in evidence. Children whose blood leads levels were 50 μg/100 ml and above have demonstrated mild CNS symptoms including behavioral difficulties. Behavioral disturbances in children such as hyperactivity have been associated with blood lead levels between 25 and 55 μg/100 ml. Animal studies have confirmed these findings. Beattie demonstrated an increased probability of mental retardation in children exposed to lead via maternal ingestion of lead in water. Elevated blood lead levels were found in the retarded children compared to the control group. There appeared to be as significant relationship between blood lead concentration and mental retardation. Mean blood lead for the retarded children was 25.5 μg/100 ml. Water lead concentrations in the maternal home during pregnancy also correlated with the blood leads from the mentally retarded children.

Motor nerve conduction velocity (NCV) decrements indicating early peripheral neuropathy have been reported in children. Early studies showed NCV decrements in children whose blood lead levels were 40 μg/100 g and above.

While a critical review of the literature leads to the conclusion that blood lead levels of 50 to 60 μg/100 ml are likely sufficient to cause significant neurobehavioral impairments, there is evidence for effects such as hyperactivity as low as 25 μg/100 g. Given the available data, OSHA concludes that in order to protect the fetus from the effects of lead on the nervous system, maternal blood lead levels should be kept below 30 μg/100 g. In general, 30 μg/100 g appears to be reasonably protective insofar as it will minimize enzyme inhibition (ALAD and FEP) in the heme biosynthetic pathway and should minimize neurological damage. OSHA agrees with the Center for Disease Control (Ex. 2 (15)) and the National Academy of Sciences (Ex. 86M) that the blood lead level in children should be maintained below 30 μg/100 g. Levels above 30 μg/100 g should be considered elevated.

As previously stated there is conclusive evidence that lead passes the placental barrier thereby causing the fetus to be exposed to lead at comparable levels to the mother. Given this in utero lead exposure the fetus is therefore subject to the adverse effects of lead. It is significant to note that an analysis of human fetal tissue demonstrated the highest concentrations of lead in the bone, kidney, liver, brain, blood, and heart. The distribution of lead within the fetus raises the serious prospect that the fetus is susceptible to lead's adverse effects throughout gestation.

There is limited data on the effects of lead on the fetus but there is more extensive information on the susceptibility of infants and children to neurological damage from lead. OSHA believes that the fetus must be considered at risk to neurological damage from lead. Given the severity of neurological disease and the evidence indicating effects at low lead levels this conclusion raised particularly difficult issues when establishing this final standard. OSHA recognizes that a PbB level is not a measure of body burden, that the fetus would only be exposed during the period of gestation, and given the independent hematopoietic system of the fetus that maternal-cord blood leads may not be an accurate reflection of blood lead level in the fetus. However, even if these considerations may suggest a lessening of risk to the fetus, OSHA believes that blood lead levels of pregnant women should be maintained below 30 μg/100 ml in order to protect the fetus.

In general, OSHA believes that the evidence overwhelmingly indicates that the blood lead levels of both male and female workers who wish to plan pregnancies should be maintained below 30 μg/100 in order to prevent adverse effects from lead on the workers' reproductive abilities. To do this would minimize the risk of genetic damage, menstrual disorders, interference with sexual function, lowered fertility, difficulties in conception, damage to the fetus during pregnancy, spontaneous miscarriage, stillbirth, toxic effects on the newborn and problems with the health development of the newborn or developing child. OSHA cannot guarantee that 30 μg/100 g is a "no effect" level but it would provide marked protection to the fetus and therefore to the reproductive capacity of the worker.

During the hearings there was considerable testimony on reproductive effects in relation to the PEL and equal employment considerations. The basic issue had been raised by OSHA in the proposed lead standard:

> Recent studies of the toxicological effects of exposure to lead indicate certain groups of adult workers may have greater susceptibility to lead intoxication than the general worker population. One such group is female employees of childbearing age. It is known that lead absorbed into the bloodstream of pregnant women crosses the placental barrier and enters the blood of the fetus. This is of great concern because excessive exposure to lead during pregnancy has caused neurological damage in children. As noted in the Academy's report, the risk to the fetus from intrauterine exposure to high levels of lead in the mother's blood is maximal in the first trimester of pregnancy when the condition of pregnancy may not be known with certainty. It has also been established that the umbilical similar to that found in the mother's blood. This raises the serious possibility that the blood lead level in the mother might harm the fetus without producing any clinical symptoms of lead exposure in the mother.
>
> The extensive data on lead intoxication in children indicate that for several reasons, including their rapid growth, children may be susceptible to lead intoxication at lower blood lead levels than adults. The U.S. Public Health Service considered this and other factors when it recommended, in March 1975, that blood lead levels in children be kept below 30 μg/100 g. (Ex. 2, P45936.)

No topics were covered in greater depth or from more vantage points than the subject of women in the lead industry. More than a dozen witnesses testified to this issue; many others offered their views in response to questions; over 400 pages of the transcript of these proceedings were devoted to this issue. Participants in the hearings argued that, given the data demonstrating adverse effects on male reproductive abilities and potential genetic effects in males and females, fertile men were equally at risk as women of childbearing age. Therefore, the standard should be designed to fully protect all exposed workers, male and female.

Dr. Stellman testified as follows:

> In summary it can be stated that there is no scientific justification for placing all women of childbearing age in the category of a susceptible subgroup of the working population. There is sufficient data available to show that a significant proportion of the population is at risk for the effects of exposure to lead, and hence can also be deemed susceptible. Further, if the intent of the OSHA standard is to protect workers from hazards to reproduction there is still no justification for treating women separately from man. (Ex. 72.)

This view was supported by other witnesses (Ex. 92; Ex. 343, 59, 60A). Dr. Hunt, for example, stated:

> There is no evidence to allow a conclusion that women of childbearing age themselves are more susceptible to the adverse effects of lead. The susceptible population is made up firstly of the fetus in utero, actually present in the work environment and secondly the offspring of male and female workers with blood lead levels high enough to alter their genetic integrity. (Ex. 59, p. 26.)

OSHA believes that the record supports the conclusions of Drs. Stellman and Hunt that women of childbearing age exposed to lead are not more susceptible to adverse effects on their reproductive capacities than are male workers. There can be no doubt that the reproductive capability of both males and females is adversely effected by lead.

The susceptibility of the fetus, however, raises the issue of whether OSHA should seek to protect the fetus. OSHA has concluded that damage to a fetus due to parental exposure to lead represents material impairment of the reproductive capacity of the parent involved. Further, OSHA believes that it has the public health responsibility to insure to the degree feasible that a fetus or newborn does not suffer ill effects or diminution of health from parental exposure to lead.

OSHA recognizes that the PEL of 50 $\mu g/m^3$ alone will not maintain all worker PbB levels below 30 $\mu g/100$ g. The mean blood lead level of workers uniformly exposed to 50 $\mu g/m^3$ will be approximately 35 $\mu g/100$ g, and the population blood lead distribution is predicted to be: less than or equal to 30 $\mu g/100$ g, 30 percent; 30–40 $\mu g/100$ g, 40 percent; greater than or equal to 40, 30 percent. When full compliance is achieved with the 50 $\mu g/m^3$ PEL through engineering and work practice controls, however, there will be other factors which will have the effect of lowering these percentages. For example, the predicted distribution does not take into account implementation of the Environmental Protection Agency's standard of 1.5 $\mu g/m^3$ for lead in air in the general environment. Achievement of this level will tend to lower blood lead levels in the entire population thereby having the effect of reducing the baseline PbB levels of workers. Normal job turnover, a factor which will further reduce blood lead levels, is not considered in the foregoing percentages. There are also numerous industries affected by the standard whose exposure levels are intermediate or low and who will be able to lower their exposure levels well below the PEL with a minimum of effort. Finally, the percentage distribution cited assumes uniform compliance with 50 $\mu g/m^3$. When compliance is achieved in a particular plant, however, there will no doubt be many areas throughout the industrial operation where the air lead levels will be substantially below the PEL—therefore further reducing the blood lead levels of the aggregate work force. However, even taking these mitigating factors into account, there will often be a substantial percentage of workers whose blood lead levels exceed 30 $\mu g/100$ g. In recognition of the inability of the PEL alone to protect the reproductive capacity of all workers at all times, the standard includes a variety of additional protective elements designed to minimize reproductive risks. Use of these procedures by concerned employers and by informed workers will provide an acceptable margin of safety for the reproductive capacity of both male and female lead exposed workers. First, the standard establishes an action level of 30 $\mu g/m^3$ to trigger environmental and biological monitoring programs, as well as other medical surveillance procedures. The action level has been set at a point commensurate with the beginning of potential risks to reproductive capacity. Initiation of education and training is also tied to the action level so that workers will be fully informed of the nature of reproductive hazards presented by lead, and how the standard addresses these hazards. Workers have the ability to plan and control when they will parent a child. They can be expected to act responsibly when informed of the reproductive hazards presented by lead, and of the special precautionary measures established by the standard. Environmental monitoring, biological monitoring, and medical records are available to employees, and can be utilized when planning for a family.

The medical surveillance program under the standard provides workers the opportunity, upon request, of obtaining a medical examination or consultation concerning the effects of current or past exposure to lead on the employee's ability to procreate a healthy child. The employee may also obtain a second medical opinion by a physician of his or her choice, at no cost to the employee. As a part of the medical removal protection program, the multiple physician review mechanism may require an employer to implement any necessary special precautionary measures for an employee. For example, the employee might be temporarily provided with a powered air purifying respirator even though the employee would otherwise use no form of respirator. If the employee were currently using a respirator, he or she could, upon request, obtain such a respirator even without the recommendation of a physician. The physician review mechanism is empowered to protect the worker's reproductive capacity by whatever measures are appropriate under the circumstances. Temporary removal of a male or female worker (whether or not pregnant) from substantial lead exposure is one alternative. And, as part of the medical removal protection program, the employee would suffer no loss of earnings, seniority or other employment rights and benefits due to the need to be temporarily removed from lead exposure, or otherwise limited pursant to the standard. The medical surveillance program also offers employees the opportunity to obtain, upon request, either a male fertility test, or a pregnancy test.

The foregoing special precautionary measures incorporate the flexibility needed to address the varied circumstances of individual workers. Adverse health effects both to male and female reproductive capacity can be minimized by the use of these procedures, and, consequentially, an acceptable level of health protection is provided to all workers.

During the hearings there was considerable discussion on whether women of child-bearing age should be excluded from work in the lead industry in order to protect the fetus. Ms. Hricko testified that women of childbearing age had been excluded from employment because "the response of industry has been to 'protect' women workers from lead's reproductive hazards by refusing to hire them or by forcing them to prove that they can no longer bear children" (Ex. 60(a)). There was also testimony which dem-

onstrates that women have and do work in production areas of battery manufacturing (Tr. 1245, 4057, 4506, 4855, 5529, 5898).

While not directly suggesting that all women of childbearing age be excluded from employment in the lead industry the LIA argued that the issue of the fetus should be settled on a case-by-case basis rather than setting a standard which would be protective of the fetus.

> The association, in other words, believes that it is preferable to deal with this very difficult and complicated problem on a case-by-case basis, rather than by setting a standard which, although enormously expensive, would not achieve the desired objective. (Ex. 335, p. 40.)

Dr. Cole elaborated on this issue in his testimony:

> Women, quite rightly, want equal employment opportunity * * * (but) there are many jobs in the lead industry where blood-lead levels simply cannot be kept at levels known to be safe for the fetus.
>
> From a health protection standpoint, there is no feasible solution to this dilemma. However, if it is decided that the commitment to equal employment opportunity overrides the health considerations, then there should be a program which would insure that the female knows the risks, that the employer is protected from liability, and that information is obtained which would help us better to understand the degree of risk.
>
> This program would include fully advising the prospective female employee of the risk to the fetus inherent in the job she wishes, and the carrying out of a full-scale joint Government-industry-labor research program, both retrospective and prospective, of the reproductive consequences of occupational exposure to lead.
>
> As I mentioned earlier, this was proposed to NIOSH (by ILZRO) with the commitment of industry funds in 1975, with no response. It is clear to us, from our conversations that we have had with labor unions, NIOSH, OSHA, and company officials that no one has a truly satisfactory answer to this problem.
>
> We can demand, demonstrate, and agitate all we wish but is will not change the basic facts. And if OSHA decides that it must set a standard so low that it is known to be fully protective of the fetus, then we all must bear in mind that there will be very few jobs, indeed, in the lead industry for either men or women. (Cole 3069-70.)

The lead industry properly acknowledged the risk to the fetus from maternal exposure to lead but did not believe a standard could or should be promulgated which would protect the fetus. The LIA disregards, however, the role that the standard's special protective measures can play in protecting reproductive capacity consistent with continued employment of all people. The impact of the typical industry approach would ultimately lead to the exclusion of women of childbearing age from the workplace.

OSHA disagrees with the LIA conclusions and believes that the final standard can protect reproductive capacity of the parent, which in turn will protect the fetus. The agency has endeavored to set a comprehensive standard which will maximize protection to the male and female worker, to the fetus and to the offspring of workers. OSHA recognizes that not all risk can be entirely eliminated given the constraints of feasibility, but the final standard does effectively minimize reproductive risks. With this in mind, OSHA asserts that an employer who fully complies with this standard has no rational basis for the exclusion of women of childbearing age from the workplace.

c. *Clinical effects below 80 μg 100 g.* A general discussion of the most severe forms of lead intoxication was given in the preceding sections. Given them overt manifestations of lead intoxication the proposed lead standard proceeded to question at what exposure levels do these symptoms appear:

> A number of studies have sought to relate clinical symptoms and effects caused by lead exposure on workers' blood lead levels. There is little disagreement that the risk of clear-cut clinical symptoms related to exposure increases as blood lead levels rise above 80 μg/100 g. In addition, a number of studies have observed symptoms and effects caused by exposure to lead at blood lead levels below 80 μg/100 g. While 80 μg/100 g is a useful lower range for observed clear-cut clinical symptoms, we do not regard it as a sharp delineation above which clear-cut symptoms occur in all workers and below which clear-cut symptoms do not occur. Further workers with blood lead levels above 80 μg/100 g without clear-cut symptoms may have milder symptoms caused by lead exposure. It should be noted that in evaluating studies which seek to relate blood lead levels to symptoms of lead exposure, it is rarely possible in clinical situations to determine the amount of lead absorbed before the onset of symptoms of lead intoxication. In summary, it is OSHA's judgment that the probability of clinical symptoms of lead intoxication appearing is increased as blood lead levels rise above 80 μg/100 g. There are also data, however, to suggest that such symptoms may occur at blood lead levels under 80 μg/100 g, although perhaps not under 50 μg/100 g.

Throughout the rulemaking period industry representatives have steadfastly maintained that there exists no persuasive evidence to indicate that clinical lead intoxication occurs below blood lead concentration of 80 μg/100 g. (Ex. 335, p. 13.) In support of this contention LIA cites Dr. Robert Kehoe's recent publication.

Dr. Robert Kehoe, perhaps the most highly respected authority on lead intoxication in the world, concluded in an article published only last year (1976):

> It appears that no case of poisoning occurs until the concentration of lead in the blood reaches *at least* 80 μg/100 ml, and most cases of poisoning occur at a level well above this (100-300 μg/100 ml). (Exhibit 294B.) (Emphasis added.)

This is consistent with the view Kehoe expressed 15 years earlier in his Harben Lectures, when he explained that no case of even the mildest type of poisoning has been induced by the absorption of inorganic compounds of lead at blood-lead concentrations below 80 μg/100 g. (Exhibit 5(33).)

This article published only last year by Dr. Kehoe contains only one reference later than 1970 and this is Goyer, R. A. (1971) Lead and the Kidney, Curr. Topics Path. (*in press*). (Emphasis added.) It is apparent that this paper by Kehoe was originally written in 1970 or 1971 and only recently published. It addresses data developed prior to 1971, and does not discuss the more recent, important work in this field.

Dr. Kehoe has maintained that no lead poisoning occurs below 80 μg/100 g. For example LIA quotes Kehoe in their early brief: (Ex. 3(72), p. 19.)

> Experience and the accumulation of voluminous data have spoken for themselves, in proclaiming that cases of lead poisoning occur only when certain limits of concentration of lead in the urine or blood (or both) have been exceeded. *The critical concentration of lead in the blood of child or adult, below which * * * no case of even the mildest type of poisoning has been induced by the absorption of inorganic compounds of lead, is approximately 0.08 mg. (80 micrograms) per 100 grams of whole blood.* (Emphasis added.)

This statement is not accurate with respect to either children or adults, but it is especially troublesome with respect to children. The Center for Disease Control in their statement of March 1975 (Ex. 32(15)) define undue or increased lead absorption as occurring at PbB levels of 30-79 μg/100 g. The committee on Toxicology, National Academy of Sciences agreed with CDC and further stated:

> In order to allow for variation among individuals, the mean blood lead concentrations for groups should not exceed 20 μg/dl. (Ex. 86M.)

In addition, this is consistent with the evidence compiled by the Environmental Protection Agency (EPA) which led that agency to establish a national ambient air quality standard of 1.5 $\mu g/m^3$ designed to address the problem of lead in the urban environment. The EPA standard was based on the following considerations:

> In establishing the final standard, EPA determined that of the general population, young children (age 1-5 years) are the most sensitive to lead exposure. In 1970, there were 20 million children in the U.S. under 5 years old, of whom 12 million lived in urban areas and 5 million lived in center cities where lead exposure is the highest. The standard is based on preventing children in

the U.S. from exceeding a blood level of 30 micrograms lead per deciliter of blood. Blood lead levels above 30 micrograms are associated with an impairment in cell function which EPA regards as adverse to the health of chronically exposed children. There are a number of other adverse health effects associated with blood lead levels above 30 micrograms in children as well as in the general population, including the possibility that nervous system damage may occur in children even without overt symptoms of lead poisoning. (EPA Press Statement, September 29, 1978.)

The basis for the EPA conclusions is found in their Criteria Document, "Air Quality Criteria for Lead" (FEIS Ref. 92).

There are numerous studies showing effects on children and adults below 80 μg/100 g. Statements which are clearly at odds with current data raise serious questions about Dr. Kehoe's overall view of this field as it affects both children and adults. What a person thought to be true in 1960 may not stand up to critical scrutiny today especially given recent advances in the early recognition and detection of disease. Dr. Needleman of Harvard University offered another view during his testimony which OSHA believes is a far more accurate representation of reality:

Knowledge of the toxic effects of lead is almost as old as knowledge of its utility. It is recorded frequently through history and just as frequently ignored. No one quarrels with the evidence that the sequelae of lead doses sufficient to produce clinical cymptomos are found in many organ systems of the body are enduring and often catastrophic. Whether lesser internal doses are important health consequences is a topic of extensive and frequently redundant debate.

Opinion on this question tends to divide in relation to the nature of the individual or institution's sponsorship. Pediatricians and public health specialists are concerned that lesser levels of lead are hazardous while industry and its spokesmen maintain that evidence for low dose effects is faulty and far from persuasive.

I am one of those who believe that a substantial body of evidence is accumulating that the threshold for significant health effect depends on the avidity, sensitivity and sophistication with which we pursue it and that the lowering of acceptable body burdens in children and adults is scientifically and economically sound.

I should like to present some data to support those assertions.

1. Studies of—quote—"subclinical" lead poisoning in children. In 1943, Randolph Byers of my institution followed 20 children who had recovered from lead poisoning, 10 of whom had no evidence on encephalopathy. He found that 10 of the 20 were failing in school, had significant problems in perceptual motor function or were severely behavior disordered. Byers asked then, some 34 years ago, how many children with cognitive or behavioral disorder in the school system were in fact unidentified cases of lead intoxication. That is the burden of my research of the children.

With the passage of time, the defined acceptable blood level for a child under 6 has moved from 60—when I began my training in pediatrics not too long ago—to 50 to 40 micrograms per deciliter. The CDC now begins to talk about 20 as the threshold for undue lead exposure. And Professor Zielhuis at the Amsterdam meeting in 1972 recommended an individual limit of 35 micrograms per deciliter and a group average of 20 micrograms per deciliter for children.

A number of studies of intellectual, perceptual and behavioral consequences of low level lead exposure in children have produced mixed results. Some have found impairment and some have not. Many, if not most of the studies are flawed in that insensitive outcome measures or inadequate measures of internal dose were used.

The import of these studies and others is that if one looks carefully for lead effects in children, you are likely to find them at lower levels of exposure than were formerly held. (Tr. 1077-79.)

There are important differences during the time that the blood brain barrier is being laid down, in that certain enzymes are being induced, but I think that the point that I was trying to generate in that argument, was that in my pediatric experience, when I started training in pediatrics, we said that children with blood leads over 80 were at high risk for the lead poisoning, and now we have been talking about children of 30, 45, or 40, and I think the same argument, serving out of sharp and clinical and experimental evidence, would apply to the worker that is, that if you look more carefully for evidence of impairment, you are going to find it.

The fact that an adult worker will spill aminolevulinic acid in his urine, at a blood lead of 40, to me says, that that is a clinical effect of significance. (Tr. 1106-07.)

During the rulemaking proceeding ASARCO submitted a study by Dr. Hine et al. entitled "Assessment of Health of Employees with Different Body Burdens of Lead." (Ex. 142G.) The authors apparently studied 652 employees with 5 or more years of service at six ASARCO locations. An extensive battery of tests were carried out which included blood pressure, measure of weight changes, hematology, blood chemistry, including kidney and liver function tests, urinalysis. The authors stated their conclusions as follows:

The results of this study demonstrated that there were no significant differences in the health of workers with blood lead concentrations between 60 and 80 μg/dl and those whose blood lead concentrations were more than 60 μg/dl. Even though the population studies has been substantially exposed above the newly proposed TLV of 0.10 mg/m³, there have been only a few cases of clinical problems related to the lead exposure, and few, in the opinion of the attending physician, have required chelation therapy for the reduction of the body burden of lead.

Based on these findings, it is our opinion that the current blood lead standard of 80 μg/dl can be kept, unless more new data will support the OSHA proposal. Also, the OSHA recommendation of monthly medical examinations appears to be too rigid. Our data indicate that it is possible to maintain a high degree of employee health with much less frequent examinations, with the frequency increased only if the blood lead concentration is found to be elevated beyond 80 μg/dl. We believe that implementation of this proposal of OSHA would not add any further dimension to the less rigorous protection program employed by ASARCO. (Journal of Occ. Med., 20, pp. 610-17; September 1978.)

Unfortunately the study suffers from problems of design which OSHA finds invalidates the authors conclusions. First, there is no well defined study population. In fact, in one table the results are given in terms of the number of determinations carried out rather than the number of subjects examined and the maximum number of determinations is 387. It is unclear how to compare 652 workers with 387 determinations. For example, out of 652 workers 387 determinations of BUN were made and there were 319 S-creat, but there were only 229 determination of the ratio of BUN to S-creat. The disparity between these numbers is not explained nor do we know whether these determinations were carried out on 100, 200, 387 workers or how many. In other words the study suffers from a serious lack of information, which could bias any conclusion. In addition there appears to have been some bias introduced in the original selection of the study group. During the study itself it is not clear how the subjects are counted. It appears some may be counted once and others several times.

The authors do not indicate whether there was uniformity in the manner in which medical examinations were given at each plant and it appears there was no company policy for general medical examinations. This could have introduced variability into the study. More significantly the laboratory analyses were done in six separate laboratories. Given the quality control problems which have been described in this record this would indicate additional variability may have been added. There are other biostatistical problems relating to the authors use of test of significance, e.g. the choice of two tailed tests and use of probability levels. There are other problems with this work especially with respect to population definition but it suffices to say this was not a well controlled epidemiologic study utilizing a precise methodology. Rather it represents a compilation of data without any well defined study objectives. The data provides no basis for the authors conclusions and accordingly OSHA believes it should be given little if any weight in these proceedings.

Industry representatives during the hearings frequently quoted Dr. Kehoe's conclusions in a totally uncritical fashion thereby raising doubts about the credibility of the argument,

for example the response of Dr. Michael Williams for the Lead Industries Association (Tr. 1899-1890):

Mr. KUCHENBECKER. On the bottom of page 1 when you talk about scanty published data and then you go on to discuss Dr. Robert Kehoe and Ronald Lane. I assume that these gentlemen were publishing studies and doing research in the 1930s, 1940s, 1950s, 1060s, is that the general range?

Dr. WILLIAMS. They certainly published studies and did research. There was very little data on air leads. Ronald Lane published an opinion. In those days the great men in the field felt able to publish their personal experiences and personal opinions, and these were usually accepted for want of anything better perhaps. But now that we have gone technical and scientific and have to back up every opinion with data and very often I am afraid the data that you collect in the study is much less valuable than the data of a lifetime experience in the field.

In later questioning, Dr. Williams again indicated his dependence on Kehoe's opinions even if they were not supported by data:

Mr. SAMUELS. Let's go to page 2 doctor. You mentioned the Kehoe data and you said, "the men remained in good health." Is it not true that Dr. Kehoe's data does not show that every man who he examined was in good health? When he drew his line for 80 μg were there not values below that line?

Dr. WILLIAMS. I am not clear about which paper you are referring to.

Mr. SAMUELS. In any of his papers where he deals with populations are all of the values showing effects above 80 μg of blood lead?

Dr. WILLIAMS. I have never read a paper of his which produced data. I am saying that this was his stated opinion.

Mr. SAMUELS. So you have never read a paper of his that produced data. You are just going by his opinion? Is that what you are saying? You have never looked at the original papers of Dr. Kehoe?

Dr. WILLIAMS. Yes, I read the lead papers in the 1930s and the Harvard lectures, but he did not give data on every case he examined. (Tr. 1930.)

The assumption of no severe morbidity below 80 μg/100 g could and may have had tragic consequences, especially given the tenacity with which this view is maintained. Cases of overt lead intoxication may have been ignored, thereby contributing to the development of even more severe chronic disease. For example, industry medical spokesmen stated that symptoms frequently associated with lead exposure would not necessarily be associated with lead unless the blood level was sufficiently high. This is noted in questioning of Dr. Williams (Tr. 1945):

Dr. BRIDBORD. If someone indicated that they did not feel well, how would you go about ruling out lead?

Dr. WILLIAMS. I would ask to see him afterwards for a full history and examination. I would undertake measurements of his lead absorption at the time. If he had a low blood lead, I would think it not likely to be due to lead.

Again in questioning regarding one of his publications in 1966, in which anemias in workers were not attributed to lead because the measurement of lead in blood was not sufficiently high, Dr. Williams responded that anemia was not considered to be lead poisoning in those days (Tr. 1976).

The same reasoning was expressed by Dr. Dennis Malcolm of Chloride, Inc., who indicated that his decision in such a case would be influenced by the blood lead level. (Tr. 2141.)

In the extreme situation, this may have very serious consequences. Dr. Wedeen, for example, told of the experience of one New Jersey worker:

One of these had repeatedly been hospitalized and even subjected to gallbladder surgery because of abdominal pain which, of course, in retrospect was probably lead colic. Lead poisoning was excluded as the cause of abdominal pain because blood leads fell within the 1972 NIOSH criteria guidelines. After a few weeks at home or in the hospital, his blood lead was always under 60 micrograms percent, and the diagnosis of lead poisoning was therefore missed for 2 years. Indeed, it at first appeared that the gallbladder surgery had cured his abdominal pain because lead colic, like abnormal blood lead levels, often disappears within weeks once lead exposure is stopped. (Tr. 1745.)

This assumption of no overt symptoms in a person or population until a blood lead of 80 μg/100 g suffers from reasonably serious methodological problems in the view of this agency. First, it would appear to ignore individual variation. Second, it oversimplifies current understanding of dose-response relationships and is contradictory to modern clinical medicine's concept of disease progression, insofar as early and milder signs are perceived to be part of a disease process which ultimately progresses to chronic, irreversible disease and even death.

The evidence in the record repeatedly indicates clinical symptoms due to lead absorption does occur in workers whose blood lead levels are less than 80 μg/100 g. Industry representatives argued studies showing effects below 80 μg/100 g do not address the issue of whether the workers may have had higher blood lead at an earlier point in time. That is, the blood lead level measured at the time of the observation may not be representative of previous exposures. LIA quotes Kehoe on this issue:

Under conditions of prolonged and gradual absorption of lead the time of onset (of symptoms) is * * * uncertain. The symptoms * * * of lead poisoning often persist after the blood concentration has declined well below 80 μg/100 ml so that, if the threshold value at the onset is to be known, the concentration must be determined close to the onset of illness. (Ex. 204B.)

This methodological criticism of numerous studies was frequently raised and OSHA has discussed it in another context when addressing studies on air-to-blood relationships. This problem is by no means limited to lead, since it is an inherent problem in cross-sectional studies. Generally one must be rather conservative about using cross-sectional data, obtained at a given point in time, to predict effects over time. Given the importance of the history of lead exposure in determining the significance of blood lead level and body burden, OSHA believes well-controlled longitudinal studies are more accurate representations of reality. This point is clearly brought out in the classic text on epidemiology by Drs. MacMahon and Pugh, where they state the following in defining the two terms:

Cross-sectional and longitudinal studies. Epidemiologic studies can also be characterized by whether the ascertainment of cause and effect relate to two different points in time or to a single point. In a longitudinal study the observations relate to two different points in time—even if both items of information are collected simultaneously. Most cohort and case-control studies are longitudinal in nature. In a cross-sectional study, on the other hand, measurements of cause and effect are made at the same point in time. Although the cross-sectional study is easier and more economical than the longitudinal, it is limited to studies of causes that are permanent, or reasonably permanent, characteristics of the individual, so that his status with respect to the cause measured at the time he has the disease has a high probability of reflecting his status at the time the disease was induced. MacMahon and Pugh, Epidemiology, Principles and Methods. Little, Brown and Company, 1970.

Despite these recognized methodological weaknesses, OSHA is obligated under the act to utilize all available scientific and medical information, especially when reasonable inferences may be drawn which in concert with other studies enable the agency to make decisions which will protect workers from occupational disease. OSHA declines to, in effect, penalize workers who may or may not have had higher levels of exposure in the past, by assuming that all morbidity was associated with former excessive exposure. That view is potentially self-serving and contradictory to other information available from other studies. OSHA has no intention of blindly accepting cross-sectional studies since spurious results are of no benefit to any party in a rulemaking process. Especially in this standard, issues of body burden, blood levels, air-lead

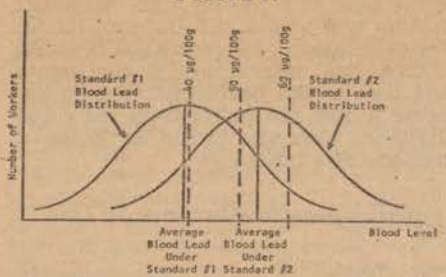
levels, and efficiency of other biological indicators have a crucial role. OSHA has weighed each study to determine its accuracy, precision soundness of methodology and has ultimately developed the PEL on the basis of all the research presented, although only a portion has been discussed in detail. In evaluating the research, OSHA has given substantial weight to published work because of peer review and to scientific testimony which must withstand the rigors of cross-examination. OSHA recognizes that blood lead level measurements are not necessarily accurate representations of past exposure, but with this in mind the agency does assert that there is conclusive evidence for "clinical effects" below 80 μg/100 g. There is in fact evidence of signs and symptoms (morbidity) at levels as low as 40 μg/100 g. That evidence is discussed in detail in the health effects section.

2. *Benefits.* The dramatic reduction in blood lead levels over 40 μg/100 g, as shown below, is a measure of the incremental benefit derived from the PEL of 50 μg/m³. OSHA has concluded that based on the health effects data in the record blood lead levels should be maintained below 40 μg/100 g to the extent feasible. Ideally, it would be desirable to express the health benefits of the lead standard in terms of decreases in the incidence and severity of the various adverse health effects of lead exposure (e.g., neurological damage, kidney damage, etc.). However, the available data does not allow meaningful quantitative estimation of the degree of prevention of the different forms of health damage likely to be achieved by lowering worker air exposures and blood lead levels by various amounts for various periods of time. The record evidence allows estimates to be made of the blood lead levels likely to result from compliance with alternative air standards. Absent better health effects data, judgment of the relative health benefits achievable with different lead standards can be based on the expected reduction in the number of workers with dangerously high blood lead levels.

The results will be expressed in terms of the number of workers expected to fall into particular blood lead level ranges over 40 μg/100 g at any one time after the establishment of the long-term equilibrium and, before consideration of the effects of the lead standard's medical removal provisions. OSHA believes that this is the single most convenient proxy for benefits for use in facilitating comparisons of different assumed compliance levels. However, there are a number of inherent limitations in this approach which need to be clearly appreciated.

First, it should be understood that a change in air lead exposure leads to a shift in the entire distribution of the blood lead levels in the population:

FIGURE 1.



Although the incremental benefits of standard No. 1 over standard No. 2 may be expressed in terms of the decrease in the number of workers (area under the curve) falling in each of the blood lead level ranges, the "benefits" of the standard are not really limited to workers who move across the lines drawn at 40, 50, and 60 μg/100 g. All of the workers are expected to have lower blood lead levels to some degree (and therefore possibly some lower level of health risk) under the lower exposure standard. It should be noted that measuring benefits by comparing differences in mean blood lead levels will markedly underestimate the benefits to a population of workers.

The public hearings included widespread discussion of the significance of small changes in blood lead levels as a result of rather large changes in air leads. (Tr. 266-67, 3114-15, Ex. 285, p. 13-14, 3064-65, 1923). For example, using the Bernard model, the difference between the average blood lead levels at 50 μg/m³ and 100 μg/m³ is approximately 5.6 μg/100 g. Seen in this context the benefits appear rather small. However, when viewed in the context of the shift in the blood lead distribution for all workers, the benefits are clearly more dramatic.

Second, it should be stressed that the measurement of benefits we have chosen represents a continuous "flow," not a "stock." As time passes and workers move into and out of employment in lead-exposed industries, the differences between compliance with different standard exposure levels continuously generate differences in the population of newly exposed workers. If two standards differ by 1,000 in the number of workers expected to be over 60 μg/100 g at any one time, over a period of 10 years, the difference is clearly 10,000 person-years at the higher blood lead level, spread out over a number of workers which depends on the labor turnover in the industries concerned, the frequency with which workers change jobs (and hence exposures) within the industry and other factors.

Third, the results only reflect the situation after longrun equilibrium of blood lead levels to the post-compliance distribution of lead exposure in the working population. Because blood lead levels of current workers to some degree may reflect the workers' entire previous career history of lead exposures, full "equilibrium" of the distribution of worker blood levels may not be achieved for many years after compliance with air-exposure requirements. Because the dynamics of changing blood levels in response to changing air levels are complex, the "flow" of benefits (reduction in number of workers in various blood level ranges) at various times prior to equilibrium may be either greater or less than the "equilibrium" values.

The primary analysis of the expected benefits of alternative compliance levels is based on the model of air lead/blood lead/job tenure relationships and other associated assumptions given by Ashford et al., in testimony for the Medical Removal Hearings (Ex. 439A). Before addressing the incremental benefits of the PEL discussion a review of the air-blood relationships is in order.

a. *Relationships between air lead levels and population-average blood lead levels.* The section on the air-to-blood level relationship discussed the difficulties with existing epidemiological studies of air lead/blood lead relationship. One point from that discussion merits emphasis here: Because available studies in working populations have only assessed the relationships between blood lead levels and air lead levels over relatively brief time periods (weeks or months) compared with the actual duration of exposure in working populations, and because existing physiological models of lead transport in the body suggest that exposures over many years contribute significantly to current blood levels, it can be expected that the results of current epidemiological studies are systematically biased in two ways:

- The slope of regression lines relating blood lead to air lead is smaller than it would be if exposure duration were properly lengthened to include workers' entire job tenure; and
- The intercept (intersection of the regression line with the zero-occupational-exposure axis) is unrealistically high because the long term buildup of lead in slow-exchanging pools creates an elevated "floor" of blood lead level which is unaffected by current exposure.

The primary implication of the first bias for the long term incremental effects of different lead standards is that the epidemiological studies will predict a more modest reduction in average blood lead levels than is likely to be the case at long term equilibrium. The effect of the second bias is complicated by the uncertainty in variability in blood lead levels about the mean, but suffice it to say here that it may sometimes be an upward bias by moving a larger percentage of

the worker population to higher blood lead levels.

The following table lists many of the relationships derived from different studies in the record. For comparison, the lower part of the table shows the relationships predicted by the Bernard model and Assumption C for exposures over 12.5 $\mu g/m^3$. The "Results" section will utilize the Barnard model for calculation of incremental benefits OSHA has determined it represents the best model developed to date because it does not suffer from the flaws discussed here and in the air to blood section. (See table 1.)

TABLE 1.—*Best Point Estimates of Ultimate Equilibrium Benefits of Reducing Air Lead Exposures*

[Blood level standard Deviation=9.5 $\mu g/100$ g]

Long-term average air lead exposure	Total number of workers	60 μg/100 g	50-60 μg/100 g	40-50 μg/100 g	Total 40 $\mu g/100$ g
Current Compliance Level					
> 100 $\mu g/m^3$	41,622	27,652	8,508	4,166	40,326
50-100 $\mu g/m^3$	55,885	5,125	14,379	19,732	39,243
	97,507	32,777	22,887	23,898	79,569
Compliance With 200 $\mu g/m^3$					
> 100 $\mu g/m^3$	41,622	9,340	13,569	11,958	34,867
50-100 $\mu g/m^3$	55,885	5,125	14,379	19,732	39,243
	97,507	14,465	27,948	31,690	74,110
Compliance With 100 $\mu g/m^3$					
> 50 $\mu g/m^3$	97,507	2,562	14,041	32,870	49,475
Compliance With 50 $\mu g/m^3$					
< 50 $\mu g/m^3$	97,507	498	5,373	22,729	28,599
Incremental Benefits					
b over a		18,312	−(5,061)	−(7,792)	5,459
c over a		30,215	8,846	−(8,972)	30,094
d over a		32,279	17,514	1,169	50,970
c over b		11,903	13,907	−(1,180)	24,635
d over b		13,967	22,575	8,961	45,511
d over c		2,064	8,668	10,141	20,876

Even if the all-industry average relationship of population-average blood lead levels to air lead level and job tenure were known with precision, there would still be many reasons why individual workers exposed at standard compliance levels would have different blood lead levels. Some of the differences arise from intrinsic biological and other differences between workers:

- Individual differences in size, body composition (relative sizes of potential lead storage pools).
- Individual differences in lead absorption (e.g., from short term fluctuations and long term differences in dietary habits, gastrointestinal function).
- Individual differences in lead excretion (e.g., from short term fluctuations and long term differences in water and salt elimination, kidney function).
- Individual differences in nonoccupational lead exposures.

Other differences arise from aspects of workers' jobs and job environment that are not controlled (or not completely controlled) by the provisions of the lead standard:

- Differences in work demands producing differences in the volume of air breathed (the potential variability arising from this factor is very large; the respiratory intake of a standard 70 kg. man varies from 3.6[3] during 8 hours of rest to 9.6 m^3 during 8 hours of light work or normal nonoccupational activity. Even larger amounts of air are taken in during heavy work. Six subjects performing heavy work (600-800 kgm-min.) on a bicycle ergometer had total ventilation averaging five times their resting rates.).
- Differences in work habits (e.g., hygiene, smoking) affecting the relative levels of inhalation and non-inhalation routes of lead exposure.
- Miscellaneous environmental conditions affecting physiological processes (heat, humidity, other chemical and physical stressors).
- Variation in the work week (overtime, etc.).
- Short-term (days or weeks) variation of total air levels.

Some of these factors produce mainly short term variability in blood lead levels (differences between worker blood level response which tend to persist for only days or weeks); other factors produce consistent systematic differences between worker blood level response over long periods, and some factors may produce both long and short-term variability. A spurious source of apparent additional short-term variation is measurement error in blood lead level determinations.

given that there will be both true short-term and true long-term variability in the blood levels which will result from air lead levels in compliance with the lead standard, we are faced with a difficult choice in the computation of incremental benefits. Should the calculation of the number of workers in various blood lead level ranges at any one time include only those whose true long-term average blood level is in a particular range, or should the calculation include workers in the range at any one time who may be only briefly elevated from their long term average levels below the lower boundary of the range?

The resolution of this issue depends on one's view of the biological significance of short periods of elevated blood lead. With the exception of measurement error, it is conceivable that all of variation (both short and long term) is of biological significance. That is, it is possible that elevated levels of lead begin to produce biological effects whenever blood lead exceeds a certain level. If that is the case, then a proper calculation of incremental benefits of controlling lead exposure should include all workers who are prevented from incurring true blood lead levels over 40, 50, or 60 $\mu g/100$ g, even if in many cases it could be expected that individual workers would only be over the indicated blood level for short periods. On the other hand, if biological damage depends only on long-term average blood lead level, the calculation will be a more accurate proxy for biological benefits if only long-term variation in blood levels is considered.

This question is clearly on the frontiers of current scientific understanding. OSHA has therefore undertaken alternative calculations based on a wide range of potential variation in blood lead level about predicted popu-

lation means. On the basis of data in the record from the Delco-Remy study, we have computed a minimum estimate of long-term (over 1 year) individual variation in blood lead level (standard deviation=5.5 μg/100 g). The estimate is likely to be an underestimate of true long-term variability because a study conducted within a single plant over a limited period of time is unlikely to include as large a diversity in the many factors producing long-term variability (see listing above) as would prevail in a random sample of all lead-using industries. As a high estimate of total variability, we have chosen to use the highest value found suggested in the record (standard deviation=15 μg/100 g), even though this value contains an allowance for measurement error, which, as previously mentioned, carries no biological significance. OSHA has chosen to base our midrange estimate calculations on the blood level variability assumption used in the original CPA report (standard deviation=9.5 μg/100 g). This estimate was originally developed as an upper bound on the long-term variability of blood lead levels, but if short-term variability is considered as well, it represents a best guess.

Although calculations were made for standard deviations of 5.5, 9./5, and 15 μg/100 g OSHA will only reproduce the values 9.5 μg/100 g since this value represents the best guess in terms of both long- and short-term variability.

b. *Results.* D.B. Associates has presented rough estimates of exposure covering many industries. OSHA bases its assessments of the incremental benefits of the air lead standard on this data and other record evidence. These estimates indicate that overall, approximately 41,622 workers are currently exposed to time-weighted-average air lead levels of over 100 μg/m³ and an additional 55,885 workers are exposed to air lead levels between 50 and 100 μg/m³.

The results presented in this section are obtained by multiplying the appropriate exposure estimates by the alternative estimates of the percentages of each population expected to have blood levels in the various blood levels ranges at any one time after the establishment of long-term equilibrium.

Figure 2 summarizes our best point estimates of the ultimate effects of achieving various air lead compliance levels (a-d). The left side of the figure shows the results of parallel computation of the number of workers in various blood lead level ranges. The right side of the figure shows the incremental benefits (reduction of the number of workers in each blood level range) of the "b_i", "c" and "d" compliance levels compared to the baseline defined by the "a" compliance level. (See figure 2.)

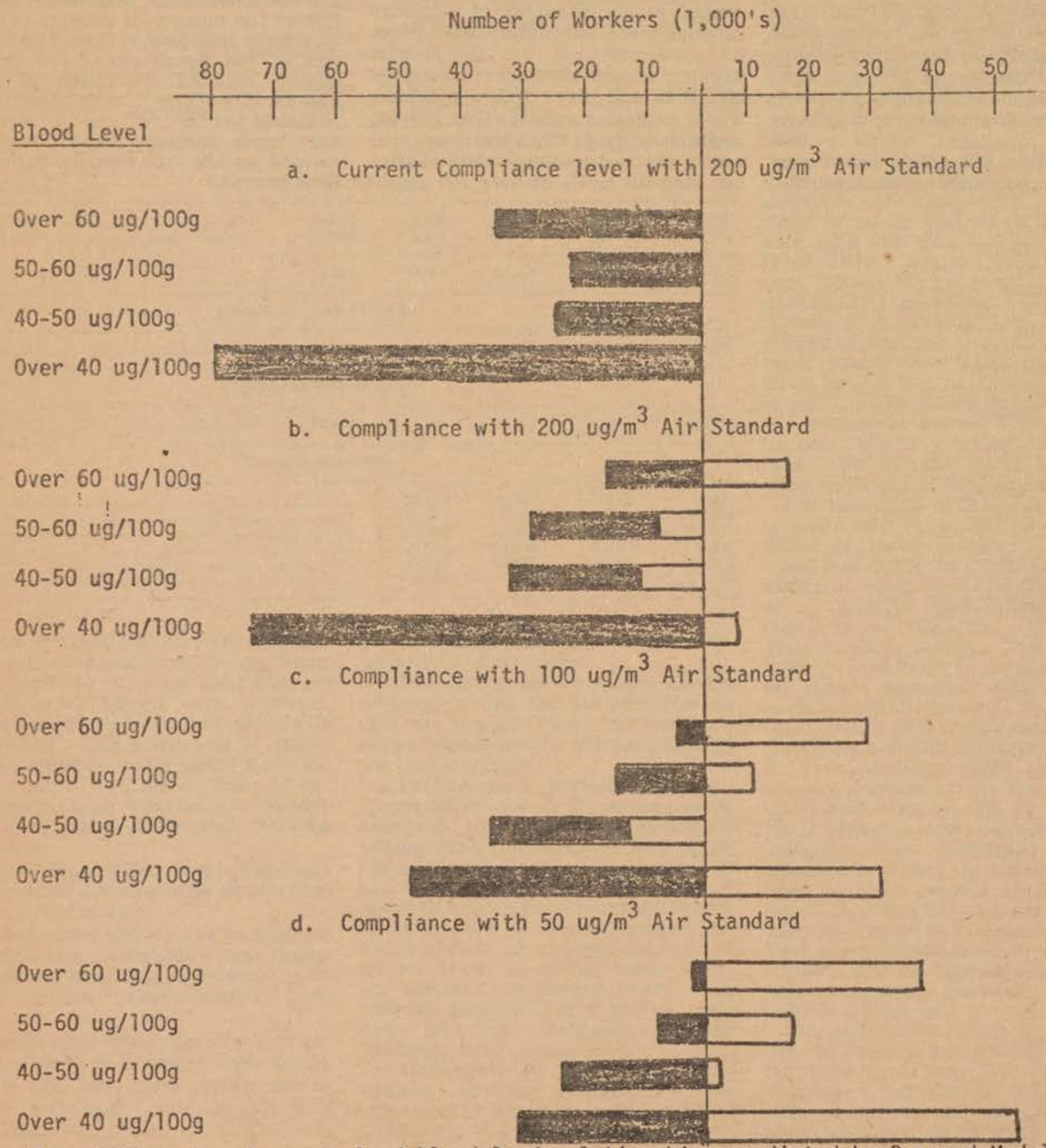
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FIGURE 1.

BEST POINT ESTIMATES OF ULTIMATE EQUILIBRIUM BENEFITS OF REDUCING AIR LEAD EXPOSURES UNDER DIFFERENT BLOOD LEAD LEVEL VARIABILITY ASSUMPTIONS*
Blood Level Standard Deviation = 9/5 ug/100g

"Residual Health Hazard" (Number Remaining in Each Blood Level Range at Any One Time After Equilibrium)

"Benefits of Regulation" (Number Prevented from Being in Indicated Blood Level Range at Any One Time, Compared to the "0" Compliance Level)



*Computations based on air lead-blood lead relationships predicted by Bernard Model and Assumption C and DBA's best point estimates of exposure.

It can be seen from figure 2 that assuming compliance with the present standard (the "a" compliance level), large numbers of workers could be expected to have potentially hazardous blood levels. At any one time, we anticipate that about 41,622 workers would have blood lead levels over 60 μg/100 g, and about 79,569 would have blood levels over 40 μg/100 g, in the absence of other remedial measures. Achievement of the "b" compliance level would reduce the number of workers over 60 μg/100 g, but would leave the number of workers in the 50-60 μg/100 g and 40-50 μg/100 g range substantially unchanged. Achievement of the "c" compliance level would be expected to make reduction to about 2,500 in the number of workers over 60 μg/100 g, and would be expected to produce some reduction in the numbers of workers in the 50-60 μg/100 g blood lead level range to 14,000. The "d" compliance level would reduce the total number of workers over 40 μg/100 g to slightly under 28,599, as compared over 79,569 for the "a" scenario. The incremental benefit of "d" over "a" in terms of workers over 40 μg/100 g would be 50,970 and for workers over 60 μg/m³ the benefit would be 32,270. These are clearly substantial reductions in the number of workers with excessive blood lead levels and would represent marked benefits to lead exposed workers.

A recent decision of the U.S. Court of Appeals for the Fifth Circuit vacated and remanded OSHA's benzene standard. (*American Petroleum Institute* v. *OSHA*, October 5, 1978.) The Court construed the language in section 3(8) of the act to require OSHA, when promulgating standard, to quantify the extent of the expected benefits and to determine whether the benefits bear a reasonable relationship to the costs the standard would impose on employers. OSHA does not accede to the Court's interpretation of the act but has nonetheless determined that the costs imposed by this lead standard (see attachment D of this preamble for cost and economic impact data) are clearly justified in view of the substantial increase in worker protection this standard would afford. OSHA has quantified the expected health benefits as described above. On the basis of the evidence in the rulemaking record OSHA has concluded that its evaluation of the relationship between the costs and benefits meets the test enunciated by the Fifth Circuit.

3. *Alternatives to the permissible exposure limit.*

a. *The LIA proposal.*

The most comprehensive alternative proposal submitted in the rulemaking record was the Lead Industries Association proposal:

> The health of workers can be best and most promptly protected by promulgating a standard which emphasizes the proper importance of biological indices and medical surveillance and which establishes a simple, effective and inexpensive enforcement procedure directly utilizing those indices. Employers covered by the standard should be required to conduct regular environmental monitoring and should adopt and submit to OSHA written compliance programs designed to reduce air-lead levels, to the extent feasible, by engineering controls; however, a specific air-lead level number should not be adopted for enforcement purposes, since such a compliance mechanism (even if based on the proposed permissible limit of 100 μg/m³) will not accomplish the objectives of protecting workers' health. (Ex. 335, p. A-1.)

The specific requirements in the LIA proposal are not entirely dissimilar from requirements in this final standard. However, there are certain significant differences which necessitate further discussion to explain OSHA's rationale. The major differences are:

a. The permissible exposure limit established by OSHA is 50 μg/m³ and primary compliance with the standard will be based on environmental monitoring by OSHA's industrial hygienists rather than relying on biological indices for enforcement.

b. OSHA has determined that the blood-lead level of employees should be maintained at or below 40 μg/100 g. The OSHA action level is an air lead level of 30 μg/m³. The LIA proposal sets 80 μg/100 g as the appropriate exposure limit for compliance purposes with a blood lead action level of 60 μg/100 g.

c. OSHA will continue to place primary reliance on engineering and work practice controls for compliance with the standard whereas LIA included the following factor to be considered in complying with the standard.

> The predicted and relative effectiveness of such (engineering) controls and *of other protective devices* (emphasis added) in protecting workers against material impairment of health and functional capacity. (emphasis added)

There are other differences but these represent the most significant issues to be addressed. Before addressing these issues in detail the following represents a summary of the reasons for OSHA's decision to adopt a PEL based upon air lead determination. In summary, OSHA has decided to place primary a reliance on a PEL which is based on environmental monitoring of air lead levels rather than relying on biological indices for the following reasons.

1. Evaluation of the industrial environment by proven industrial hygiene techniques is a direct measure of the sources of lead exposure, adequacy of control technology, progress in implementation of engineering controls, and in general represents a continual check on lead exposure. Since OSHA believes that control of an air contaminant should be accomplished at the source, environmental monitoring then is a direct measure of the control of lead exposure. Biological monitoring is designed to ascertain problems in individual workers and is an indirect and inadequate measure of the control of lead. In this regard environmental monitoring is better suited to serve as a basis for enforcement.

2. Biological monitoring for compliance purposes is not feasible since there is no discrete value which would serve as the basis for citation. OSHA believes that based on consideration of health effects a PEL of 80, 70, or 60 μg/100 g would be excessive and would not protect workers health adequately. It would be infeasible to require controls to maintain blood lead levels at the desired 40 μg/100 g and below. Rather when all controls have been implemented 30 percent of all workers PbB will range from 40 to 60 μg/100 g. Given this distribution of blood lead levels at compliance in a worker population there is no discrete value which would serve as a biological PEL. That is, OSHA believes a PbB above 60 μg/100 g is excessive but a PbB level in an individual worker between 40 to 50 μg/100 g may be the result of excessive exposure or it may represent the individual variation within a well controlled environment. Air lead determinations would differentiate between the two situations.

3. A biological standard is not only infeasible it would provide inadequate protection of workers. Excessive exposure to lead would not immediately cause excessive blood lead levels. In fact, some workers' blood leads might not rise to excessive levels for years, although their body burden would be increasing. Workers should not be expected to wait for protection until their blood leads become excessive. Air monitoring pinpoints overexposures immediately. This technique is preferable, therefore, for compliance purposes.

4. Worker Groups uniformly and vehemently oppose biological monitoring for compliance purposes. OSHA views this opposition seriously since workers would be the subjects of a compliance program based upon biological monitoring and their participation in such an invasive process would be crucial.

5. Industry's arguments that biological monitoring is preferred due to lack of an air lead-blood lead relationship are unsubtantiated. OSHA believes there is no doubt that an air to blood relationship exists and is best described in the CPA application of the Bernard model.

6. Although both biological and air monitoring are subject to errors OSHA believes that the uncertainties

associated with either measurement is not a sufficient basis for choosing one technique over the other. OSHA recognizes there are errors associated with air sampling but nonetheless believes that evaluation of the plant environment is best and most directly accomplished through a comprehensive industrial hygiene survey as compared to biological sampling.

7. The record indicates that there are currently a significant number of industries who carry out biological monitoring. Given the current distribution of high blood lead levels throughout industry and the admitted lack of compliance with the current air lead standard, OSHA has concluded there is little or no basis for accepting the asserted success of future biological monitoring.

8. OSHA is concerned that a biological standard could impact negatively on workers with high blood leads and extended job tenure. Employers might terminate employment of these individuals to avoid citations for overexposure to lead. In addition, an employer could attempt to circumvent the standard by using respirators rather than implementing engineering controls. The use of respirators is not a satisfactory method for compliance. However, indiscriminate use of respirators would be a confounding factor in ascertaining successful compliance with the standard.

Based on these considerations OSHA will rely on a PEL which utilizes determination of air lead levels to ascertain compliance. The rest of this section will discuss OSHA's decision in detail.

a. *Environmental versus biological monitoring.* The record of these proceedings indicates virtual unanimity of the view that both biological and environmental monitoring should be required in the final standard. Grover Wrenn testified on this issue at the outset of the hearings.

> To protect employees against the myriad of health effects of lead exposure, it appears necessary to establish a comprehensive air and biological monitoring program. (Tr. 32.)

Mr. Knowlton Caplan, engineering consultant and witness for AMAX, made a similar statement:

> The thing that doesn't appeal to me is this either/or (either air leads or blood leads), we either have to do it this way or that way. Why don't we use both tools for what they are best for is my feeling. (Tr. 5739.)

Dr. Jerome Cole, testifying on behalf of the Lead Industries Association, stated, "First regarding monitoring, as I have already noted, both OSHA and LIA agree that the new standard should require all employers covered by the Act to conduct both biological monitoring and environmental monitoring." (Tr. 2997.) Similar testimony was presented by most other witnesses, including those appearing on behalf of Government agencies (NIOSH 1330-31), industry (AMAX 1703; Cominco 2226-28; Cole 3167-68; Caplan 3866; Globe Union 4312; General Battery 4551) and labor (Teamsters 2203-04; McBride 2961-62, 2973; Woodcock 5040).

The fundamental difference of opinion was on which technique, biological or environmental, would OSHA rely to determine compliance with the standard. That is, would OSHA establish an air level standard which would be enforced by environmental monitoring, or a blood level limit which would be enforced by biological monitoring.

There has been such intense controversy over this issue that prior to discussing the basis of OSHA's decision a basic framework of the role of monitoring in general needs to be addressed. This final standard requires that engineering controls and work practices be used to control employee exposure to inorganic lead. The final standard allows joint use of engineering and work practice controls. Respiratory protection may be used only during the time period necessary to install engineering controls, where engineering controls may be inappropriate such as during some maintenance operations, or in those cases when both engineering controls and work practices do not succeed in reducing exposures below the permissible exposure limit.

This compliance strategy has been OSHA's policy consistently and has been followed in prior standards. This policy is based upon the view that the most effective means of controlling employee exposure is to contain emissions of toxic substances at their source through the use of mechanical means combined with work practices.

This policy is consistent with the traditional principles for controlling the occupational environment. These principles are based on the proven conclusion that reduction of exposure to toxic substances is maximized when controlled by the techniques of substitution, isolation, and ventilation. Not all of these basic control principles are applicable to every form of hazard, but all occupational hazards can be controlled by the use of at least one of the principles. It is in this context that OSHA views the discussion of air level versus blood level as the primary compliance mechanism.

Given that compliance with the standard is to be achieved through the use of control technology, it is incumbent on OSHA to specify its view of the role of biological and environmental monitoring as carried out by the employer. The purpose of environmental monitoring is threefold. First, it enables the employer to determine if he is in compliance with the PEL and, if not, to determine the sources of emision which will enable him to achieve compliance through implementation of engineering controls. Second, monitoring during implementation demonstrates progress being achieved, and third, it enables the employer to determine on a continuing basis the adequacy of the controls.

In general, biological monitoring in the context of a medical surveillance program is not designed to be the method for controlling the occupational environment as a whole. It is intended to focus on the health status of the individual worker rather than an employee population and is therefore intended to act on individual problems associated with exposure to lead. While the PEL is intended to protect the entire working population, there will always be some individual variation which needs to be followed through a medical surveillance program. For example, in the short term a particular worker may develop a higher blood lead than predicted by the standard because of nonoccupational exposure, differences in work habits, and other individual differences. While the number of persons with such blood lead differences is small and represents short-term problems, they must nonetheless be addressed and corrected to avoid their becoming chronic, long-term problems.

Implementation of engineering controls represents the long-term solution to occupational lead exposure in the workplace for all employees and medical surveillance/biological monitoring represents short-term solutions to acute problems of individual employees. Biological monitoring in OSHA's view is not a technique that is useful as the primary means of determining compliance with a standard. Biological monitoring is not a measure of the control of lead exposure, rather it only provides information on the results of exposure and is therefore after the fact. The only satisfactory means of determining compliance with the standard is through the use of environmental sampling which directly measures adequacy of control technology. The basic problem with the LIA proposal is that biological monitoring is not feasible as a primary means of determining compliance with a standard which requires implementation of engineering controls.

Due to individual variation both in the short and long term the blood lead level in an individual worker would not necessarily be indicative of the environmental controls in a particular plant. Based upon analysis of the adverse effects associated with exposure to lead, OSHA believes PbB levels should be kept below 40/μg/100 g.

However, the likelihood of achieving this level through engineering controls is limited by feasibility constraints. OSHA does believe that controls can be implemented which will lower the PbB levels of more than 70 percent of all lead exposed workers to below 40/μ"g/100 g. In fact, many industries will be able to achieve an even higher percentage of workers below 40/μ"g/100 g.

Based upon the CPA application of the Bernard Model, OSHA has calculated that when compliance with the PEL is achieved, 0.5 percent of lead exposed employees will have a PbB greater than 60μ"g/100 g, 5 percent will be between 50 and 60μ"g/1m^3 and 23.3 percent will be between 40 and 50/μ"g/100 g for a total of 29.3 percent. The mean PbB for the entire population will be approximately 35 μ"g/100 g. Assuming compliance with 100μ"g/m^3 the percentages would be: greater than 60, 2.6; 50-60, 14.4; and 40-50, 33.7 for a total of 50.7 percent. OSHA will require that blood leads be kept below 60μ"g/100 g or the worker will be removed from that job to one in which the air lead level is below 30μ"g/m^3 (see MRP section). When full compliance with 50μ"g/m^3 is achieved, the number of employees whose PbB is above 60μ"g/100 g will be virtually eliminated. However, there will be a sizable number of workers above 40 at 50μ"g/m^3 (29.3 percent) and there would be at 100μ"g/m^3 (50.7 percent). In other words, at any air lead level there will be a distribution of PbB levels within a given worker population, not a discrete PbB level. Although OSHA would prefer to keep all PbB levels below 40μ"g/100 g the agency is limited by feasibility constraints; however, OSHA has concluded that full use of control technology would yield the distribution cited between 40 and 60μ"g/100 g. Therefore there is no discrete blood lead level which could serve as the PEL for an entire working population.

Given the distribution of PbB levels at compliance due to individual variation, it would be almost impossible to obtain information as to the plant environment in general and the adequacy of controls in particular based on individual PbB levels obtained by a compliance officer. The evaluation would be aided by monitoring results obtained by the company but even then the usefulness of the results would be questionable. This would be particularly true in smaller plants, for as the number of employees diminishes, the statistical base is narrowed, thus reducing the meaningfulness of the monitoring results. On the other hand, a comprehensive industrial hygiene survey performed by a competent, professional OSHA industrial hygienist will more accurately ascertain the levels of compliance and the sources of exposure.

The population distribution of blood lead levels at any one time reflects not only present lead exposure but also exposure which has been experienced over the entire period of the work population's history. The period from the effective date of the standard until the population reaches its ultimate exposure equilibrium is defined as the short run time frame. The period after the population reaches its ultimate exposure equilibrium is defined as the long run time frame. OSHA predicts that eventually 0.5 percent of the lead exposed employees will have a PbB level greater than 60 μg/100 g.

Ultimately, all workers whose PbB levels rise above 60 μg/100 g will be removed under the medical removal protection provisions of this standard. At no time should any worker have a PbB greater than 60 μg/100 g. PbB levels will range from very low numbers to the fifties, and therefore the concept of biological monitoring for compliance purposes is meaningless, since the preferred value of 40 μg/100 g cannot be achieved because of feasibility constraints and because OSHA expects that even with the optimum controls 30 percent of all workers will have PbB between 40 and 60 μg/100 g. There is no precise PbB which reflect implementation of controls. The percentage of workers whose PbB levels are between 40 and 60 μg/100 g will vary depending upon the air lead level, but OSHA finds it is not feasible to use variation in population distributions as the mechanism for determining compliance through biological monitoring at individual firms. OSHA will therefore rely on an air lead level permissible exposure limit as the primary means of determining compliance with the standard.

Biological monitoring is also not a feasible method for determining compliance because it does not take job tenure into consideration, that is, it assumes an equilibrium situation. For example a new population of workers who have had no prior exposure to lead who begin work in an environment where there is exposure to lead will not be at equilibrium. Their blood leads will rise until equilibrium is reached. However, prior to equilibrium their blood leads would have been lower and high exposure conditions would not have been identified if the OSHA compliance officer had relied on biological monitoring. For example, if a worker with no prior exposure had a lead intake of 0.2112 mg/day (184 μg/m^3) his blood lead level (using assumption C—Bernard model) would rise as follows: 6 months—49 μg/100 g; 1 year—55 μg/100 g; 2 years—60 μg/100 g; and 4 years—68μg/100 g. If OSHA had established 60 μg/100 g as a compliance level (assuming no MRP removal) this particular worker would not exceed it for 2 years although his exposure would have been excessive from his first day of employment. Biological monitoring for compliance would be ineffective in this example whereas an industrial hygienist could have pinpointed the problem on the first day of this worker's employment.

In general, blood leads do not reflect the body burden of lead. Blood leads are rather a measure of absorption. Blood leads do not provide a good measurement of body burden. Under questioning by Dr. Bridbord, Dr. Hammond agreed:

> It is true that the longer a person is exposed, the more lead is stored in the body for any given blood lead.

In other words, he said:

> A given blood lead level, maintained for 6 months, will give you a considerable lesser body burden than the same blood lead maintained 6 years or 20 years. (Tr. 292.)

Citing the work of Dr. Chisholm, Dr. Bridbord added:

> The blood lead would underestimate the amount of lead in the body, particularly at high blood lead levels (Tr. 292).

A paper entitled "A More Rationale Basis for Air Sampling Programs" by S. A. Roach in the American Industrial Hygiene Association Journal (January-February 1966) acknowledges the need to know body burdens of substances that are retained in the body in order to know more about the effect on the individual. While one might ordinarily think that a biological determination would provide a better clue as to body burden, this paper states otherwise:

> Although the body burden by itself is not necessarily an adequate indication of whether a worker is affected by a contaminant, it is better than air concentration. This *does not mean* that the analysis of body excretions or blood is preferable to air analysis. Even with those few substances where biological samples can be useful indictors of absorption of a contaminent, it is arguable that *an intelligent appraisal of accurate air sampling results might be a better guide to body burden.* (Emphasis added.)

Biological monitoring in the above example would have detected the worker's overexposure, after 2 years that is, 2 years after the fact. OSHA believes this is morally, practically, and scientifically indefensible. A worker should not be required to wait until the blood lead level becomes excessive before action is taken. This point was stressed during the hearings by numerous participants, Lloyd (Tr. 4700-4703), Wolfe, "Biological monitoring provides evidence of injury already done to the worker" (Tr. 4168), First (Tr. 2312) and Stewart:

I would not put my primary emphasis on biological monitoring because of the simple reason that that is after the fact (Tr. 2608).

If by air monitoring we can determine that we have a fault within the system, we may very well be able to correct the system long before there is a response by the body (Tr. 2610).

OSHA should not adopt a compliance strategy which might cause a worker's loss of employment. In the example cited above it is entirely possible that this worker might be laid off and a new, unexposed worker hired as the former worker's blood lead exceeded 60 μg/100 g in an effort by the employer to avoid a citation and to enable him to avoid engineering controls. None of this would be possible through the use of an air standard. In OSHA's view biological monitoring for compliance purposes is more easily used to circumvent the requirements of the standard. OSHA is not suggesting that a majority of employers would follow such a course but even if these problems arose in a very few cases they could be avoided by use of an air lead standard. OSHA should not adopt a compliance mechanism which might be circumvented, if only by a very few employers, when there is a better alternative available. Thus, OSHA reaffirms its decision to use air lead monitoring as the basis for compliance given the infeasibility of and the moral and practical weaknesses of a biological standard.

When considering the issue of an air lead standard versus a biological lead standard, OSHA has given consideration to the testimony of the participating unions since a high percentage of employee participation would be necessary for the success of such a invasive procedure as biological monitoring. Union representatives adamantly opposed the use of biological monitoring for compliance purposes. Dr. Lloyd testified as follows for the United Steelworkers of America (USWA):

The United Steelworkers of America takes the position that the only reasonable approach to the control of lead exposures is at the source, and the only measure of success in that regard is the extent to which environmental exposures are decreased. To suggest that the control of the lead hazard in a working environment should be assessed by measuring the intake of this poison by the worker is inconsistent with good industrial hygiene practices and is an invasion of the worker's person which should not be considered, except as an only choice approach (Ex. 154A).

Ms. Claudia Miller, representing the USWA, testified similarly:

I know of no one who would seriously argue that exclusive reliance should be placed on either air sampling data or blood lead measurements. Both have their usefulness and both should be employed.

Just as important, both have significant limitations and, therefore, disadvantages. Rather than tiresomely arguing the relative merits of each, the participants at these hearings should try to arrive at some agreement as to exactly what it is that each of these measures tells us and what limitations each has.

Then, based upon this knowledge, employ that information from both air and biological measurements to trigger actions appropriate to each.

The most important reason why we expect OSHA to adopt an air level as its primary standard is that the very idea of workers forever having needles stuck in their arms in order to determine whether the company is complying with an OSHA standard sickens us.

It has been argued that blood leads should be the primary compliance tool because any air lead standard would merely be based on a poor correlation with blood leads. If the company had taken an air sample on a worker for every time they stuck a needle in his arm, we might have more data on which to base an air standard. (Tr. 4715-16.)

These views were supported by testimony from the AFL-CIO (Ex. 149), United Auto Workers (UAW) (Tr. 5040, 5291, 5298-9), the Oil, Chemical and Atomic Workers (OCAW) (Tr. 1032-3), including Drs. Epstein (Tr. 1052-4) and Johnson (Tr. 1094) who testified for OCAW, and the International Brotherhood of Teamsters (Tr. 2055-6, 2090-1). Dr. Epstein testified:

As the OSHA standard proposes, permissible exposure must be primarily based on environmental monitoring. Biological monitoring, including blood lead determinations and the sensitive and practical indices of toxic responses such as the zinc protoporphyrin test is a useful ancillary for the determination of biological responses to occupational exposures. The role for biological monitoring reflects the fact that current environmental monitoring doesn't discriminate between the effects of chemical composition and particle size on lead availability. Environmental monitoring also doesn't reflect possible incremental exposures from ingestion in the workplace. On the other hand, blood lead levels reflect only relatively recent exposures and take appreciable time to equilibrate following such exposure. Even for this purpose, the validity and sensitivity of blood lead determinations has been recently challenged. Blood lead levels also reflect incremental nonexposure—nonoccupational exposures.

Additionally, serious questions have been recently raised as to the reliability of blood lead measurements in the absence of careful and meticulous quality control procedures.

The poor performance of commercial laboratories with regard to blood lead testing is now a matter of record. Contrastingly, the recently developed ZPP test is highly sensitive, provides immediate results, and can be performed by relatively unskilled personnel and furthermore is highly economic and practical.

Finally, blood lead levels underestimate exposure and lead body burdens at higher exposure levels particularly above 50 micrograms per cubic meter, at which level the air blood relationship curve begins to flatten out. Furthermore, as demonstrated in children, the total body burden of lead increases exponentially with arithmetic increases in blood lead levels, particularly above approximately 40 micrograms per 100 grams as stated by Chisholm in his 1976 publication, and this figure of 40 micrograms per 100 grams corresponds to an air lead level of about 50 micrograms per cubic meter. Thus, a fourfold increase in blood lead was associated with a tenfold or greater increase in chelatable lead. In other words, blood lead is a very very inaccurate and insensitive indication of body lead determinations—of body burden determinations.

Mr. George Becker of USWA in his testimony quoted from a decision by the U.S. Court of Appeals, Eighth Circuit, opinion which rejected an appeal by ASARCO and which discussed the issue of biological versus air monitoring. (Ex. 168.) We quote from the opinion:

The Petitioner's second major contention is that it has instituted many protective measures that prevent the likelihood of harm to the employees. We agree with the Administrative Law Judge that Petitioner's program has not reduced the likelihood of serious physical harm.

Most important in the Petitioner's view is a reliance on a biological monitoring program, which involves the testing of each employee's blood and urine to determine the concentration of lead. Dr. Nelson, the Petitioner's Director of Environmental Sciences, stated that this testing is "a far more effective way of securing the safety of employees." Dr. Kehoe prefers biological monitoring, since air measurement "is not a standard which we regard as crucial in relation to the individual." For as yet unexplained reasons, differing individuals can be exposed to higher amounts of lead without becoming ill. The candid Dr. Kehoe, however, had this exchange with the Administrative Law Judge:

(Administrative Law Judge): "Which procedure, Doctor, in your opinion would most greatly detect a change in the lead environment of a workplace, biological sampling or air sampling?"

(Dr. Kehoe): "Either one. I don't know that there is too much to choose from in this. But what I, as a physician am concerned with is John Doe."

Although a carefully conducted biological monitoring system might prevent the likelihood of lead poisoning harm to employees, we think it was more than reasonable for the Secretary to rely on the effective and efficient air sampling method. In addition, the disadvantages of the biological sampling system are demonstrated in this case. About 10 percent of employees tested from 1970-71 were found to have unsafe levels of lead concentration in their blood and urine, yet generally these employees were not tested frequently enough, according to petitioner's expert testimony, to ascertain whether they should be changed to another working area in the plant. In fact, the plant manager had no direct involvement with the monitoring plan. The plant's physician did not "follow up" with what happened to individual employees who had high concentrations of lead in their blood and urine. Further, disruption of employees' working habits and the plant operation would result from transferring employees to new positions within the plant where exposure would be lessened. Most significantly, the record does not indicate that any employee was transferred due to high levels of lead concentration discov-

ered by biological monitoring. The biological monitoring did not eliminate or even reduce the hazard; it merely disclosed it. Although testing of the blood and urine is the most important test for each individual, the use of air sampling tests is the most efficient and practical way for the Secretary to check for a hazard likely to cause death or serious physical harm to workers as a group. We think it also the most efficient manner for the employer to check the existence of a hazard. We do not intend to minimize the importance of the biological monitoring program; obviously, medical examinations of the individual are the most signficant manner of assuring safety of the individual worker, provided that remedial steps are taken at the first indication of a hazardous concentration of lead accumulating in the blood and urine of a worker. The evidence showed that each human responds differently to exposure to lead. Yet we think that the safety of the workers and the practicality of detecting unsafe levels of airborne lead concentrations are best served by the air sampling method. Workers should not be subjected to hazardous concentrations of airborne lead; biological monitoring should complement an industrial hygiene program for clean or at least safe air, but is not a substitute for a healthful working environment.

LIA responded to this contention in their post-hearing comments (Ex. 335):

The petitioner in that case, when charged with exposing its employees to hazardous airborne concentrations of lead, argued that air sampling was inferior to the company's periodic biological testing program. Both the Court and the administrative law judge disagree with this argument, but they did so primarily because the particular health program under challenge had "not reduced the likelihood of serious physical harm." 501 F. 2d at 514. As the Court observed, * * * the plant manager had no direct involvement with the monitoring plan. The plant's physician did not "follow up" with what happened to individual employees who had high concentrations of lead in their blood and urine. * * * Most significantly, the record does not indicate that any employee was transferred due to high levels of lead concentation discovered by biological monitoring. Id. at 514-15.

By comparison, the biological monitoring programs contained in both the Proposed Standard and in the Association's alternate proposal would rectify these shortcomings, since both would ensure that the appropriate "follow up" did occur and that the hazard to the employee was not only detected but also prevented and eliminated.

OSHA rejects this argument by LIA since it is speculative and contrary to existing evidence and experience. OSHA (and presumably in this case the Eighth Circuit) must base its decision not on what is asserted to be possible in the future but rather on the history of the industry's effort to achieve compliance. The evidence demonstrates a poor record of compliance which LIA itself acknowledges.

Since few of the major segments of the lead industry appear to be in compliance with the existing standard of 200 $\mu g/m^3$, * * * (Ex. 335)

Given the fact that the act has been in effect for 7 years this record of compliance raises serious doubts about the contention that OSHA should rely on the asserted success of a future biological monitoring program.

Mr. Leonard Woodcock, then President of the UAW, testified during the hearings and stated the position of his union:

One. The standard should rely primarily on airborne lead measurements to enforce lead exposure control. The UAW supports the proposed standard requirements that both environmental and biological measurements should be made to evaluate exposure in lead operations but that environmental exposure should be the basis for enforcement. The standard should not be enforced by blood lead criterion. The practice of using the worker as a monitoring device means not only polluting the work environment up to a limit, it means polluting the worker's themselves.

Lead should be treated as other occupational health hazards with adverse health effects to exposure. I directly relate it to the chemicals concentration in the air. In the case of lead, the blood lead measurement has been a useful tool in evaluation. However, the environmental index must not be discarded because in this case, a biological index is useful. (Tr. 5040)

Even if OSHA agreed with the LIA proposal for reliance on biological monitoring, OSHA would hesitate to force this technique on employees who so vehemently rejected its proposal during the hearings, especially given the invasive nature of the technique. OSHA finds impractical a compliance technique which workers consider an invasion of privacy especially when another method exists which OSHA has concluded is a superior measure of a plant environment's condition.

There is no guarantee that lead exposed employees will participate willingly in biological monitoring for compliance purposes. Many workers could consider this an invasion of privacy on religious, philosophical, or other grounds and refuse to participate. There are numerous objections to putting the burden on the worker. NIOSH addressed this in testimony:

In other words, you are testing to find out whether a company is in compliance, and putting the burden of that test on the worker. I might add that that test is not without risk.

I think it goes against industrial hygiene practice, been established over many many years.

Dr. BRIDBORD. I think it would also present difficulties in the case where a given worker for religious or other reasons might not want to have a blood sample taken, then that whole system begins to break down, where at least in the air monitoring side, you have an opportunity to monitor frequently and catch problems as they might arise there.

Mr. WAGNER. It also brings up the issue of requiring the worker to take a blood test for OSHA's purposes. Suppose the person refuses to take the blood test? I think it would be pretty flimsy ground to build a compliance program around the voluntary action of the workers to submit themselves to blood tests. (Tr. 1454-5.)

In addition to these objections, biological monitoring for compliance purposes could lead to intimidation and coercion. A worker could refuse a PbB test by OSHA for fear of his job, especially those workers of long tenure, or for fear of the impact on a marginal firm. There are protective legal mechanisms available to employees in these circumstances but OSHA is hesitant to adopt a strategy which may be opposed by the very persons it is designed to protect and who are crucial to its implementation. Under the Act the burden for a safe and heathful workplace is on the employer, not the employee. OSHA believes a strategy designed to determine adequacy of engineering controls is more in keeping with the purposes of the Act.

LIA argues that "Employee resistance (to biological monitoring) is almost nonexistent, apparently for the obvious reason that the workers know that the best way to stay healthy is to have regular check-ups." (Tr. 3081-82; 6464-651 exhibit 248 at 4.) OSHA believes this statement is entirely without medical foundation and believes that the best way "to stay healthy" is to have exposure to airborne lead minimized. The statement implies an after-the-fact approach which is especially dangerous when one considers the health effects associated with exposure to lead. A "check-up" is an inadequate means of ascertaining a disease process since often the disease is either silent until end stages are reached or only detected by very sophisticated techniques, e.g., behavioral testing and MNCV for neurological disease, GFR for urinary disease, or sperm evaluation. As OSHA has repeatedly stated, the object of this standard cannot be to prevent the most onerous aspects of disease, rather, it is the intent of the agency to prevent diseases from lead at the earliest point feasible. Reduced exposure through implementation of engineering controls form the only satisfactory means of control. Biological monitoring and physical exams are most useful in detecting particular acute problems, not in preventing chronic disease. While PbB levels are important in a properly managed biological monitoring program they are largely remedial and must be understood as such.

In addition, the Lead Industries Association argued that there were other reasons why a biological rather than an environmental standard should be used for enforcement purposes:

(1) OSHA's proposal rests on the assumption that particular blood-lead levels can be correlated with and predicted from particu-

lar air-lead concentrations. The evidence presented at the hearing establishes conclusively that this assumption is incorrect and that continued use of a single air-lead number for enforcement purposes will not accomplish the intended goal of protecting the individual worker's health.

This point has been addressed in the section on air to blood relationships and will not be addressed here. The fundamental problem appears to be LIA's misunderstanding of the value of the cross sectional studies in the record, the need to address populations rather than seek an individual air to blood correlation and the need to consider confounding variables of job tenure, particle size and others rather than assuming there is not an air to blood relationship.

A brief discussion of the existing studies on the air to blood correlation is relevant here. As summarizations of available data on different populations, the existing studies are reasonably valid. It is one thing to say, however, that a linear relationship was observed between the blood lead levels and air lead exposure at a given level of statistical significance, for a given sample of workers, and another thing entirely to use the observed relationship to predict the effect of lowering air lead exposure on even that same sample of workers, let alone to generalize to other samples. Generally, one must be rather conservative about using cross sectional data, obtained at a given point in time, to predict effects over time. Rarely can it be predicted for certain, that in this kind of situation, all other factors will be held constant.

Recognizing these limitations by no means should be taken to imply that the data are useless or that no reliable relationship exists between long-term air lead exposures and blood lead levels. To the extent that the likely systematic errors in the short-term cross sectional studies are understood (e.g., underprediction of blood lead-air lead slope coefficient and overprediction of the intercept at zero occupational exposure), the observed regressions can be used to bound estimates of the true long-term relationships of blood lead to occupational air lead exposure. To the extent that the sources of uncontrolled variation within and between studies are understood, estimates of the likely effects of such factors can be explicitly incorporated into a more comprehensive description of the general system.

Because of the difficulties and deficiences in observational studies of air lead-blood lead relationships, it is useful to supplement the empirical air lead-blood lead correlations with relationships derived from physiological models of lead transport in the body. It is concluded that the weight of the evidence demonstrates that the CPA application of the Bernard model is likely to be an accurate tool for assessing the blood lead level response to alternative air lead exposure.

OSHA has concluded that not only is there a definite air to blood relationship but that relationship is most closely approximated by application of the Bernard model with its inclusion of job tenure and particle size.

LIA's second argument was stated:

> Air sampling is subject to enormous errors, does not measure total exposure and absorption, and cannot reliably be used to indicate specific sources of emissions. Although environmental monitoring serves an important function in helping to evaluate the effectiveness of and need for engineering controls, it is neither the most effective nor the most direct indicator of employee risk. Biological monitoring, by comparison, both identifies and makes it possible to prevent overexposure from all sources, whether occupational or nonoccupational; it detects potential hazards which may exist even when air-lead levels are extremely low; it provides an enforcement mechanism which would significantly reduce OSHA's administrative burdens and would allow OSHA to enforce the standard more easily.

During the hearings there was considerable testimony which addressed problems associated with measurement of both air and blood leads. OSHA agrees that there are problems associated with any measurement which could affect both the accuracy and the precision of the determination, but does not believe that the drawbacks in both should be the basis for choosing one technique over the other. Since all parties agreed that both techniques were useful and should be employed, it should be obvious that both will be used but in different contexts. As stated earlier in this section environmental and biological monitoring are used for different reasons to achieve different ends and must be viewed in that context. While there are obvious errors associated with an industrial hygiene survey (environmental monitoring) it is the only means available to evaluate a plant environment. The industrial hygiene survey will determine the sources of emission and efficacy of existing controls, the progress made during implementation of new controls, and finally the success of full implementation. From then on environmental monitoring will locate problems as they arise. In order to completely evaluate the control technology in particular and the plant environment in general a comprehensive industrial hygiene survey must be carried out.

Biological monitoring will not accomplish this task. Since OSHA places implementation of engineering controls as the highest priority in the control of toxic substances, it must choose environmental monitoring as its method of determining compliance with the standard to assure a direct relationship between the knowledge required (engineering controls) and the measurement (environmental monitoring-industrial hygiene). Use of a PbB level as a measure to determine compliance would be indirect and would not be useful since there will be significant variation in blood lead levels for any particular individual.

OSHA has reviewed the Summary Report on Proficiency Testing of blood lead for 1976, and has found the results disturbing. The agency agrees with NIOSH that:

> Perhaps the most frequently employed measure of lead absorption into the body is the quantity of lead in the blood. Most clinical measures of lead toxicity have been related to blood lead measurements. One of the greatest difficulties with the measurement of blood leads is the high level of skill required in analytical techniques and the great care demanded to avoid the risk of sample contamination by lead or of lead loss. The proficiency record of laboratories in blood lead determinations has at times been less than adequate as shown by a recently completed Center for Disease Control (CDC) study of commercial clinical laboratories. It was disturbing to find that only one-third of all commercial laboratories in this study performed acceptably * * *. A copy of the commercial laboratory proficiency testing study is being submitted for the hearing record. The poor record of commercial laboratories on blood lead testing is but one of the reasons why NIOSH opposes setting an occupational led standard based solely upon blood lead levels. (Ex. 84, pp. 7.)

In addition other witnesses expressed concern with laboratory accuracy. (Tr. 1647, 1675, Ex. 335, page 5, Ex. 452, p. 52, 61, Tr. 7587-92.) These concerns have led OSHA to address this issue in the biological monitoring section of medical surveillance, and the agency assumes the additional requirements will suffice to improve the record of laboratories. Until such time as the evidences of accuracy demonstrates otherwise, OSHA cannot base its PEL on a biological indicator.

OSHA recognizes that a professional, thorough industrial hygiene survey is required to adequately evaluate the plant environment. It has therefore developed sophisticated training for its compliance officers in the evaluation of a plant environment and requires each compliance officer to carry out extensive observation of the workplace (chapter 1, Industrial Hygiene Field Operations Manual). This is the proper method required to address shortcomings in environmental sampling rather than adopting a method which is not adequate to provide the information necessary to determine compliance.

Biological monitoring will enable the employer who has come into compliance with the PEL, housekeeping and personal hygiene requirements of the

standard to identify workers with improper work practices since these workers may develop elevated PbB levels from ingestion. This is precisely the value of biological monitoring especially when all the provisions of the standard are complied with including the training and education aspects. OSHA recognizes the need to avoid lead exposure from eating and smoking in exposed areas, the biting of fingernails, carelessness in handling lead materials or from other inadequate work practices although there is debate on the significance of this means of absorption. (Tr. 352, 3104, 3260, 6475.) The agency will not attempt to address this debate in detail since OSHA believes that debate is moot. Control of airborne lead with good housekeeping, change of clothes, showers, and proper hygiene in general, along with education and training of workers, should for all practical purposes virtually eliminate this method of entry.

The importance of control of airborne lead cannot be underestimated since the potential for ingestion of lead arises from airborne lead having settled onto surfaces. One could argue that essentially all lead exposure arises from airborne lead except where the worker comes in direct contact with it at his work station. Nonetheless, for the few workers who will still have a problem, biological monitoring is especially suited for detection of elevated PbB levels. These points are also relevant to the individual whose PbB level is elevated from off-the-job activities and nonoccupational exposure.

There is a practical problem associated with compliance by biological monitoring which further renders it questionable as a compliance strategy. LIA argues that OSHA would not need to conduct biological monitoring but could rely on employer's monitoring records to determine compliance. Unfortunately, OSHA cannot rely on the good faith efforts of employers in all cases and unions would be unlikely to accept this as a basis for citations. (Ex. 343, p. 61.) This would then require OSHA to conduct biological monitoring to ascertain compliance with the standard. The problem is clearly surmountable but it would create a hardship for an agency with no trained medical personnel.

During the hearings there was support for an air lead standard from numerous witnesses. (Tr. 655, 2312, 2804, 2972-73, 4127, 5738-9, 5980-2.) NIOSH wholly supported establishing a PEL based upon air lead levels:

Mr. BELICZKY. Mr. Baier, in the absence of a standard on worker exposure levels and sole reliance on blood lead determination, do you feel that such an approach would encourage industry to initiate, on a voluntary basis, engineered controls to reduce exposure and thereby decrease absorption?

Mr. BAIER. It is difficult to say. Certainly some companies probably would, but I don't know where they would start, just based on a blood lead. I mean, if that was the sole source of information, as we pointed out before, it simply tells that you have been overexposed. You don't know the source of exposure. I don't know what the incentive would be.

Mr. BELICZKY. Does anyone else care to respond to that?

Dr. BRIDBORD. Yeah, my personal feeling would be that that would tend to be a disincentive toward the development of engineering controls and it would tend to try to put reliance more on personal protective devices and other administrative controls to keep a lid on the blood leads which we feel you know, are not as assured and not as good as having control of lead at the source. I think it would decrease the emphasis on the engineering controls. I am sure that responsible companies would still develop good engineering controls, but I think in a general way. It would be a disincentive.

Mr. BELICZKY. Do you feel that a promulgated standard, based solely on blood lead levels, would encourage the indiscriminate use of respirators?

Dr. BRIDBORD. I think that it has that potential. Yes sir. (TR. 1457-8.)

OSHA considers the point of Mr. Beliczky and Dr. Bridbord extremely important; that is, how a biological standard would be enforced. Given the definition of feasible controls in the LIA proposal and the history of lack of compliance by the industry, a particular firm might choose to keep PbB levels low through use of respirators rather than through implementation of controls. OSHA doubts the efficiency of such an approach given the shortcomings of respirators but such an approach would nonetheless be confounding to an effective enforcement program based on biological monitoring. The LIA proposal appears to suggest that some forms of personal protective equipment may be part of "feasible" controls when they suggest as a guideline "the availability and relative effectiveness of other means of protecting the workers." (Ex. 335.)

OSHA is in complete agreement with Dr. M. Furst who has concluded:

In my opinion, biological tests for lead absorption should be employed only for the differential diagnosis of illness and not as a means of routine evaluation of an engineering control program. This is because biological manifestations of poor lead-in-air control occur late in time, cannot be correlated with specific events of malfunctioning devices, and are difficult to interpret as a result of the great variability of human response to lead inhalation, its metabolism, and its ultimate elimination or storage. By contrast, personal sampling gives an immediate and very specific assessment of the efficacy of control practices and, when combined with skillfully placed area samplers, can differentiate between personnel and material failures. This makes it possible to take prompt remedial measures and to prevent the occurrence of high concentrations of lead in blood or urine.

Reliance on routine air sampling represents an important input to a conscientious lead control program, because personal samples measure worker exposure directly and precisely and give a quantitative result sufficiently close in time to the events that took place when the samples were obtained to, first, avoid continuing an overexposure (as would be the case where one is using biological monitoring that requires weeks of overexposure before readings reach a level that alerts management to the existence of trouble spots) and, second, identify the offending operations of malfunctioning controls, be they of a human or a material nature.

Personal sampling equipment has improved enormously in reliability and accuracy over the past 6 years and has currently reached such a level of perfection as to generate great confidence in its use on the part of industrial hygienists. The currently recommended practice of selecting the most highly exposed workers for sampling helps to assure that maximum airborne lead levels are being monitored and the NIOSH-recommended statistical interpretation of sampling results provides an important quality control standard for conducting such surveys.

I have laid special stress on: (a) The use of engineering controls in preference to reliance on respirators and personnel rotation and (b) on air sampling in preference to biological monitoring because I believe they are feasible controls of airborne concentrations. Of equal importance is an informed, well trained, and responsible work force because total reliance on machinery, alone, will prove to be inadequate. This, too, reflects well established industrial hygiene experience in every industry. I believe a failure to recognize this important interrelationship between good work practices and good control engineering accounts for the astonishingly high estimates that have been submitted as to the cost of compliance with a 100 $\mu g/m^3$ air standard (Ex. 270, p. 21-23).

For the reasons cited above, OSHA will place primary reliance on its PEL of 50 $\mu g/m^3$ as determined by environmental monitoring.

b. *100 $\mu g/m^3$—The Proposal.* In its proposal OSHA stated its intent as follows:

Our present judgment is that in order to provide the appropriate margin of safety, as well as to provide significant protection against the effects, clinical or subclinical, and the mild symptoms which may occur at blood lead level below 80 $\mu g/100$ g, it is necessary to set an airborne level which will limit blood lead level to 60 $\mu g/100$ g. A maximum blood lead level of 60 $\mu g/100$ g corresponds to a mean blood lead level of about 40 $\mu g/100$ g, will result in a range in workers of approximately 20 $\mu g/100$ g at the lower limits to 60 $\mu g/100$ g at the upper limits. Having determined the maximum blood lead level which the protection of employees and prudence permits, and the corresponding mean blood lead level, it is necessary to correlate these levels to the extent possible with air lead levels in order to establish the permissible exposure limit.

As noted, the proposal would establish a permissible exposure limit for airborne concentrations of lead at 100 $\mu g/m^3$ as determined on an 8-hour time-weighted average based on a 40-hour workweek. It would not

establish as a requirement of the standard maximum employee blood lead levels with which the employer would have to comply, because of the many individual variables involved over which the employer has little direct control, such as poor personal hygiene of employees and off-the-job exposures. However, the correlation between blood lead levels and air lead levels have been used in arriving at the proposed air lead exposure limit because the data indicate that if air lead levels of 100 $\mu g/m^3$ are maintained, the maximum upper blood lead levels of workers should remain below 60 $\mu g/100$ g. (Ex. 2, p. 45938.)

These conclusions were based essentially on two studies, one by Williams and the other by Sakurai et al. OSHA based the proposed PEL on these works even though the proposal noted certain limitations to each study:

Although these data are the best available evidence, they do not precisely define the air lead level within the 50-150 $\mu g/m^3$ range which corresponds to a mean blood lead level of 40 $\mu g/100$ g and an upper blood lead level of 60 $\mu g/100$ g. In these circumstances, we believe it is appropriate to propose for the permissible exposure limit the air lead concentration that falls in the middle of this range, that is, 100 $\mu g/m^3$ as the air lead level which is likely to maintain the upper range of workers' blood lead levels below 60 $\mu g/100$ g. (Ex. 2, pp. 45938-39.)

OSHA has discussed the air to blood relationships in the record in that section and will not repeat those arguments but rather will use the conclusions from the section as it relates to incremental benefits.

Based upon a thorough evaluation of the record OSHA has reached the following conclusions which form the basis for lowering the PEL from 100 $\mu g/m^3$ to 50 $\mu g/m^3$.

1. The health effects data indicate that to the extent feasible blood lead levels should be kept at or below 40 $\mu g/100$ g. This contrasts with the proposal which set 40 $\mu g/100$ g as a mean with 60 $\mu g/100$ g as a maximum. OSHA recognizes that the lack of feasibility data in the record inhibit complete achievement of the goal of 40 $\mu g/100$ g as a maximum but nevertheless it forms an important foundation for OSHA's decision to reduce the PEL to 50 $\mu g/m^3$. In its final standard OSHA has classified blood lead levels as follows:

40-49 $\mu g/100$ g—minimally elevated.
50-59 $\mu g/100$ g—elevated.
>60 $\mu g/100$ g—unacceptable.

2. The Bernard model predicts a mean blood level at 50 $\mu g/m^3$ of 34.6 $\mu g/100$ g assuming compliance with the standard. Similarly, compliance with 100 $\mu g/m^3$ yields a mean of 40.2 $\mu g/100$ g. The distribution of blood lead levels when compliance with 50 $\mu g/m^3$ is achieved may be compared to the distribution at 100 $\mu g/m^3$.

Blood lead level	Percent, workers	
	50 $\mu g/m^3$	100 $\mu g/m^3$
>60 $\mu g/100$ g	0.5	2.6
50 to 60 $\mu g/100$ g	5.5	14.4
40 to 50 $\mu g/100$ g	23.3	33.7
>40 $\mu g/100$ g (total)	29.3	50.7

It is apparent that there is a substantial reduction in the number of workers whose blood lead levels exceed 40 $\mu g/100$ g and whose PbB levels are in the 50-60 $\mu g/100$ g range when the air lead level is reduced from 100 $\mu g/m^3$ to 50 $\mu g/m^3$.

3. The incremental benefits of a 50 $\mu g/m^3$, 100 $\mu g/m^3$ and 200 $\mu g/m^3$ were described in the Benefits Section. We shall discuss the results, first assuming rigorous compliance and second assuming minimal compliance. Both situations would be found to exist in the workplace such that the results in terms of benefits would be mixed, but for these purposes OSHA will address them separately.

For workers whose PbB levels were initially greater than 60 $\mu g/100$ g there will be a substantial reduction from 32,777 to 498 with compliance at 50 $\mu g/m^3$. For 100 $\mu g/m^3$ the benefits are also substantial, 32,777 to 2,562 with the incremental benefit for 50 over 100 being 2,064. There are 22,887 workers whose PbB are between 50 and 60 $\mu g/100$ g. Compliance with 50 $\mu g/m^3$ would reduce that number by 17,514 whereas at 100 $\mu g/m^3$ the number would be 8,846 with an incremental benefit of 8,668 for 50 versus 100 $\mu g/m^3$. Between 40 and 50 $\mu g/100$ g there are 23,898 and compliance with 50 and 100 $\mu g/m^3$ result in a decrease at 50 $\mu g/m^3$ of 10,141 and an increase at 100 $\mu g/m^3$ of 8,972 with a benefit of 50 versus 100 $\mu g/m^3$ of 10,141. Lastly, there are 79,569 workers whose PbB levels are above 40 $\mu g/100$ g. Compliance with 50 $\mu g/m^3$ and 100 $\mu g/m^3$ respectively would reduce the numbers to 28,599 and 49,475 with an incremental benefit of 20,876 for 50 $\mu g/m^3$ versus 100 $\mu g/m^3$.

Summary 50 $\mu g/m^3$ versus 100 $\mu g/m^3$.

INCREMENTAL BENEFIT (BY NUMBER OF WORKERS)

$$\underset{2,064}{>60\ ug/100g} \qquad \underset{8,668}{50\text{-}60\ ug/100g} \qquad \underset{10,141}{40\text{-}50\ ug/100g} \qquad \geq \underset{20,876}{40\ ug/100g}$$

It is apparent from the calculations that the incremental benefits of 50 $\mu g/m^3$ over 100 $\mu g/m^3$ are substantial. Approximately 20,000 workers will have their PbB levels reduced below 40 $\mu g/100$ g and there are substantial benefits in all ranges.

In summary, OSHA finds 50 $\mu g/m^3$ more closely reflects the goals outlined in the proposal of a maximum of 60 $\mu g/100$ g and a mean of 40 $\mu g/100$ g blood lead, and within the limits of feasibility provides substantial incremental benefits toward achieving a maximum of 40 $\mu g/100$ g lead in blood. In light of those conclusions, OSHA has adopted a PEL of 50 $\mu g/m^3$ in its final standard.

c. *The LIA Second Alternative—200 $\mu g/m^3$.* The LIA has proposed that if OSHA decides to retain a single air lead exposure limit as opposed to a standard with primary reliance on biological monitoring, the limit should not be lower than 200 $\mu g/m^3$. They justify this level with the following reasons:

(1) Until OSHA knows whether the health of lead workers can be protected through compliance with the existing air-lead standard, there is no reason to modify that standard.

(2) Reducing air-lead levels from 200 $\mu g/m^3$ to 100 $\mu g/m^3$ would accomplish very little (if any) reduction even in average blood-lead levels, despite the enormous expense and despite the fact that the individual worker would still not be adequately protected.

(3) The proposed environmental exposure limit is economically and, in many instances, technically infeasible and, notwithstanding the minimal health gains, would materially alter and disrupt the competitive market structure which now exists in the major sectors of the lead industry. (Ex. 335.)

The first argument set forth by LIA is perplexing insofar as it argues for inactivity on OSHA's part pending compliance with an OSHA standard already in effect for 7 years. This argument appears to place the burden on OSHA to insure compliance with the standard when the Act clearly places the responsibility to provide safe and healthful working conditions on the employer. If OSHA were to adopt this view no standard could be promulgated pending either compliance with the current standard, clearly a disincentive for industry, and completion of prospective research studies, which could take up to 40 years given the need to study chronic disease development which is associated with a lifetime of work exposed to lead. This proposal places an undue burden on affected employees and is without merit considering the Act's requirement that standard be set "on the basis of the best available evi-

dence." The proposed lead standard and this final standard are based on a careful, thorough evaluation of all information contained in the scientific literature and the rulemaking record. OSHA believes its conclusions are based on solid scientific evidence already in existence and finds no basis for a delay, particulary in light of the severity of the disease processes described herein and the large numbers of workers who continue to be unprotected.

The second point of the LIA argument has been addressed in the Air to Blood Relationship and Benefits sections and need not be repeated. The benefits of compliance with 50 $\mu g/m^3$ versus the current level of compliance with 200 $\mu g/m^3$ are substantial. The number of workers whose PbB levels are greater than 60 $\mu g/100$ g would be reduced from 32,777 to 498 and the number of workers whose PbB levels would be reduced below 40 $\mu g/100$ g is 50,970. To summarize the benefits:

INCREMENTAL BENEFITS

≥60 ug/100g	50-50 ug/100g	40-50 ug/100g	≥40 ug/100g
32,277	17,514	1,159	50,970

	200 $\mu g/m^3$	*50 $\mu g/m^3$*
50 $\mu g/100$ g	22.4	0.5
50-60 $\mu g/100$ g	32.6	5.5
40-50 $\mu g/100$ g	28.7	23.3
40 $\mu g/100$ g (total)	83.8	29.3

INCREMENTAL BENEFITS OF 200 $\mu G/M^3$ VS. 50 $\mu G/M^3$

Even assuming OSHA delayed promulgation of its standard until compliance with 200 $\mu g/m^3$ was achieved the benefits would be substantial. Compliance with 200 $\mu g/m^3$ would yield the following blood lead distribution (in percent):

blood lead	≥50 ug/100g	50-50 ug/100g	40-50 ug/100g	≥40 ug/100g
Number of Workers Removed	13,957	22,575	8,961	45,511

It is important to note that the correct method of determining benefits is to compare a shift in the entire distribution of blood lead levels in the entire population. Comparison of the differences in average blood lead levels is irrelevant to an accurate understanding of the impact of the standard.

The section on feasibility addresses the issues of feasibility set forth in (3) here and will not be repeated. It is sufficient to say that OSHA has found the standard feasible both technologically and economically. For the reasons set forth OSHA concludes that there are substantial benefits to be achieved from promulgation of a 50 $\mu g/m^3$ standard and that the arguments set forth under this are alternative not compelling.

d. *40 $\mu g/m^3$.* The United Steelworkers of America stated their proposed alternative as follows:

> Having concluded that the "safe" level of PbB in blood should be 30 $\mu g/100$ g or less but allowing for a range up to 50 $\mu g/100$ g with appropriate biological monitoring, a Permissible Exposure Level of lead in air must be chosen, consistent with the observed relationship between these two variables for the PbB range of 30-50 $\mu g/100$ g. Referring again to the table, it is seen that the midpoint of this range, 40 $\mu g/100$ g PbB, would be predicted by air lead levels somewhere between 40 and 60 $\mu g/m^3$.
>
> The limited information available to guide us in choosing the "safe" air level leads us to exclude consideration of the values above 40 $\mu g/m^3$. First, the General Motors data reviewed by NIOSH indicates that almost 20 percent of workers exposed to air lead concentrations of less than 40 $\mu g/m^3$ will have PbB levels greater than 50 $\mu g/100$ g (Ex. 86D). Second, considering the range of values expected around mean PbB levels in this range, the upper limit would be greater than 50 $\mu g/100$ g (Ex. 96). Third, as suggested by Epstein, some allowance should be made for a margin of safety (Ex. 68). For these reasons, the United Steelworkers of America recommend that the Permissible Exposure Level be set as the Time Weighted Average of 40 $\mu g/m^3$ of air for a 40-hour work week. It is felt, based on the evidence at hand that enforcement of this level will assure that the blood lead levels for the great majority of workers exposed to lead will be maintained at a level less than or equal to 50 $\mu g/100$ g (Ex. 343 p.78-9).

OSHA has calculated the equilibrium distribution of blood lead levels assuming rigorous compliance with 40 $\mu g/m^3$ and have compared these results to a similar calculation for 50 $\mu g/m^3$. The results are as follows:

	Blood Lead Distribution (%)			
	≥40ug/100g	40-50ug/100g	50-60ug/100g	≥60ug/100g
40 ug/m^3	24.2%	19.9%	4%	0.3%
50 ug/m^3	29.3%	23.3%	5.5%	0.5%

Blood Lead Standard Deviation = 9.5 ug/100g.

OSHA has detemined that the incremental benefit of 40 $\mu g/m^3$ versus 50 $\mu g/m^3$ is negligible. While OSHA agrees with the goal that blood lead levels should be kept below 50 $\mu g/100$ g where possible and in fact preferably below 40 $\mu g/100$ g the air lead level required to assure that all employees achieve latter value are clearly impractical in the foreseeable future. Based on these considerations OSHA believes

the considerations which form the final standard are valid and will be sustained.

C. MEDICAL REMOVAL PROTECTION

As an aid to the readers of this Attachment concerning Medical Removal Protection, the following is a brief Table of Contents:

1. Temporary Removal of Workers at Risk of Sustaining Material Impairment.
2. Medical Removal Protection Benefits.
   a. Introduction.
   b. Medical Removal Protection as a Means of Effectuating the Medical Surveillance Sections of the Lead Standard.
      —Roles played by medical surveillance in the standard.
      —Summary of the need for MRP.
      —Worker fears of adverse economic consequences from participation in medical surveillance programs.
      —Existing industry practices concerning economic protection for workers temporarily removed due to occupational health problems.
      —Impact of the final lead standard on disincentives to participation.
      —Importance of meaningful worker participation in the standard's medical surveillance program.
      —Experience under the Black Lung medical surveillance and transfer program.
      —The scope of the need for MRP.
   c. Medical Removal Protection Benefits as a Means of Allocating the Costs of Temporary Medical Removals.
   d. Alternatives to Medical Removal Protection Considered by OSHA.
      —Mandating that workers participate in medical surveillance.
      —Mandating that temporary medical removals occur only at the election of individual workers at risk of material impairment.
      —Permitting the use of respiratory protection in lieu of temporary medical removals.
   e. Feasibility.
      —Overview of the phasing-in of MRP.
      —Impossibility of immediate implementation of the ultimate MRP program.
      —Immediate 80 μg blood lead level removal trigger.
      —70 μg blood lead level removal trigger 1 year after the effective date of the standard.
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      —Six month 50 μg average blood lead level removal trigger 4 years after the effective date of the standard.
      —Impact of ultimate blood lead level removal criteria.
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   h. Employees Whose Blood Lead Levels Do Not Adequately Decline Within 18 Months of Removal.
   i. Follow-up Medical Surveillance During the Period of Employee Removal or Limitation.
   j. Medical Removal Protection and Worker's Compensation Claims.
   k. Other Credits.
   l. Voluntary Removal or Restriction of an Employee.
   m. Miscellaneous Matters.
      —Personal hygiene and work practice rules.
      —MRP and employee conditions "not the fault" of the employer.
      —Worker abuse of MRP.

1. *Temporary Removal of Workers at Risk of Sustaining Material Impairment.*

The final standard requires that an employer temporarily remove from lead exposure in excess of 30 μg PbA/m³ TWA any worker determined to be at increased risk of sustaining material impairment to health or functional capacity by continued exposure to inorganic lead. A determination that a worker is at increased risk of sustaining material impairment would derive either from the worker's latest blood lead level measurement or from an examining physician's medical opinion. Followup medical surveillance must be made available during the period of a worker's removal from his or her job. And, return of the worker to his or her original job is required once the worker's blood lead level has declined to an acceptable level; or alternatively, once the examining physician's written opinion so permits.

The sole reason for requiring the temporary removal of a worker at risk is to prevent material impairment to that worker's health or functional capacity. Temporary removal of a worker, therefore, is a preventive, protective mechanism. OSHA views this temporary removal mechanism as both an essential and indispensable element of the overall lead standard. The Permissible Exposure Limit (PEL) section of the preamble explains that the PEL of 50 μg PbA/m³ TWA (along with other provisions of the standard) is designed to protect the vast majority of workers from adverse health effects due to inorganic lead exposure. As noted, the PEL of 50 μg includes only a very small margin of safety. Due to this small margin of safety and the wide variability of worker response to lead, some small percentage of the work force, much less than 6 percent, will probably not be protected even by total compliance with both the PEL and other provisions of the standard (e.g., engineering controls, work practices, housekeeping, hygiene facilities, etc). Temporary removal is the only means of assuring adequate protection to this small minority of workers.

As explained in the Feasibility section of the preamble, some segments of the lead industry cannot be expected to achieve total compliance with the PEL through engineering and work practice controls for several years. During this period of time primary reliance will have to be placed on personal respiratory protective equipment as a means of preventing health impairment. Respiratory protection has serious drawbacks, however, and it is to be expected that some workers will not receive adequate protection from respiratory protection alone. Temporary removal where necessary is a means of assuring additional protection to these workers.

The preceding paragraphs explain the two main protective, preventive functions that temporary removal of workers at risk of sustaining material impairment should serve. Temporary removal should protect the small minority of exposed workers which we anticipate will not be afforded adequate protection by total compliance with the inorganic lead standard. Temporary removal should also protect those workers who receive insufficient protection from personal protective equipment. OSHA views these two protective, preventive functions as crucial to the overall success of the inorganic lead standard. And OSHA can think of no alternative protective mechanism, nor has any participant in the lead proceeding suggested an alternative mechanism, which would equally serve these protective functions. Due to the lack of alternatives and the major functions served by temporary medical removal, OSHA views this protective mechanism as an indispensible provision of the inorganic lead standard.

It must be stressed, however, that OSHA does not view temporary removal as and alternative means for employers to control employee lead exposure, but rather as a last-ditch, fall-back mechanism to protect individual workers in circumstances where other protective mechanisms have not sufficed. The standard places primary reliance on engineering and work practice controls, on environmental monitoring, on hygiene facilities and practices, and on education and training as means of protecting worker health. These measures should prove inadequate in only the most unusual of circumstances. Where primary reliance

must be placed on respirators, employers should be able to protect most workers by persistent dedication to maintaining all elements of an effective respiratory protection program. OSHA also anticipates that the majority of affected employers will voluntarily comply with all provisions of the inorganic lead standard such that temporary removal of most workers is unnecessary. In spite of the above, OSHA is convinced that a significant number of workers in the coming years will need the benefits of a temporary medical removal, and the final lead standard includes a mandatory temporary removal provision for this reason.

The record evidence developed in the lead proceeding demonstrates that temporary removal of workers at substantial risk of sustaining material impairment is a protective mechanism recognized by and acceptable to both management and labor. Many lead firms have existing medical survillance programs incorporating the temporary removal of any worker whose medical condition meets specific criteria. (Ex. 157, p. 10; Ex. 158, p. 68; Ex. 389, p. 14; Ex. 401B, p. 15; Ex. 404B(D-1), p. 4; Ex. 404B(D-2), p. 17; ex. 404B(D-4), p. 70; Ex. 404B(D-5), p. 48; Ex. 404B(D-6), p. 34; Ex. 423, p. 23; Ex. 424, p. 12; Ex. 425, p. 6; Ex. 427, p. 58; Ex. 430D(17), p. 37; Ex. 430D(23), p. 12; Ex. 430D(26) (section 8.) The 1975 proposed inorganic lead standard contained provisions which essentially prohibited an employer from keeping any employee at existing exposure to lead if such exposure posed an increased risk of material impairment to health. (40 FR 45934 (1975) (to be codified in 29 CFR §§ 1910.1025(K)(2)(ii)(B)(*2*) and 1910.1025(K)(4)(ii).) Temporary removal was not mandated, but obviously was contemplated as one employer option. The comments and testimony which followed the 1975 proposal raised no substantial opposition to the propriety of temporary removal as a protective mechanism. (*See*, Ex. 3; Ex. 4; Ex. 28.) In September 1977, OSHA through the FEDERAL REGISTER explicitly stated its intention to mandate temporary removal of workers at risk (42 FR 46547 (1977)). Subsequently, both industry and labor representatives readily endorsed the proposition that temporary removal is an appropriate means of protecting workers found to be at risk of sustaining material impairment to health. (*See*, Ex. 354 responses to 42 FR supra, Question 6: "Should employees be permitted to remain on their job despite the risk of material impairment of their health?")

2. *Medical Removal Protection Benefits.*

a. *Introduction.*

The final standard requires that an employer maintain the earnings, seniority, and other rights and benefits of any worker temporarily removed from current lead exposure due to the risk of sustaining material impairment if such exposure continues. OSHA terms this provision of economic benefits "medical removal protection (MRP) benefits," although the phrase "rate retention" has often been used in a generic sense to signify the same form of economic protection. This component of the overall MRP program has been a controversial issue throughout the lead proceeding, and the inorganic lead standard is the first OSHA health standard to incorporate such a broad provision. (See, however, limited MRP provisions in OSHA's asbestos and cotton dust standards. (Asbestos, 29 CFR section 1910.1001(d)(2)(iv)(c) (1977), *Industrial Union Depart, AFL-CIO* v. *Hodgson*, 499 F.2d 467, 485 (1974); Cotton Dust, 43 Fed. Reg. 27350 (1978) to be codified in 29 CFR section 1910.1043(f)(2)(v).) For both of these reasons, the following sections of this attachment discuss at great length the reasons for OSHA having adopted MRP, and the alternatives the agency considered. Attention is then focused on each aspect of the MRP program, and the agency's decisionmaking is explained in depth. OSHA has included this lengthy descriptive process in response to the specifics of industry opposition to MRP that were voiced throughout the lead proceeding. OSHA hopes that employers after reading the agency's explanations will view MRP for what it is—a regulatory device adopted specifically to advance worker health, and a mechanism whose costs are a reasonable and necessary price of doing business.

The final inorganic lead standard contains a MRP provision for two reasons. First, OSHA views MRP as the most effective device for maximizing meaningful worker participation in the medical surveillance program provided by the standard. Second, since temporary medical removal is fundamentally a protective, control mechanism, OSHA has determined that the costs of this control mechanism should be borne by employers. MRP is meant to place such costs of worker protection directly on the industry rather than on the shoulders of individual workers unfortunate enough to be at risk of material impairment to health due to occupational exposure to lead. OSHA views each of these two reasons as independent and compelling reasons for the adoption of MRP provisions.

b. *Medical removal protection as a means of effectuating the medical surveillance sections of the lead standard.*

Roles played by medical surveillance in the standard.—As just noted, OSHA views MRP as the most effective device for maximizing meaningful worker participation in medical surveillance provided by the final standard. Before discussing the need to maximize meaningful participation, it is appropriate to emphasize the crucial roles that medical surveillance will play in protecting the health of workers exposed to lead. Section 6(b)(7) of the Act specifies numerous elements that, where appropriate, must be included in an OSHA occupational health standard. These elements include the requirement of cautionary labels, the prescription of protective equipment and control procedures, the provision of environmental monitoring, and the specification of medical examinations and other biological tests. These elements along with the rest of the OSH Act demonstrate OSHA's mandate to promulgate comprehensive health standards which assure, to the extent feasible, that no worker suffers diminished health, functional capacity, or life expectancy as a result of work experience. Medical surveillance is just one aspect of the act's integrated approach to worker protection, but it is clear that a preventive occupational health program cannot succeed unless all elements accomplish their intended purpose.

Medical surveillance is a crucial component of this occupational health standard since it is the only method of determining whether or not individual workers have been afforded adequate protection. Reliance is placed on primary control measures, such as engineering controls, to minimize worker exposure to lead, but only medical surveillance can determine the effectiveness of these measures in protecting specific workers. The detection of unexpected or undesired health effects can prompt the correction of inoperative or ineffective control measures. Timely and meaningful medical surveillance can detect the early, reversible stages of occupational lead disease so that treatment can be performed to preclude permanent health impairment. Even where a lead-related occupational disease is not reversible, medical surveillance still may serve to identify workers who merit special protection so that additional exposure does not worsen or quicken the development of disease. In all situations, medical surveillance also serves the important function of informing individual workers of their personal health status. Where a lead-related disease has been or is being contracted, the worker has a right to know of this as soon as possible so that he or she can make personal decisions about health care and employment matters.

Summary of the need for MRP.—It is clear that the medical surveillance provisions of the inorganic lead standard should serve numerous critically important functions. These functions will be served and the purposes of the Act furthered, however, only to the extent that workers freely and confidently participate in offered medical surveillance. Participation in medical surveillance offered under the lead standard will sometimes prompt the temporary medical removal of workers at risk. Absent some countervailing requirement, the medical removal of a worker could easily take the form of a job transfer to a lower paying job, a temporary layoff, or possibly even a permanent termination. Each of these consequences of an OSHA-mandated medical removal would have a dramatic economic impact on the affected worker. Without MRP, many workers exposed to inorganic lead would face a painful dilemma. A worker could fully participate in the medical surveillance offered by the standard and risk losing his or her livelihood, or resist participating in a meaningful fashion and thereby lose the many benefits that medical surveillance can provide. OSHA is convinced by the evidence presented during the lead proceeding that, absent MRP, many workers will either refuse or resist meaningful participation in medical surveillance offered by the final standard. The economic disincentives to participation are severe, and must be removed if the medical surveillance provisions of the lead standard are to substantially advance the goals of the Act. MRP was included in the final lead standard so as to eliminate economic disincentives to participation.

Much of the evidence in the lead proceeding documents the extent to which worker participation is adversely affected by the fear that adverse employment consequences will result from participation in medical surveillance programs. This problem was emphasized by the testimony of many workers and worker representatives. The problem was seen as widespread throughout industry, and as having already seriously affected participation in medical surveillance programs under at least several OSHA health standards. Evidence concerning the issue of worker fear impeding participation, however, was not confined simply to testimony from worker representatives. A wide variety of experts verified the existence of this problem, as did several industry representatives. The evidence suggests that economic disincentives to worker participation are currently a problem in the lead industry. OSHA is convinced that the temporary medical removal section of the final lead standard, as well as other sections of the standard, would greatly increase the likelihood of worker resistance to participation in the absence of MRP. Lastly, in deciding to provide MRP in the final standard, OSHA was significantly influenced by experience gathered under the black lung medical surveillance and transfer program of the Federal Coal Mine Health and Safety Act of 1969. Experience under this program reveals the extent to which economic disincentives adversely affect participation even in medical surveillance programs where job transfer and limited economic protection are guaranteed.

Worker fears of adverse economic consequences from participation in medical surveillance programs. Extensive testimony by workers and worker representatives focused on the fear of adverse consequences from medical surveillance. Emphasis was placed on the fact that most Americans work for one simple reason—to provide for oneself and one's family. Anything that jeopardizes the ability to feed, house, and clothe a family is to be avoided at all costs, even if the price is health impairment. Testimony at the November 1977 hearings on MRP by Mr. Anthony Mazzocchi, vice president of the Oil, Chemical and Atomic Workers Union (Tr. 8050), is illustrative of the worker and union testimony on this matter. On November 9, 1977, the following exchange took place:

> Q: To what extent do you think the workers that you represent worry about the possibility of adverse economic consequences if they contract an occupational illness or disease or for one reason or another can't work in their particular job for health reasons?
>
> Mr. MAZZOCCHI: It is foremost in their minds. The job situation today, of course, is a very perilous situation. There are 10 million people unemployed. Job security is foremost in mind to the people we represent. It probably is foremost in the minds of most workers. Their experience demonstrates adequately that if they suffer abnormality on the job, they are removed. That is a fear. In fact, we thought the Act would eliminate this fear. Instead, the debate over the question of wage retention is essentially, if it is not successful, is the self destruct mechanism of this Act. Workers will resist taking physical examinations and will continue to play roulette with their lives because they have no viable option. It is discussed probably more than any other subject because it is real. It is not an abstraction to a worker. The imperative of feeding one's family that very evening cannot be denied, as opposed to being completely incapacitated or dying of a disease somewhere down the line. (Tr. 8059-8060.)

Mr. Lloyd McBride, president of the United Steelworkers of America (Ex. 355BB) testified on March 30, 1977, in the following fashion:

> • • • (O)ur concern is that unless there is earnings protection, that the desire of a responsible family head to provide food and shelter and clothing for their loved ones would cause them to continue to work with this bad condition, and continue to absorb increased lead to a level that ultimately would be fatal: that the drive—the reason the worker is in the workplace in the first place is to provide for the necessities for oneself and the family. In most of these cases, a family is involved. And the sacrifice the parent will make to insure food for the family, food for the youngsters, decent clothing if possible, decent housing, there is no limit to what a person will do in order to provide those things, including the willingness to risk one's health. That is our concern. That the human equation will cause a person to avoid taking the medical examination if one of the results is likelihood of removal from being gainfully employed; or having their income substantially reduced. That would pose a threat to the safe and proper administration of the standard. That is our basic reason for asking for rate retention and urging it. It is kind of a human equation, perhaps, but I think it is one that most of us would identify with. If we were confronted as the bread winner of a family, of tolerating a health hazard, perhaps, in order to continue to provide for our families, most of us, absent from some other way to do it, and the high unemployment economy, there is very little other opportunity for the person in the lead plant to go out and get other employment—faced with that combination of circumstances, I think most of us would put up with the contined health hazard. That is why we are asking for maintenance of earnings. (Tr. 2980-2981.)

Numerous workers from a variety of industries have testified as to their personal knowledge and experiences concerning (1) adverse economic consequences due to health impairments, (2) worker fear of such consequences flowing from participation in medical surveillance, and (3) worker refusal or reluctance to participate in available medical surveillance due to this reason. Such testimony has been received by lead industry workers (Tr. 2965, 2980, 2983(20)-2983(21), 4755, 4768, 4797-4799, 4800-4801, 4810-4812, 4814, 4823-4824, 4827, 4833, 4842, 4847-4848, 5008, 5047, 5520-5521, 5523, 5539, 5585-5586, 5599, 5669, 5678, 5866, 5878, 6026, 6033, 6099, 6120-6123, 6125-6126, 6134, 6136-6137, 6162-6163, 6165, 6260, 7604, 7606, 7621-7622, 7635, 7709-7710, 7883, 7907-7908, 7934-7936, 7941-7942, 8057-8060, 8071, 8075, 8213-8216; Ex. 431; Ex. 452, pp. 9-13), by workers employed in coke oven batteries (Ex. 374, pp. 125, 128-133, 140-141, 143-145; Ex. 390B, pp. 145, 225-227, 240-243; Ex. 390C, pp. 2687-2688, 2713, 2959-2961, 3015-3016, 3099-3100, 3230-3231, 3380-3381), by workers exposed to cotton dust (Ex. 379A, pp. 12-15, 32-33, 1301-1303, 1305, 3023, 3029; Ex. 429, pp. 1-2), and by workers exposed to benzene. (Tr. 8046-8047; Ex. 373, pp. 1253-1254, 1260-1263, 1265.) The scope and severity of this problem has been stressed in great detail by representatives of the following major labor organizations: AFL-CIO (Ex. 372, pp. 6-11; Ex. 450B, pp. 5-6), United Steelworkers of America (Tr. 2964-2967, 2979-2981, 2983(7)-2983(8), 2983(20)-2983(21),

5008, 7179-7180, 7184-7186, 7219-7220; Ex. 165, p. 2; Ex. 374, pp. 126-133; Ex. 378, pp. 2-3; Ex. 390A, pp. 84, 105-106, 143; Ex. 452, pp. 1, 6-7, 9, 11-13, 34), United Automobile, Aerospace Agricultural Implement Workers of America (Tr. 5046-5047; 8242, 8264-8265; Ex. 171, pp. 12-14; Ex. 349, p. 2), International Brotherhood of Teamsters, Chauffeurs, Warehousemen Helpers of America (Tr. 8075-8079, 8092-8093; Ex. 401A pp. 1-2), Amalgamated Clothing Textile Workers Union (Tr. 7262-7263; Ex. 379A, pp. 12-15, 32-33, 1301-1303, 1305, 3023, 3029), United Paperworkers International Union (Tr. 7603-7607, 7610, 7622), Oil, Chemical Atomic Workers International Union (Tr. 8059-8060; 8064, 8068-8069; Ex. 400A, pp. 3-5), United Electrical, Radio, and Machine Workers of America (Ex. 354D, pp. 2, 5), United Rubber, Cork, Linoleum Plastic Workers of America (Tr. 4264; Ex. 281, pp. 3-5, 9-10, app. A), International Chemical Workers Union (Ex. 410A, pp. 2-3), and the United Mine Workers of America (Tr. 8427-8428, 8434-8437, 8440-8441, 8449-8450; Ex. 408, pp. 3-5, 9-10, App. A). Testimony included specific examples of how worker fear has affected participation in medical surveillance programs provided by OSHA standards. OSHA's vinyl chloride standard contains a mandatory medical removal provision without the presence of an MRP benefits component. (29 CFR section 1910.1017(K)(5)(1977).) Witnesses testified that there was widespread concern among vinyl chloride workers about potential adverse employment consequences of participation in offered medical surveillance, and that the level of participation had been adversely affected. (Tr. 7983, 8055, 8064-8065; Ex. 393, p. 3; Ex. 400A, p. 4.) One local union representing workers exposed to vinyl chloride was even considering urging its members not to participate in medical surveillance provided pursuant to the standard. (Tr. 8055.) Another local union representing workers exposed to benzene has repeatedly advised its members not to take medical examinations offered pursuant to OSHA's emergency temporary standard for benzene, with the result being that most workers declined to participate. (Tr. 8054.) Evidence in the record also points toward worker nonparticipation in medical surveillance offered pursuant to OSHA's asbestos standard. (Tr. 7315-7316; Ex. 3(98), p. 12; Ex. 354M, p. 1.)

The lead record contains compelling evidence from many other sources which document the necessity for MRP. Dr. James Merchant, Director of NIOSH's Division of Respiratory Disease Studies (Ex. 382B), testified both in OSHA's cotton dust proceeding, and lead proceeding, as to worker fear of adverse employment consequences due to participation in medical surveillance programs. His testimony concerning medical surveillance (See, Tr. 7353-7354) included the following:

> I think in order for employees to accept these programs, it has been our experience that they must have a very clear understanding in regard to the confidentiality of the data; that this be data that is available only to the physician and is not data that is entered into their personnel file, or possibly could affect their employment. Now, I am not saying that this happens, or that it doesn't happen. All I am saying is that this is a common fear that workers have, whether they are textile workers, whether they are coal miners, whether they are steel workers; we run into it all of the time, in our programs and our epidemiological studies. Workers must continually be reassured that the data, the medical information that is collected on them, is confidential; and we go to great lengths in our surveys to explain the provisions of the Privacy Act, and assure the workers that this information will not be compromised. Despite that, we have very often a proportion of the workers who are reluctant to participate, because in their view they are not confident that this information will be kept confidential * * *. (Ex. 379A(3), pp. 1301-1302.) Now, in regard to textile workers, I think my experiences are very largely from seeing lots of patients clinically in chest clinics and talking to textile workers, and getting their perceptions of their worries about losing their job or being laid off or things like that. And I would hasten to say that this is their perception. And I think it is a very real perception, and I think it will have a very real bearing on the success or failure of any surveillance program * * *. This is based upon my impressions of talking and examining several textile workers; but I think that is—you know, it is my opinion—that this is an area which is a problem, and I think it is certainly not common, or it is not to be found only in the textile industry, but it is common in other industries. So I think it is a general phenomenon. (Ex. 379A(3), pp. 1305-1306.)

Dr. John Finklea, then Director of NIOSH, noted that OSHA health standards have not heretofore contained MRP. He stated that "Consequently unprotected workers may hesitate to seek desirable medical follow-up because their current employment may be jeopardized or future job opportunities limited." (Ex. 379B, p. 11.) Dr. Finklea also advised that:

> Medical surveillance programs should be structured in a way to encourage worker participation. Workers should not have to fear that abnormal medical findings may lead to the loss of employment or other adverse employment effects. (Ex. 422, p. 1.)

NIOSH summarized its position as to MRP as one of agreement that such a provision is both necessary and appropriate. (Ex. 422, p. 2)

NIOSH statements concerning worker fear of the consequences of medical surveillance were amplified by the testimony during the lead proceeding of Dr. Daniel T. Teitlebaum, a physician with extensive experience in lead toxicology. (Ex. 56 (C.V.).) Dr. Titlebaum is also Director of Poisonlab, a licensed industrial consulting toxicology laboratory, (Tr. 370-72) and he described some of his experience with lead workers as follows:

> A. * * * In studies which we have done, there have been workers who have refused to participate in studies because of fear of losing their jobs. * * * I, as an independent laboratory and as a private physician, I have conducted studies on behalf of patients, on behalf of the State Compensation Funds, on behalf of private insurance companies, and the behalf of the company, on behalf of the union, on behalf of OSHA. Where we have entered into the situation as an independent with no axe to grind on anybody who is involved. It doesn't matter. There are some people who simply feel if the number is on the paper and it is elevated, that they are going to lose their jobs.
>
> Q. And you have had this concern expressed to you firsthand?
>
> A. Absolutely. (Tr. 422-423.)

OSHA also attaches importance to the experience voiced by the two State occupational safety and health agencies which submitted formal comments to the September, 1977 MRP FEDERAL REGISTER Notice. The California Occupational Safety and Health Administration agreed that MRP was necessary and appropriate (Tr. 464A), and stated that:

> Medical surveillance programs are essential to the development of early warning systems regarding exposure to toxic substances. If employee earnings, seniority, and other job rights are not protected, when medical examinations demonstrate a need for transfer or removal, it is our tragic experience that many workers choose not to participate in such programs. The risk of long-run health hazards is psychologically discounted when the employee is faced with the short-run spectre of being unable to support his or her family. Thus in Lathrop, California, some workers were aware of their possible infertility for almost a year before seeking medical assistance, due to their fear of job and income loss resulting from medical examination results. As a direct consequence, it is almost certain that more workers were over-exposed to DBCP. (Tr. 464B.)

OSHA's Kentucky counterpart stated that:

> A refusal of employees to participate in a medical surveillance program because of fear of loss or removal from their jobs has been a problem associated with a number of substances such as the carcinogens, asbestos, etc. (Ex. 354M, p. 1.)

OSHA attaches similar significance to evidence presented by several industry representatives—evidence which verifies the necessity for inclusion of a MRP provision in the final lead standard. First of all, industry spokesmen verified that the problem of workers refusing to participate in medical sur-

veillance programs had arisen under both OSHA's asbestos and vinyl chloride standards. (Tr. 7315-7316, 7565-7567; Ex. 3(98), p. 12.) Numerous industry representatives denied that a problem existed in general (Tr. 7456-7457, 7468-7469, 7520-7521, 7759-7760, 7861-7862; Ex. 354H, p. 1; Ex. 354L, p. 1; Ex. 354(O), p. 5; Ex. 354U, p. 2; Ex. 354Y, p. 1; Ex. 354FF, p. 1; Ex. 354HH, p. 2; Ex. 385, pp. 7-8, Ex. 394A, p. 2; Ex. 396A, p. 2) but several business spokespersons freely acknowledged that worker participation in medical surveillance is influenced by perceptions concerning adverse employment consequences. NL Industries, for example, a major resource manufacturer operating in over 30 States, and the largest recycler of lead in the country (Ex. 3(118), pp. 1-2), stated that its experience was that workers at risk often decline to participate in medical surveillance programs. (Ex. 3(118), p. 11.) The National Association of Manufacturers agreed that the absence of MRP in OSHA standards is in fact an obstacle to worker participation in medical surveillance programs. (Ex. 354(O), p. 5.) A representative of the Occupational Safety and Health Group of Organization Resources Counselors, Inc., a consulting firm to some 50 medium to large corporations (Ex. 385, p. 2), when asked about worker fear of adverse economic consequences, responded that "I am sure it must be a concern of some employees," but he did not know the extent of such worker concern in the lead industry. (Tr. 7521-7522.) Finally, ESB, Inc., a corporation operating some 21 battery and lead related plants in this country (Ex. 354U, p. 1), provides earnings protection as part of its overall medical surveillance program. (Id., p. 2.) Though voicing opposition to OSHA's MRP proposal, ESB described its own medical removal protection program as follows:

> While removal protection is not required, ESB recognizes that it can affect workers' attitudes towards their jobs and the medical surveillance system and, where appropriate, ESB has adopted rate protection programs as an appropriate means of facilitating the success of our total program. (Id.)

OSHA's reasoning for including MRP in the final lead standard parallels the experience of this large battery manufacturer.

Existing industry practices concerning economic protection for workers temporarily removed due to occupational health problems.

OSHA believes the foregoing evidence compels two conclusions. First, absent MRP, many workers covered by the inorganic lead standard will fear that participation in offered medical surveillance will lead to adverse employment consequences. And second, this fear will manifest itself in substantial resistance to meaningful participation in medical surveillance programs. As noted, the lead record documents the existence of significant worker fear of adverse economic consequences. The lead record also amply documents the fact that such fear is often fully justified in light of existing industry practices. Workers who must be temporarily removed from their jobs due to the risk of sustaining material impairment frequently face wage loss or even discharge.

In 1976 the U.S. Bureau of Labor Statistics published a detailed study of occupational safety and health provisions in major American collective bargaining agreements. (Ex. 365.) This study examines some 1,724 major agreements covering 7.9 million workers—approximately half of the total work force under union contract in the industries examined. (Id., pp. 1-2.) The study reveals that most contracts lack clauses dealing with occupational illnesses or diseases, although provisions addressing "disabilities" or "injuries" might encompass occupational health problems. (Id., p. 33.) Most contracts contain some form of compensation for occupationally disabled workers, but most often this compensation only lasts for one day. (Id., p. 42.) Some contracts contain "red circle," or "rate retention," provisions maintaining the former rate of pay of a worker transferred to a lower paying job due to occupational safety or health reasons. (Id., p. 43.) Some 5 percent of the 1724 contracts contain such provisions, covering 3 percent of the 7.9 million workers. (Id., p. 55 (Table 22).) A larger percentage of the 1,724 contracts, 41 percent, contain some benefit provision such as liberal vacation or holiday payments, or supplements to workers' compensation benefits, that would not be available to workers having no health impairment. (Id.) The lead record contains numerous such red circle and workers' compensation supplement provisions. (Ex. 157, p. 10; Ex. 158, p. 68; Ex. 261, p. 21; Ex. 368, p. 83; Ex. 369, pp. 56-57; Ex. 379A, Memorandum; Ex. 389, p. 14; Ex. 400B, passim; Ex. 401B, p. 15; Ex. 404B(D-1), p. 4; Ex. 404B(D-2), p. 17; Ex. 404B(D-5), p. 48; Ex. 404B(D-6), p. 34; Ex. 415B, p. 76; Ex. 415C, p. 23; Ex. 423, p. 23; Ex. 424, p. 12; Ex. 425, p. 6; Ex. 426, Art. 19C; Ex. 427, p. 45; Ex. 430C-2; Ex. 430C-3; Ex. 430D(1), p. 33; Ex. 430D(4a), p. 18; Ex. 430D(4b), Section 62; Ex. 430D(7), p. 10; Ex. 430D(13), p. 17; Ex. 430D(16), Section 5; Ex. 430D(17), p. 37; Ex. 430D(23), p. 12; Ex. 430D(25), p. 14; Ex. 430D(26), p. 96; Ex. 430D(27), Section 4; Ex. 430D(28), Art. 12(d); see also, citations to workers' compensation supplement provisions at "MRP and workers' compensation claims" discussion, infra.)

The Bureau of Labor Statistics study, however, shows that the general rule throughout industry is that union workers temporarily removed from their jobs so as to protect their health cannot expect to have their livelihood maintained during the period of removal. (See, Tr. 7522-7523) Nonunion workers presumably have no greater economic protection in this respect than union members. Consequently, absent MRP, many workers in the approximately 45 industries affected by the lead standard can expect to sustain economic loss if removed pursuant to the temporary medical removal provision of the final standard.

Red circle rate provisions, or rate retention clauses, appear to be common throughout two of the prime sectors of the lead industry (primary lead smelting and battery manufacturing), but the economic protection afforded by these provisions is often very limited in duration. Primary lead smelter collective bargaining agreements generally limit rate retention to less than 4 months duration (See, Ex. 389, p. 14), while rate retention where utilized in the battery industry is generally limited to a 90-day maximum. (See, e.g., Ex. 379A, p. 4: Ex. 401B, p. 15) Workers still on removal status when these periods expire face a substantial reduction in earnings. Experience shows that workers with the greatest exposure to lead get removed with the greatest frequency (Tr. 2172-2173), and transfer is often from some of the highest paying positions to some of the lowest paying positions. (Ex. 354U, p. 2; Ex. 391, p. 2) Collective bargaining agreements reveal that, absent earnings protection, a worker in a primary lead smelter under such circumstances could easily incur a 21.1 percent pay reduction ($218 on a monthly basis) by being transferred. (Ex. 430D(7), p. 18) A worker in a battery manufacturing plant could easily incur a 25 percent pay reduction ($346 on a monthly basis) by being transferred from a high paying to a low paying position. (Ex. 404B(D-4), pp. 119-120) A much greater pay loss would occur if a worker were to be sent home instead of being transferred, since apparently none of the industry collective bargaining provisions maintain earnings in this event. (Ex. 7740-7741)

Although a limited form of MRP is provided by many primary lead smelters and battery manufacturing plants, there exist countervailing industry practices which in essence guarantee that some workers sustain economic loss by participating in medical surveillance. In some instances, explicit corporate policy promises a discharge to any worker unfortunate enough to absorb harmful quantities of lead. The Lead Industries Association has proposed that "as a general rule a worker

should not be discharged because he has an elevated blood lead" (Ex. 28(7); Ex. 354AA, pp. 2-3), but such a policy is not uniformly applied within the prime sectors of the lead industry. For example, one of the primary lead smelters adopted a new policy in 1975 concerning the effects of participation in medical surveillance. (Tr. 4720, 4877, 5017-5018; Ex. 170) The policy, which was posted at the plant and distributed to all employees (Ex. 170), included the following elements:

> Any employee whose blood lead level is found to be above 80 micrograms will be presumed to have been in habitual violation of these policy requirements (concerning respirators) . . . (Ex. 170, p. 2)
> * * * (A)n employee showing a blood lead level of 80 or above will be given a Written Warning notice and advised that his blood lead level must be returned to a level below 80 within the next 90 days. The employee's blood lead level will be checked each thirty (30) days and he will be advised of the results. If at the end of the ninety (90) day period the employee has failed to return his blood level to less than 80, excepting extraordinary mitigating circumstances, he shall be discharged. (Id., p. 3)

When faced with such an explicit policy, many workers would understandably decline to freely and meaningfully participate in offered medical surveillance.

At the time this policy was adopted, working conditions within this large primary lead smelter virtually assured high blood lead levels elevations. Late in 1975, NIOSH studied this smelter in great detail. (Ex. 300) Over two thousand full shift personal air samples were collected—many of which grossly exceeded the existing 200 μg PbA/m^3 lead in air standard. (Id., section I.B.—Industrial Hygiene Surveys, Tables 2-7) Many of these measurements exceeded the current OSHA standard by a factor of 10 to 200 times. (Id., Tables 2-7) NIOSH noted that "The existing ventilation systems should be evaluated for effectiveness—many systems are poorly designed and/or maintained. (Id., section V—General Recommendations) NIOSH discovered that even some air lead levels in plant lunch rooms exceeded 200 μg PbA/m^3. (Id., section V—Recommendations—Lunchrooms, Table 30)

In view of these factors, it is OSHA's judgment that it was a virtual certainty that persistent blood lead levels close to or in excess of 80 μg PbB/100g were to be encountered. At this primary lead smelter, the possiblity of losing one's job as a conseqence of participation in medical surveillance was genuine. This formal corporate discharge policy was apparently rescinded soon after its adoption. (Tr. 4720, 4877, 5017-5018) The mere consideration of such a corporate policy, however, undoubtedly increases worker apprehension about the risks inherent in participation in medical surveillance programs.

The lead record further reveals that the preceding discharge policy is not an isolated occurrence. The Battery Council International (BCI), the major trade association representing battery manufacturers (Ex. 137, p. 1), recommends that workers either be discharged or permanently transferred (with no maintenance of earnings) whenever their blood lead levels repeatedly exceed 80 μg PbB/100 g. (Ex. 397A, pp. 4, 6-7) The BCI stated that it is "generally accepted" within industry that a third incident of elevated blood lead levels merits such action. (Id., p. 7) The BCI also recommends similar action with regards to "workers who are more than ordinarily susceptible to lead absorption or to the effects of lead." (Id., p. 4) To the extent that the BCI's statements reflect prevalent industry attitudes and policies, it is clear that lead battery workers have justification for concern about participation in medical surveillance. Only through participation can a worker be classified as "more than ordinarily susceptible" or detected as having a highly elevated blood lead level on several occasions. And, highly elevated blood lead levels are a virtual certainty in many plants due to the widespread failure to reduce air lead levels below the existing 200 μg PbA/m^3 OSHA standard. (Tr. 782-783, 1284, 3250; Ex. 3(5), p. 1; Ex. 3(26), p. 2; Ex. 3(44), p. 4; Ex. 3(56), p. 2; Ex. 3(76), p. 2; Ex. 3(89), p. 2; Ex. 3(93); Ex. 3(103), pp. 9-10, 85-86, 88, 90, 92; Ex. 3(106), pp. 2-5, App. 1-4; Ex. 3(110), p. 1; Ex. 3(111), pp. 14-16; Ex. 3(127), p. 1; Ex. 4(6), p. 1; Ex. 80, p. 2; Ex. 84, pp. 10-11; Ex. 101A; Ex. 104, p. 27a; Ex. 123, p. 5; Ex. 125, p. 26; Ex. 128C; Ex. 335, pp. 6, 101-103)

The lead record reveals that at least several battery manufacturers implement the recommendations of the BCI. At one plant, workers are permanently laid off and barred from any lead job upon the second occurrence of an elevated blood lead level (with seniority determining whether or not individual workers are able to secure non-lead jobs in the plant). (Tr. 7709-7710; Ex. 427, pp. 58-59) At another battery plant, the second occurrence of an elevated blood lead level results in a permanent transfer, without earnings protection, to a lower exposure job. (Tr. 8453-8454) A third battery manufacturer, in a vague letter to plant employees, stated that even legitimate reoccurring medical absences were "inconsistent with holding a job", and thus would count against a worker's personnel record. (Tr. 5254; Ex. 179A) Presumably this applies to workers showing repeated occurrences of lead intoxication or poisoning.

Impact of the final lead standard on disincentives to participation.—OSHA believes the foregoing evidence demonstrates that, absent MRP, many workers exposed to inorganic lead will decline to meaningfully participate or actively resist participation in medical surveillance offered under the final standard. The economic disincentives to participation are real and substantial; worker fears of adverse economic consequences due to participation are widespread and justified in light of industry practices. OSHA views MRP, therefore, as an essential element of the medical surveillance program offered by the final lead standard. MRP is even more indispensable in light of aspects of the final standard which would heighten disincentives to participation in the absence of MRP.

First of all, the final standard mandates the temporary medical removal of workers at substantial risk of sustaining material impairment. The prior lead standard contained no such requirement. Although many lead companies have some form of temporary medical transfer policy, such policies do not appear to be universally applied throughout the various lead industries. (Ex. 26, pp. 5-38, 5-81, 5-99; Ex. 65B, pp. 20, 33, 35, 38) For many lead workers, the mandatory temporary medical removal provision of the final standard will for the first time pose a major threat of economic loss due to removal—a threat heightened by the setting of explicit blood lead level removal criteria. Absent MRP, the temporary medical removal provision even creates a substantial disincentive to participation in medical surveillance where one may not have previously existed.

Secondly, the blood lead level removal criteria of the final standard are much more stringent than criteria currently used by industry. While most lead firms do not transfer a worker until his or her blood lead level exceeds 80 μg/100g (Tr. 1274, 1666, 7695-7696, 7894, 7908-7910, 8284, 8326-8327; Ex. 26, pp. 5-11; Ex. 404B, p. 68; Ex. 453, p. 15), the final standard when fully implemented will require removal when a worker's blood lead level over time exceeds 50 μg/100g. The standard's removal criteria are set at a preventive level so that workers are removed prior to the onset of clinical lead poisoning. The much higher industry 80 μg figure, however, represents a point where many workers begin to experience clinical signs of lead poisoning. (See Tr. 7161.) Lead industry representatives have opposed the establishment of lower blood lead level removal criteria. (See, e.g., Ex. 335.) In light of this opposition, one would expect may lead firms to decline to voluntarily maintain the earnings of workers (having "acceptable" blood

lead levels from an industry viewpoint) who are temporarily removed pursuant to the OSHA standard. In the absence of MRP in the final standard, therefore, it is to be expected that the temporary medical removal provision will result in many more removals without economic protection to the removed workers than is currently the case in the lead industry. Without MRP this increase in economic disincentives to participation would substantially increase the present reluctance of workers to seek the benefits of medical surveillance programs.

Absent MRP, a similar increase in economic disincentives to participation will result from the ultimate transfer requirements of the final standard. Two years after the effective date of the standard, a worker being temporarily removed from current lead exposure (due to an elevated blood lead level) may only be transferred to a position having an air lead level exposure below 30 μg PbA/m³ TWA. This transfer requirement is necessary to assure a steady decline in the worker's blood lead level (See, infra, discussion of temporary medical removal from work at or above the action level), but represents a requirement far more stringent than practiced within industry today. Lead industry removal programs typically have the goal of only reducing a worker's blood lead level from 80 μg PbB/100g to about 60 μg PbB/100g (Tr. 1274, 5637, 8284-8285, 8326-8330; Ex. 179, pp. 3-4; Ex. 354U, pp. 2-3), and apparently there has been little difficulty in finding alternative positions which would permit a decline to 60 μg PbB/100g. Although OSHA is confident that a diligent company can provide substantial numbers of transfer opportunities which will satisfy the 30 μg PbA/m³ TWA requirement, this will not always be possible. In some instances no transfer positions will be available—particularly if the company has failed to come into compliance with the central provisions of the lead standard. In these instances a worker will likely have to be sent home until a transfer opportunity arises. As noted earlier, few if any lead firms currently maintain a worker's earnings in such a situation. Thus, absent MRP, the temporary removal provisions of the final standard will sometimes create one of the most forceful economic disincentives to participation—a layoff.

Finally, the duration of temporary medical removals resulting from the final standard will also increase economic disincentives to participation, particularly during the first several years of the standard's effect. As noted earlier, industry rate retention programs where in effect generally limit compensation to a 3 to 4 month maximum. (Ex. 158, p. 68; Ex. 354U, pp. 2-3; Ex. 401B, p. 15; Ex. 404B(D-1), p. 4; Ex. 404B(D-5), p. 48; Ex. 404B(D-6), p. 34; Ex. 424, p. 12; Ex. 425, p. 6; Ex. 430D(4a), p. 18) Medical removals mandated under the final standard, however, will often substantially exceed four months in duration (See detailed discussion, infra, concerning duration of MRP benefits). It is not possible to precisely estimate an average period of removal. But, the likelihood of lengthy removals is reflected by the observation of one large battery manufacturer that it generally takes considerably longer for worker blood lead levels to decline from 60 μg PbB/100g to 40 μg PbB/100g than to decline from 80 μg PbB/100g to 60 μg PbB/100g. (Ex. 354U, p. 5) Absent MRP, the effect of extended periods of removal will often be substantial economic loss to removed workers. This would most likely be a problem during the first several years of the new standard during which time medical removals would often be of long term workers having substantial body burdens of lead (see detailed discussion, infra, concerning the phasing-in of MRP.) In any event, absent MRP, the duration of medical removals to be anticipated once the final standard is issued will undoubtedly increase economic disincentives to meaningful worker participation in medical surveillance programs.

Importance of meaningful worker participation in the standard's medical surveillance program.—Having discussed MRP as a necessity to effectuate meaningful participation in offered medical surveillance, it is appropriate to emphasize the importance of meaningful participation. The medical surveillance program provided by the final standard consists of three central elements: (1) periodic blood lead level biological monitoring, (2) periodic medical examinations, and (3) the opportunity for a medical examination upon the request of a worker. The success of each of these three elements depends not only on the fact of worker participation, but more importantly on the quality of participation. Workers must feel free to seek medical attention when they feel ill; they must fully cooperate with examining physicians to facilitate accurate medical diagnoses; and also refrain from efforts to conceal their health status. In the absence of these qualities of participation, the medical surveillance program provided by the final standard cannot serve to eliminate occupational lead diseases.

The success of periodic blood lead level biological monitoring depends not only on workers permitting blood samples to be extracted, but also on workers refraining from efforts to alter their blood lead levels. Chelating agents, despite their potentially harmful aspects, effect a rapid, short term reduction in blood lead levels. (Tr. 217, 234) These drugs pose a means whereby workers can manipulate biological monitoring results and thereby avoid adverse employment consequences. The lead record contains evidence that some workers have apparently illicitly secured prescription chelating agents in Mexico precisely to conceal their true blood lead levels. (Tr. 4076) Similarly, workers have also apparently diluted urine samples so as to yield low urine lead level measurements. (Ex. 3(109), Medical Study, pp. 16, 23) Any such deliberate attempts to pervert biological monitoring tests are lamentable, and should be discouraged. Such actions by workers, however, reveal a desperate effort to avoid economic loss no matter what the consequences to one's health. OSHA anticipates that, absent MRP, worker misuse of chelating agents could develop into a major problem in situations where workers would be unable to evade participation in blood lead level monitoring. The use of chelating agents would not only directly endanger worker health but would destroy the value of blood level biological monitoring.

Another problem is raised by workers failing to fully cooperate with examining physicians in the course of periodic medical examinations. Blood lead level biological monitoring is of great importance in the final standard, but equal significance must be attached to periodic medical examinations. There is a wide worker variability of response to lead, and our understanding of low lead exposure health effects is by no means complete. The 50 μg PbA/m³ TWA PEL incorporates only a very modest margin of safety. Some adverse health effects from lead exposure do not readily correlate to blood lead level. For all of these reasons, OSHA is convinced that blood lead level biological monitoring by itself cannot afford workers adequate protection from material impairment to health. Effective periodic medical examinations permit the flexibility and informed judgment that only a physician in a one-on-one situation can provide.

The success of medical examinations in achieving accurate medical diagnoses, however, depends substantially on the degree of voluntary worker cooperation with the examining physician. Many early symptoms of lead poisoning—Such as tiredness, sensory-motor uncoordination, fine tremors, fatigue, nervousness, sleeplessness or sleepiness, memory difficulties, anxiety, irritability, loss of appetite, constipation, malaise, weakness, headache, and muscle and joint pains (Ex. 101A; Ex. 119A, p. 2) are highly subjective in nature. If a worker denies or masks these symptoms where they

occur, a proper diagnosis is made extremely difficult if not impossible.

Worker reluctance to divulge pertinent information to a physician can preclude the taking of an adequate medical history—a crucial element of a competent medical examination. Mr. Melvin A. Glasser, Director of the Social Security Department of the International Union, United Automobile, Aerospace and Agricultural Implement Workers of America (Tr. 8213), and a member of the governing council of the Institute of Medicine of the National Academy of Sciences (Tr. 8229), stressed the value of a medical history in the following terms:

> With regard to general or non-occupational health care, a most important preventive medical procedure is a careful medical history-very simply, the patient telling his or her physician about health practices, medical conditions and symptoms before there are obvious outward signs of disease. It is estimated that 80 percent of pathological conditions can be detected simply with proper administration and analysis of a medical history, even by a non-physician. Administration of such a history as part of a continuing integrated health care program is generally agreed to be worth far more in the prevention of disease than the extremely expensive battery of laboratory tests given under the rubric of annual physical examinations. (Tr. 8214–8215; Ex. 404a, p. 2)

Dr. Daniel T. Teitlebaum, Assistant clinical Professor of Preventive Medicine and Comprehensive Health Care at the University of Colorado Medical Center, and Director of the Poisoning Treatment Center at St. Anthony Hospital in Denver, Colorado, (Ex. 56, p. 1) attaches similar importance to an effective medical history:

> In my own practice, I can state that when I have completed history taking, I have a very strong feeling as to whether or not I will find the physicial or laboratory findings consistent with lead poisoning in a vast majority of cases. It is exceptionally unusual to make the diagnosis of lead poisoning only on the basis of a physical examination or laboratory results without the characteristic history. If I were to choose between the physical and the history examination as a screening test for lead, I would definitely place my reliance on the history, which would probably detect lead poisoning some months before overt signs can be noted. (Id., p. 44)

Mr. Glasser and Dr. Teitlebaum's testimony parallels that of Dr. James Merchant, Director of NIOSH's Division of Respiratory Disease Studies (Tr. 7359-7361), and is also supported by several studies contained in the general medical literature. (Tr. 8229-8230; Ex. 404B, pp.1-2)

If workers fear adverse economic consequences from participation in medical examinations, then an adequate medical history is unlikely. This is particularly true in view of the general medical phenomenon of patient denial of symptoms. As noted by Mr. Glasser (and echoed by Dr. Merchant (Tr. 7359-7361)):

> Experts agree that even when the true doctor-patient relationship exists, there are barriers to the patient revealing symptoms. Barriers are much higher if the doctor-patient relationship doesn't exist and workers don't have such with the company doctor. If the result of diagnosis of lead intoxication is loss of income, or even loss of employment, then the barrier will be even greater. (Tr. 8215; Ex. 404A, p. 2)

MRP is expressly designed to minimize these barriers to open and effective communication between examining physician and inorganic lead worker during periodic medical examinations.

The value of the third central element of the final standard's medical surveillance program—the opportunity for a medical examination upon the request of a worker—is also critically dependent upon voluntary, meaningful participation by affected workers. Few workers can be expected to initiate a medical examination if fear of adverse employment consequences is widespread. And, the opportunity for a medical examination upon the request of a worker is a crucial element of the standard's medical surveillance program for all of the same reasons that periodic medical examinations are provided. Periodic examinations need only be generally provided on an annual basis, thus there is an appreciable opportunity for adverse health effects to arise in between scheduled examinations. The quicker such effects receive medical attention, the less likely the worker is to sustain permanent health impairment. MRP is designed to minimize the probability that substantial numbers of lead workers will continue to tolerate ill health rather than take advantage of immediately available medical examinations.

Experience under the Black Lung medical surveillance and transfer program.—In deciding to provide MRP in the final standard, OSHA was significantly influenced by experience gained under the Black Lung Medical Surveillance and Transfer Program of the Federal Coal Mine Health and Safety Act of 1969 (the "Coal Act"). (See generally, Federal Coal Mine Health and Safety Act, Pub. L. 91-173, 83 Stat. 792 (1969) as amended by Black Lung Benefit Act, Pub. L. 92-303, 86 Stat. 155 (1972) and Federal Coal Mine Health and Safety Amendments Act, Pub. L. 95-164, 91 Stat. 1290 (1977)) Experience under this program demonstrates that economic disincentives do adversely affect worker participation in medical surveillance programs, even where job transfer and limited economic protection are guaranteed.

The Black Lung Medical Surveillance and Transfer Program is mandated by section 203 of the 1969 Coal Act, and is jointly administered by NIOSH and the Department of Labor's Mine Health and Safety Administration. (30 U.S.C. 951(b) (Supp. 1978)) Under the program, working underground coal miners are offered an opportunity every several years to have a chest X-ray taken to ascertain any evidence of the development of coal mine workers' pneumoconiosis ("Black Lung"), a chronic, irreversible lung disease. (42 CFR 37.3 (1977)) The X-rays are performed by facilities completely independent of coal mine operators. (42 CFR 37.42 (1977)) These facilities must meet stringent quality control and confidentiality requirements established by NIOSH. (30 CFR 90.20 (1977); 42 CFR 37.42 (1977)) The X-rays are performed without cost to participating coal miners (42 CFR 37.3 (1977)), and the X-ray films are analyzed only by medical experts who have successfully completed a training and certification program established by NIOSH. (42 CFR 37.51 (1977)) Since 1969, there have been two rounds of offered X-ray examinations—the first round occurring between August, 1970 and December, 1971, and the second round occurring between July, 1973 and March, 1975. (Tr. 7345; Ex. 411A, p. 8) Miners whose X-rays revealed evidence of the development of Black Lung were notified of this fact, as well as notified of their transfer rights under the program. (30 CFR 90.10 (1977))

Miners with evidence of Black Lung receive a form letter they can use to elect to transfer to a position having a low respirable coal dust level. (30 CFR 90.20 (1977)) Upon notification of a "letter carrier's" election to transfer to a low dust position, a coal mine operator must either transfer the miner or guarantee that the miner's current position satisfies the applicable low dust level requirements. (30 CFR 90.31, 90.32; 42 CFR 37.7 (1977)) The previous regular rate of pay is retained by any miner transferred to a low dust position. (30 CFR 90.34 (1977)) Due to the irreversible character of Black Lung disease, transfers are essentially of permanent duration. (Tr. 7386)

More than 8 years have passed since Congress established this medical surveillance and transfer program. Statistics have been collected from which one can gauge the success of this program in (1) identifying miners who are contracting Black Lung disease, and (2) thereafter relocating them in low dust areas so as to minimize progression of the disease. The statistics reveal that over 150,000 miners have been X-rayed at least once, and over 1,200 miners showing evidence of the development of Black Lung have exercised the right to be transferred to low dust positions. (Tr. 7416-7417, 7429)

The same statistics supplemented by the experience of those intimately involved with the program, however, reveal that worker fear of adverse economic consequences due to participation has undermined the overall success of the program.

First, only approximately 60 percent of the eligible working coal miners participated in the first two rounds of offered X-rays. (Tr. 7362-7363) This modest level of participation is striking in view of the seriousness and historically high incidence of Black Lung disease ((1969) U.S. Code Cong. and Ad. News 2503, 2506), and the fact that medical transfers under the program are voluntary on the part of coal miners. (Tr. 7344, 7348, 7389, 8411) The moderate level of participation may have partially resulted from lack of understanding of the program by affected coal miners (Tr. 7364, 7412-7413, 8415), or from the possibility that some operators failed to provide X-rays as required by the law (Tr. 7364, 7368), or from the apparently inconvenient circumstances under which the X-rays were sometimes provided. (Tr. 8429-8430) The relative contribution of these factors to the lack of participation is unknown. Both NIOSH and United Mine Workers of America (UMWA) witnesses agreed, however, that fear by miners of adverse employment consequences also adversely affected participation. (Tr. 7391, 7432, 8436-8437; Ex. 390a, p. 143; Ex. 408, App. A, p. 2; Ex. 408, App. E, p. 211) NIOSH witnesses stated that assuring the confidentiality of X-ray results was a major issue in the development of the overall program, and that the stringent confidentiality regulations adopted were expressly designed to overcome fear by miners that test results could be used against them. (Tr. 7347, 7353-7354, 8412-8417; Ex. 408, App. E, p. 211)

The adequacy of the program's confidentiality requirements was a source of great controversy during the first round of X-rays, with the UMWA apparently cautioning its members about the risks of participation (Tr. 7368-7369, 8413-8414; Ex. 408, App. A, pp. 3, 8) even in the face of an explicit threat of possible prosecution by the U.S. Government. (Ex. 408, App. A, p. 7) Undoubtedly, this conflict adversely affected the level of participation in the first round of X-rays. Witnesses testified that this dispute was largely resolved prior to the bulk of the second round of X-rays. (Tr. 8426-8427) Dr. Lorin Kerr, director of the UMWA Department of Occupational Health (Tr. 8410), however, stated that even though the union fully supported the second round of X-rays, there was great difficulty in convincing coal miners that confidentiality problems had been satisfactorily resolved. (Tr. 8427) Dr. Kerr also explained the lack of participation in the second round of X-rays as substantially resulting from the fear that negative X-ray results would be used against miners filing claims for Black Lung disability benefits (Tr. 8413, 8434-8435; Ex. 408, App. E, p. 210)—benefits which at that time were being processed at a very slow pace. ((1972) U.S. Code Cong. and Ad. News 2307, 2329-2330)

On the basis of the evidence presented in the lead proceeding, OSHA is incapable of definitively explaining why some 40 percent of working coal miners declined to participate in the first two rounds of chest X-rays. OSHA is convinced, however, that much of this lack of participation was due to fears that X-ray results might adversely affect miners' employment or future disability benefits. Fear of adverse economic consequences has also apparently significantly affected the decisionmaking of miners eligible to transfer to low dust positions.

Statistics reveal that as a result of the first two rounds of X-rays, over 5,800 miners have been notified that they are acquiring Black Lung disease and thus have the option of transferring to low dust positions. (Tr. 8421; Ex. 383A, p. 2) Only some 20 percent of these miners, however, have chosen to exercise their transfer rights. (Tr. 7349, 7429) Again, the reasons for this dramatic lack of participation are not fully understood, but several possibilities have been suggested. Some miners faced with the realization that they are acquiring Black Lung might leave the industry (Tr. 7374, 7429), while others perceive no need to transfer jobs since they currently suffer no apparent symptoms of Black Lung. (Tr. 7412) Some miners might choose not to exercise their transfer rights due to the undesirability of the transfer positions (Tr. 7420-7422, 7431, 8421, 8437), while still others might see no need to transfer since they feel they are currently working at a low dust level job. (Tr. 7409, 7430, 8437) The United Mine Workers of America collective bargaining agreement also has a provision whereby miners eligible to transfer can directly bid on low exposure jobs without notifying the Government. (Ex. 368, p. 74)

Although the role of the previous factors is unclear, all witnesses agreed that concern by miners over adverse economic consequences contributes to the low level of participation. First, there is the fear that employer knowledge about the development of Black Lung disease will be used to discriminate against miners. (Tr. 7391, 7430, 8412-8413; Ex. 408, App. E, p. 210) Miners apparently do not worry as much about forms of immediate discrimination (Tr. 8437-8438) as about future job consequences. Individual coal mines do not operate indefinitely, thus many miners change employers during their working years. Prior to the recent amendments to the Coal Act, the Black Lung transfer program offered no protection whatsoever to miners experiencing hiring discrimination (Tr. 7391, 7430; Ex. 408, App. E, p. 210), thus miners apparently feared they would be unable to get jobs in the future if they revealed their health status by coming forward to transfer to a low dust job. (Tr. 7391-7392; Ex. 408, supra)

The second major economic reason for miners choosing not to exercise their rights results from the limited form of rate retention offered to those who transfer. Section 203(b)(3) of the Coal Act has been interpreted to mean that miners who elect to transfer retain their previous hourly rate of pay, but receive no subsequent wage increases until the rate of pay of the new job catches up to the rate of pay of the old job. (30 CFR 90.34 (1977); *Jesse Higgins* v. *Marshall*, No. 77-1829, U.S. App. D.C. (July 25, 1978); 1978 Employee. Safety and Health Guide (CCH) Paragraph 22,908) Transfer is often from a high-paying to a low-paying position. (Tr. 8439-8440) Five or six years are likely to pass before the rate of pay of the low-pay job catches up to the former rate of the high-pay job. (Tr. 8440-8441; Ex. 368, p. 137) The resulting effect on a miner who transfers is that his or her rate of pay is frozen for some 5 or 6 years, while everyone else's rate of pay climbs perhaps 4 percent each year. (Tr. 7426-7427; Ex. 368, p. 137) Since transfers are permanent, this loss of 5 or 6 years of pay increases is never reversed. The adverse impact to a miner over several years would be thousands of dollars lost that would have been received if no decision to transfer had been made. Witnesses in the lead proceeding agreed that this economic disincentive was a deterrent to participation (Tr. 8441), was a source of repeated complaints by coal miners (Tr. 7426-7427, 7432, 8421), and perhaps was even the foremost reason why miners choose not to elect to transfer to a less dusty position. (Tr. 8436-8437; Ex. 390A, p. 143; Ex. 408, App. E, p. 210)

OSHA believes that the experience gained under this Black Lung Medical Surveillance and Transfer program highlights the need for MRP in the final lead standard. The Black Lung program is a comprehensive program embodying numerous concepts which OSHA has incorporated into the lead standard. Despite this, participation in the Black Lung program has been adversely affected by economic disincentives and fear on the part of miners of adverse employment consequences.

These factors have partially if not substantially accounted for 40 percent of miners declining to take X-rays, and 80 percent of eligible miners declining to formally transfer to low dust positions. These figures convince OSHA that success of the lead standard's medical surveillance program depends on the agency squarely confronting the need to include MRP so as to maximize meaningful worker participation. Although there are numerous differences between the lead industry, and the coal mining industry, lead workers presumably care no less about their job security and earnings than do coal miners. The Black Lung program, as does previously discussed evidence in the lead proceeding, demonstrates that genuine job security and earnings concerns can dramatically undermine efforts to protect worker health. OSHA has adopted MRP specifically to minimize the adverse impact of these factors on the level and quality of worker participation in the medical surveillance program provided by the final lead standard.

The scope of the need for MRP.— Before discussing other aspects of MRP, it is appropriate to note that OSHA does not view worker resistance or reluctance to meaningfully participate in medical surveillance as a universal problem affecting the actions of every lead worker in every lead plant. The foregoing paragraphs discussing evidence from many different sources do, however, reflect OSHA's judgment that significant worker reluctance or resistance would seriously diminish the overall success of the lead standard in the absence of MRP. Numerous industry representatives stated that they had experienced no noticeable reluctance of their employees to participate in medical surveillance programs, and thus they questioned the necessity for MRP. Most industry participants in the lead proceeding offered no comments as to whether or not they had experienced worker reluctance to participate in their medical surveillance programs. While some industry comments are irreconcilable with OSHA's view of the evidence on this issue, numerous industry statements are fully consistent with the agency's reasoning.

OSHA is confident that at least some firms through vigorous industrial hygiene programs have already virtually eliminated harmful exposure of their employees to inorganic lead. As a result, no workers get sick or are known to have ever contracted lead-related diseases. Temporary removals due to excessive blood lead levels do not occur, and no worker has ever experienced job loss or wage loss due to occupational health considerations. In this setting there should be little cause for concern by workers about participation in medical surveillance, thus it is understandable that some employers see no need for MRP. OSHA is also confident that some firms, although harmful exposure to lead is an ever-present problem, nonetheless provide comprehensive forms of economic protection for workers temporarily removed due to excessive lead exposures. Such economic protection, differing little from MRP in this standard, serves to remove any economic disincentive to participation that workers might otherwise have felt. Again, it is quite understandable that these employers see no need for MRP since their own employees exhibit no reluctance to participate.

Finally, OSHA is confident that some workers place their personal health above all other considerations and thus fully participate in medical surveillance programs irrespective of the possible economic consequences to themselves or their families. Also, some workers most likely freely participate because they have heretofore been totally unaware of the harmful effects of lead, and thus never considered the possibility that they could be adversely affected by occupational lead exposure.

The foregoing considerations all lend some support to industry comments to the effect that worker reluctance to participate in medical surveillance has not yet been a problem in some plants. OSHA, however, has adopted MRP on the basis of compelling evidence contained throughout the entire lead record and on the problems present throughout the industry. The many preceding paragraphs explain in great detail the agency's reasoning. OSHA realizes that conditions in some plants may present little need for MRP. Those plants are likely to experience no impact from MRP's existence either because there will be no need for temporary medical removals to occur, or because the company's existing economic protection policies differ little from MRP. OSHA also submits that the possibility that some workers would fully participate without MRP in no way detracts from the pressing need to provide MRP for the many workers who would otherwise resist meaningful participation in medical surveillance programs. OSHA is determined to protect all inorganic lead workers so far as is feasible, and feels the conclusion is inescapable that MRP is essential to maximize meaningful worker participation in the medical surveillance program provided by the final standard.

c. *Medical removal protection benefits as a means of allocating the costs of temporary medical removals.*

OSHA's second reason for including MRP benefits in the final lead standard is tied to the nature of MRP's temporary medical removal provisions. Temporary medical removal is fundamentally a protective, control mechanism, thus OSHA has determined that the costs of this control mechanism should be borne by employers in the first instance.

Temporary medical removal is a last-ditch, fall-back mechanism to protect individual workers in circumstances where other protective mechanisms have not sufficed. It is a protective mechanism recognized by and acceptable to both management and labor. There are costs, however, associated with temporary medical removals, both to employers and to temporarily removed employees. When a worker is temporarily removed from a job, the employer loses the sevices of someone trained and experienced in that particular job. The employer might easily incur dislocation costs involved in locating and training a temporary replacement, and might also experience reduced productivity due to the replacement's inexperience. A worker who is temporarily removed might easily lose substantial earnings or other rights and benefits by virtue of the removal. All of these costs are a direct result of the use of temporary medical removals as a means of protecting worker health.

OSHA has determined that the foregoing costs should be borne by employers in the first instance. Since employers automatically will absorb any temporary dislocation or reduced productivity costs associated with temporary medical removals, OSHA's determination primarily involves the lost wages or other costs removed workers might bear in the absence of MRP. MRP is meant to place those costs of worker protection directly on the industry at large rather than on the shoulders of individual workers unfortunate enough to be at risk of material impairment to health due to occupational exposure to lead. The costs of protecting worker health are appropriate costs of doing business, thus employers should properly bear the economic impact of temporary medical removals. The Occupational Safety and Health Act ("the Act"), as does all other recent environmental legislation, recognized that the costs which consumers pay for goods should reflect all costs of production, including costs associated with preventing adverse public health impacts such as air and water pollution, or occupational disease. Under the act, employers have the primary obligation to provide a safe and healthful work experience, thus should incur the costs necessary to satisfy this obligation.

One beneficial side effect of MRP will be its role as an economic incentive for employers to comply with the inorganic lead standard. In recent

years, increasing attention has been focused on the desirability of administrative regulations incorporating economic incentives to compliance. (See, H. Owen and C. Schultze, "Setting National Priorities, The Next Ten Years," 464-477 (1976); C. Schultze, "The Public Use of the Private Interest," Harper's, May, 1977, at 43-62; F. Anderson, A, Kneese, P. Reed, R. Stevenson, and S. Taylor, "Environmental Improvement Through Economic Incentives" (1978).) In other words, an agency should structure its regulations such that regulated parties comply out of simple economic self-interest. MRP was not adopted specifically to serve this purpose, but OSHA feels that MRP will strengthen the protection afforded by the overall inorganic lead standard due to MRP's inevitable impact on compliance.

The phasing-in of the ultimately desirable MRP biological removal triggers is structured such that employers making diligent good faith efforts to comply with the total standard should incur minor MRP costs. Employers who make serious attempts to comply with the standard will experience only small numbers of temporary medical removals—removals which likely can be absorbed by available transfer alternatives. Quite a different result will be experienced by employers who make only cursory attempts to comply with the central provisions of the standard. A simple rule will prevail—the greater the degree of noncompliance, the greater the number of temporary medical removals and associated MRP costs. MRP, in and of itself, will thus serve as an economic stimulus for employers to protect worker health voluntarily by complying with the standard rather than waiting for OSHA to compel compliance. MRP will also reward employers who through innovation and creativity devise new ways of protecting worker health not explicitly contemplated by the formal standard.

OSHA anticipates that MRP will hasten the pace by which employers comply with the new lead standard. Absent MRP, compliance with the new standard could follow the slow pace with which employers have complied with the earlier 1971 200 μg PbA/m^3 TWA standard. As noted earlier, the record of the lead proceeding reveals that many, many employers still have not come into full compliance with this 7-year-old standard. MRP should preclude such a similar history for the new lead standard.

d. *Alternatives to Medical Removal Protection Considered by OSHA*

Before deciding to include MRP in the final standard, OSHA considered and rejected three possible alternatives. First, OSHA could have mandated that all workers participate in medical surveillance offered under the standard. Second, OSHA could have mandated that temporary medical removals occur only at the election of individual workers. And third, OSHA could have permitted the use of respiratory protection in lieu of temporary medical removals. The agency decided against the adoption of any of these possibilities for the following reasons.

Mandating that workers participate in medical surveillance.—This alternative to MRP would require employers to compel all employees to participate in offered physical examinations and biological monitoring. To meet this obligation, employers would have to discipline and ultimately discharge any worker who refused to submit to medical surveillance. Under this theory, compulsion would be used to override any reluctance a worker might have concerning participation. Since participation would apparently be assured, there would seem to be no need to include MRP as a means of effectuating participation. This theory has been implicitly advocated as an alternative to MRP. (Ex. 385, p. 10.)

OSHA rejected this alternative for several reasons. First, mandating worker participation would not affect the issue of appropriately allocating the costs of temporary medical removals. Temporary medical removal is fundamentally a protective, control mechanism, and OSHA has determined that the costs of this control mechanism should properly be borne by employers. This judgment is unrelated to whether or not workers voluntarily participate in medical surveillance, thus OSHA would include MRP in the final standard even if total worker participation were somehow assured without MRP.

Second, OSHA is convinced that mandating worker participation in the absence of MRP cannot serve to assure voluntary and meaningful worker participation—upon which success of the standard's medical surveillance program depends. Mere participation is not an end in and of itself. The quality of participation is crucial due to the special nature of lead poisoning. Mandatory participation should succeed in forcing workers to permit blood samples to be taken. No degree of compulsion, however, can prevent workers from obtaining and misusing chelating agents so as to yield apparently low-blood-lead level results. Mandatory participation should succeed in forcing workers to stand before physicians for physical examinations. Again, no degree of compulsion can force workers to reveal subtle, subjective symptoms of lead poisoning which a physician needs to know as part of an adequate medical history.

As described earlier in great detail, OSHA is convinced that MRP is essential to maximize not just participation, but meaningful participation. Absent MRP, many workers will fear the possible adverse economic consequences of participation, therefore they will resist or refuse to participate in a meaningful fashion. Mandatory participation cannot and will not change this fact so long as the economic consequences of participation continue to be of concern.

An example affecting some lead workers is instructive. We know that lead exposure can damage male sperm cells, possibly causing birth defects, stillbirths, miscarriages, and sterility. (See, Health Effects Attachment) Male lead workers planning to father a child should consider consulting an examining physician in the course of the next periodic physical examination offered under the standard, or even request an immediate physical examination to verify the absence of damage to his reproductive system. If such an examination reveals that the worker's reproductive system has been damaged, the worker may have to be removed from current exposure for 2 or 3 months so that new, undamaged sperm can be produced. (Tr. 567-568, 583.) With MRP, male workers have nothing to fear from a 2 or 3 month removal—and the standard's medical surveillance program should thereby serve to prevent stillbirths, miscarriages, and birth defects among the potential children fathered by male lead workers. Without MRP, many workers will fear that if they come forward and chance to have impaired reproductive ability, they will be discharged, laid off for several months, or transferred temporarily or permanently with a large cut in pay. In light of these fears, many male workers would keep quiet about their plans to father children and take the chance that their reproductive capacity was not seriously damaged. Mandating worker participation in medical surveillance without MRP will have no effect on this situation, because once again workers will keep quiet about their parenting plans.

A third factor relevant to mandating worker participation in medical surveillance is the proper role of Government in this sensitive area. Attempting to compel workers to subject themselves to detailed medical examinations presents the possibility of clashes with legitimate privacy and religious concerns. Health in general is an intensely personal matter, and this is particularly true with respect to lead medical surveillance due to the adverse effects of lead on male and female reproduction. Absent some compelling justification, OSHA is extremely hesitant to mandate detailed

inquiries into the most private aspects of the lives of American workers. Governmental coercion in this context would often prove counterproductive to the goal of achieving meaningful worker participation in medical surveillance. Finally, personal privacy and religious concerns arise irrespective of the provision of MRP benefits to removed workers. Thus, mandatory worker participation with MRP is no more satisfactory an alternative than mandatory worker participation without MRP.

The lead record reveals that many employers either currently mandate that their employees participate in medical surveillance programs, or advocate mandatory participation as their management philosophy. (Tr. 7799, 8461; Ex. 3(52), p. 1; Ex. 3(118), p. 11; Ex. 354E, p. 3; Ex. 354F, pp. 1, 3; Ex. 354H, p. 3; Ex. 354I, p. 2; Ex. 354L, p. 1; Ex. 354(O), p. 4; Ex. 354P, p. 3; Ex. 354Q, p. 3; Ex. 354U, p. 4; Ex. 354V, p. 3; Ex. 354W, p. 1; Ex. 354Y, p. 6; Ex. 354Z, p. 1; Ex. 354DD, p. 4; Ex. 354HH, p. 3; Ex. 354II, p. 3; Ex. 375, p. 5; Ex. 394D, p. 3; Ex. 396A, p. 5; Ex. 397A, p. 7; Ex. 402, p. 11; Ex. 447, pp. 2-3; Ex. 456, p. 9; Ex. 457, pp. 23, 40; Ex. 463, p. 2). Some worker representatives voice similar feelings on this issue. (Tr. 7622-7623; Ex. 410A, pp. 2, 5; See also, Tr. 7199; Ex. 452, p. 79) Without passing judgment on these practices and approaches, OSHA has decided against the agency mandating worker participation as part of the lead standard. The agency may well have adequate legal authority to adopt a mandatory participation requirement but has chosen not to do so on factual and policy grounds. An effective medical surveillance program cannot exist without workers having faith and confidence in the confidentiality, concern, and competence of company medical personnel. A close doctor-patient relationship is needed, especially where such intensely personal and private issues as health and reproduction are matters of concern. Such a position of trust and confidence can only be earned—it cannot possibly be created by governmental coercion. Rather than try to force worker cooperation, OSHA urges that employers and employees work together to evolve a successful medical surveillance program which encourages and achieves meaningful worker participation. Under the most unusual of circumstances, such as those presented by commercial diving operations (29 CFR 1910.411), OSHA would consider mandating worker participation in medical surveillance. OSHA, however, sees no compelling reason for the agency to mandate participation in the context of the lead standard, either in lieu of or in combination with the provision of MRP benefits.

Mandating that temporary medical removals occur only at the election of individual workers at risk of material impairment.—A second alternative to MRP considered by OSHA was to mandate that temporary medical removals occur only at the election of individual workers at risk of material impairment. This approach would preclude the removal of a worker clearly at risk of health impairment unless the worker consented to the removal. Since the worker would control the immediate consequences of participation in medical surveillance, all reluctance to participate should disappear, thereby removing the need for MRP. This alternative, however, would merely inform workers of their current medical status without providing affirmative protection to those who need it. Absent MRP, far too often workers who should be removed from further lead exposure would choose not to be. Employers would even be prevented from utilizing removal in situations where it was imperative. These results are inconsistent with the preventive purpose of the Act, and thwart the level of health protection which temporary medical removals can provide. Earlier in this Attachment, OSHA explained in detail the functions which temporary medical removals serve, and the necessity for OSHA mandating temporary medical removals in the final lead standard. These considerations dictate that OSHA reject any alternative to MRP which reduces the effectiveness of temporary medical removal as a protective mechanism.

Permitting the use of respiratory protection in lieu of temporary medical removals.—A third alternative to MRP considered by OSHA was to permit the use of respiratory protection in lieu of temporary medical removal. Under this view, some form of specialized respiratory protection would come into force once a worker's blood lead level or other medical factor indicated that the worker was at increased risk of material impairment to health. For example, a respirator might be required where none was worn before, or a new respirator having a higher protection factor might be substituted for the form of respirator previously worn. This new respirator regimen would then be relied upon to prevent further harmful exposure, and allow the worker's blood lead level (or other biological index) to gradually return to an acceptable condition. Due to the use of respirators, no physical removal of the worker from lead exposure would be required, therefore no MRP benefits need be provided.

OSHA has rejected this respirator alternative to MRP due to the inherent limitations of respiratory protection. The blood lead level triggers set for the removal of a worker are such that any further substantial exposure of lead presents unacceptable risks to the worker's health. The need to temporarily remove a worker under these circumstances is essentially a matter of medical necessity. Relying on a respirator to protect a worker from exposure beyond such a point is simply too risky in light of the numerous inadequacies of respiratory protection—inadequacies described in detail elsewhere in the preamble. OSHA's decision to reject this use of respirators is supported both by the National Association of Manufacturers (Ex. 354(O), p. 3) and by the East Penn Manufacturing Co., a manufacturer of storage batteries. (EX. 354II, p. 2.)

To conclude that respirators are not an acceptable alternative to MRP is by no means to eliminate a role for them. Respiratory protection, along with engineering and work practice controls, hygiene practices, etc., is one means of seeking to assure in advance that no worker need ever be removed. The need to temporarily remove a worker due to medical reasons will infrequently arise without advance warning. For example, in most cases, a worker's blood lead level will have been increasing over many months before the blood lead level removal trigger is exceeded. By closely following a worker's biological condition, an employer can take individual precautionary measures as dictated by the application of sound industrial hygiene principles. Respiratory protection may very well be dictated under the circumstances. If respiratory protection does prove to be totally effective in practice, then there will be no need to temporarily remove the worker. As a result, although OSHA rejects respiratory protection as an alternative to MRP, experience should demonstrate that respirators play a constructive role in preventing temporary medical removals from occurring.

e. *Feasibility.*

Overview of the phasing-in of medical removal protection.—Two competing goals shaped OSHA's adoption of a 4-year phasing-in process for MRP. OSHA sought to quickly require the application of the ultimate MRP removal and return criteria so as to maximize the level of health protection which MRP will afford. At the same time, however, OSHA sought to gradually implement MRP so that employers would have a reasonable opportunity to reduce their employees' blood lead levels before particular blood lead level removal triggers came into effect.

This 4-year process incorporates the following elements: (1) upon the effective date of the standard, the temporary medical removal of employees

having blood lead levels at or above 80 μg/100 g of whole blood; (2) 1 year after the effective date of the standard, the temporary medical removal of those having blood lead levels at or above 70 μg; (3) 2 years after the effective date of the standard, and thereafter, the temporary medical removal of those having blood lead levels at or above 60 μg; (4) 4 years after the effective date of the standard, and thereafter, the temporary medical removal of those having 6-month average blood lead levels at or above 50 μg; and (5) upon the effective date of the standard, and thereafter, the temporary medical removal of employees found by physician determinations to be at risk of sustaining material impairment to health. The effect of this 4-year phasing-in process is that employers who comply with the new lead standard should face minimal economic impact from MRP's existence.

MRP as structured in the final standard is a feasible regulatory device. The imposition of ultimate blood lead level removal criteria in phases will permit firms to gradually reduce current blood lead levels and thus avoid most temporary medical removals. Disruption of plant production operations should be minimal since few removals will occur. The gradual phasing-in schedule will enable employees to structure their production operations so that transfer opportunities are provided to all removed workers. Four years will allow collective bargaining agreements to be altered if necessary so that all removals can be smoothly accommodated. Since full implementation of feasible engineering controls throughout the lead industry will impose substantial costs on several industry segments and since MRP as a control mechanism is of secondary importance to primary control measures such as engineering controls, OSHA has chosen to phase-in MRP slowly. Firms will therefore be able to avoid the possibility of MRP costs interfering with the rapid elimination of harmful lead exposure. As a result, firms that comply with the new standard should be able to avoid virtually all MRP costs. OSHA recognizes that the 4 years provided for the full implementation of MRP necessarily includes some short-term compromising of optimal worker protection. The agency is convinced, however, that this drawback is outweighed by the assurance the MRP can be implemented in an orderly fashion without significant disruption to any segment of the total lead industry.

Impossibility of immediate implementation of the ultimate MRP program.—The weight of the evidence in the lead record demonstrates that immediate imposition of the entire ultimate MRP program is not feasible. Put simply, existing worker blood lead levels are so high that major segments of the lead industry would have to immediately remove at least 25 percent to 40 percent of their production work force from lead exposure. Sufficient transfer opportunities would not exist thus extensive layoffs would result with accompanying MRP costs. Though OSHA has not made detailed cost calculations, we are convinced that major segments of the lead industry would be significantly impacted by these layoffs. Most firms have low worker blood lead levels and would not be so heavily impacted; other firms could shoulder such large costs and survive. However, OSHA is persuaded that several industry segments could not reasonably be expected to comply with an immediate imposition of the overall MRP program.

The lead record contains considerable blood lead level distribution data which bear out the preceding statements. Tables C-1, C-2, C-3, and C-4 summarize the record evidence on blood lead level distributions found within the battery industry, the primary lead smelting industry, the secondary lead smelting industry, and in other lead plants. Tables C-1, C-2, and C-3 suggest that over 30 percent of battery workers, 25 percent of primary lead smelting workers, and over 40 percent of secondary lead smelting workers, respectively, have blood lead levels at or above 60 μg/100 g of whole blood. Table C-4 indicates that blood lead level distributions in other particular lead industry plants are comparable. Individual plant blood lead level distributions vary dramatically within these four tables, but OSHA believes that the aggregate data presents a representative overview of existing worker blood lead levels.

[4510-26 C]

TABLE C-1

OBSERVED BLOOD LEAD DISTRIBUTIONS IN THE BATTERY INDUSTRY

FIRM	Percent of Workers in Given Blood Lead (ug/100g) Range						Total Number of Workers	Exhibit Number
	<40	40-49	50-59	60-69	70-79	>80		
Health Research Group	31.9	← 50.5 →		← 16.2 →		1.4	210	146 A
Delco-Remy	38.6	46.6	13.2	1.5	0	0	765	29(12)
Chloride	1.5	9.1	31.8	33.3	15.2	10.6	66	100A
UAW	16.0	26.0	18.1	18.1	12.0	10.1	393	404B
	10.1	18.0	19.0	20.3	15.2	17.1	316	"
	7.0	←	67.0	→	16.0	9.0	1083	"
NIOSH Study	33.3	37.6	22.4	6.1	.6	0	330	334
Estee	10.6	21.7	30.7	25.3	10.4	1.1	74	315A, B
Bell City	5.6	22.2	11.1	27.8	22.2	11.1	18	312
Battery Systems	64.4	22.2	8.9	4.4	0	0	45	297 C1-C10
General Battery	15.9	19.0	24.6	22.2	13.4	4.7	126	297 C1-C10, 152
Gould	7.5	32.5	27.5	25.0	5.0	2.5	40	297 C1-C10
Teledyne	19.3	25.0	20.7	23.6	8.6	2.9	70	297 C1-C10
Trojan	30.8	31.9	23.3	10.9	2.3	0.6	68	297 C1-C10
Plates	13.4	21.6	37.1	23.7	3.1	2.1	24	297 C1-C10
Prestolite	--	--	--	33.3	→		?	297 C1-C10
C and D Battery	61.8	←	32.7	→	← 5.3 →	0.2	1020	354Q
Globe-Union				3.3	0.8	0	?	235
Voltmaster	15.6	34.5	31.3	18.6	0	0	32	293
Total N	743	1040	761	570	338	228	3680	
Total Percent	20.2	28.3	20.6	15.5	9.2	6.2	100.0	
D.B. Associates Study Percent	←		69.2	14.7	9.0	7.0	N=12800	

TABLE C-2

DISTRIBUTION OF BLOOD LEAD LEVELS IN THE PRIMARY SMELTING INDUSTRY

FIRM/PLANT	Percent of Workers in Given Blood Lead (ug/100g) Range						Total Number of Workers	Exhibit Number
	<40	40-49	50-59	60-69	70-79	≥80		
ASARCO								3(106)
East Helena	18.6	←40.5→		←38.7→		2.2	274	"
Omaha	16.3	←48.5→		←33.2→		2	295	"
Glover	27.8	←55.0→		←13.2→		4	151	"
Whiting	38.4	←52.0→		←9.6→		0	125	"
El Paso	43.3	←45.2→		←11.3→		0.1	876	"
St. Joe								
Herculaneum (76)	13.9	19.0	23.4	25.9	13.1	4.6	563	3(103)
" (77)	22.8	18.6	23.4	19.9	13.2	2.1	607	407(B)
J.W. Lorio	0	25.0	29.2	33.3	12.5	0	24	122(A)
Bunker Hill (NIOSH)	25.6	16.7	25.2	18.7	7.3	6.5	262	299
Total N	(852)		(1415)		(835)	(75)	(3177)	
Total Percent	26.8	←44.5→		←26.3→		2.4	100.0	
D.B. Associates Study Percent	19.0	21.0	25.0	20.0	11.0	4.0	N=3055	

TABLE C-3

DISTRIBUTION OF BLOOD LEAD LEVELS IN THE SECONDARY SMELTING INDUSTRY

SOURCE	Percent of Workers in Given Blood Lead (ug/100g) Range						Total Number of Workers	Exhibit Number
	<40	40-49	50-59	60-69	70-79	≥80		
California	17.1	← 61.6 →		← 20.2 →		1.1	99	86C
4 Smelters	17.9	← 15.4 →		← 33.3 →		33.3	78	86B
	15.4	← 15.4 →		← 43.6 →		25.6	39	"
	3.4	← 17.2 →		← 6.8 →		72.4	29	"
	7.9	← 15.8 →		← 44.7 →		31.6	38	"
Indianapolis	1.3	← 21.8 →		← 48.1 →		28.8	156	23 (Lillis)
ASARCO								3 (106)
Perth Amboy	75.7	← 24.3 →		← 0 →		0	115	"
Newark	47.4	← 36.1 →		← 15.8 →		0.8	133	"
Keystone	51.4	21.6	27.0	0	0	0	37	430G-1
Total N	(212)		(218)		(178)	(116)	(724)	
Total Percent	29.3	← 30.1 →		← 24.6 →		16.0	100	
Lead Industry Association Study	← 38 →		24	19		19	N=?	354(AA)

TABLE C-4

BLOOD LEAD DISTRIBUTIONS IN OTHER LEAD INDUSTRIES

INDUSTRY/SOURCE	Percent of Workers in Given Blood Lead (ug/100g) Range <40	40-49	50-59	60-69	70-79	>80	Total Number of Workers	Exhibit Number
Pigments								
D.B. Associates	1.9	← 15.1 →		← 47.2 →		35.8	53	26
King	26.4	14.7	44.1	11.8	2.9	0	34	234(22)
Zinc Smelting								
St. Joe Josephtown	76.7	16.7	5.3	1.3	0	0	150	3 (103)
Steel								
Bethlehem	58.8	17.6	11.8	5.9	5.9	0	17	44, 237
Printing								
R.M. Banknote	91.3	8.7	0	0	0	0	23	38B
Lead Products								
NL Industries	←		78.2	←	18.0 →	3.8	2501	394C
NL-Hoyt Plant	10.7	0	17.9	14.2	25.0	32.8	28	23(NIOSH)
Soldering								
Western Electric	100.0	0	0	0	0	0	37	3(79)
Lead Chemicals								
Eagle Pitcher (NIOSH)	5.9	5.9	17.6	5.9	23.5	41.2	17	113
Eagle Pitcher (Data)	←		15.4	48.1	→	36.5	52	38(C)

The tables paint a bleak picture of existing worker blood lead levels, but the situation can and should rapidly improve upon the implementation of the protective measures required by the final standard. A significant contributing factor to existing high blood lead levels has been widespread noncompliance with the 7-year-old 200 μg/m³ air lead standard. Noncomplying employers can quickly eliminate this contribution to their employee blood lead level distributions. Current industry blood lead level distributions are also a function of numerous other factors, all of which will be impacted by the new standard. The final standard sets a permissible exposure limit of 50 μg/m³ TWA. Although engineering controls to meet this level may require years to implement, respiratory protection to a 50 μg/m³ level is immediately required. Worker blood lead levels should quickly begin to fall as carefully designed and managed respiratory protection programs are implemented. The lead record indicates that poor industrial hygiene practices such as poor housekeeping, sloppy work practices, inadequate hygiene facilties, superficial respiratory protection, etc. have been common throughout the lead industry in the past. (Tr. 1257, 2173-74, 2181-83, 2196-98, 2530-36, 2577-93, 2614-16, 2983-21 to 24, 3635, 4720-21, 4761, 4762, 4786, 4789, 4793-96, 4834-46, 4840, 4878-79, 4991-97, 5003-04, 5038, 5241-46, 5279-87, 5295, 5311-12, 5506-09, 5516, 5518, 5530, 5559, 5561, 5562, 5565-71, 5580-82, 5592, 5636-37, 5835-37, 5857, 5863, 6026, 6026, 6038-39, 6040-41, 6107, 6139, 6154, 6156, 6207, 6209-10, 6258, 6284, 6287, 6289-90, 6292, 6297, 6317, 6329, 6876-78, 6881, 7616-17; Ex. 3(111), pp. 14-16) The final standard addresses all of these matters by establishing protective requirements which employers can promptly and feasibly implement.

The intangible factors of employer and worker knowledge and perception will also be influenced by the final standard, and these two factors may well have the largest influence on the expeditious reduction of existing blood lead levels. The lead record contains numerous assertions by employer representatives to the effect that a blood lead level of 80 μg/100 g of whole blood is an absolutely safe level, with no possible harmful effects ever occurring at lower blood lead levels. (Ex. 3(4), p. 2; Ex. 3(65), pp. 2, 4-5, 11; Ex. 3(72), pp. 17-20; Ex. 3(74), p. 2; Ex. 3(96), p. 1; Ex. 3(106), Comments, p. 1; Ex. 28(16), p. 1; Ex. 93, pp. 1, 5, App.) Undoubtedly, many firms have believed such assertions to be true, and thus have seen little need for corrective measures to respond to worker blood lead levels below 80 μg. The health effects sections of the preamble, however, demonstrate that an 80 μg blood lead level is not a safe level. Assertions that 80 μg is a safe level may once have been fully justified in light of the then existing state of medical knowledge, but must now be discarded. As employer knowledge and appreciation of the health implications of long term blood lead levels in excess of 40 μg/100 g of whole blood increases, worker blood lead levels will rapidly decline since employers will manage their operations so as to treat lead with the care it deserves.

Changed worker understanding and perception should have a similar effect. In many instances workers have never been told that blood lead levels below 80 μg are a matter of concern; certainly few employers have provided such information. As a result, many workers have probably been totally unconcerned about their personal blood lead levels. The final standard, particularly given the education and training provisions, will spark new worker interest in eliminating all contributing factors to elevated blood lead levels. Better personal hygiene habits, work practices, housekeeping and equipment maintenance procedures should result simply from new worker appreciation that these matters are vitally important to health.

Immediate 80 μg blood lead level removal trigger.—The preceding discussion demonstrates that existing worker blood lead levels should begin to quickly decline upon compliance with the new standard's requirements. MRP blood lead level removal triggers are slowly implemented consistent with modest goals for the decline of worker blood lead levels. An 80 μg/100 g of whole blood removal trigger is imposed immediately upon the effective date of the standard, and continues for one year thereafter. This 80 μg figure is essentially a 1 year continuation of the status quo since many of the firms in the most heavily impacted segments of the lead industry already remove workers whose blood lead levels exceed 80 μg, and provide economic protection to those removed.

The economic impact of the 80 μg removal trigger during the first year of the lead standard will be minimal. Tables C-1 through C-3 indicate that 6, 3, and 16 percent of exposed workers in the battery industry, primary lead smelting, and secondary lead smelting industries, respectively, may require removal. Many of these workers are probably already on removal status due to existing employer policies. The standard permits workers removed with blood lead levels in excess of 80 μg to be transferred to positions having an air lead level (without regard to the use of respirators) below 100 μg/m³. Abundant transfer opportunities already exist to accommodate these removals. (See, Ex. 26, pp. 3.4, 5.44; Ex. 334, Tables 8, 9; Ex. 404B, Att. B; Ex. 407B, Ex. C; Ex. 430G-1, Tables 3, 4.) Greater transfer opportunities will arise as the new standard is implemented.

The costs to employers of transferring small percentages of their work force will be insignificant. As part of the lead proceeding, the Center for Policy Alternatives estimated the first year direct cost of an 80 μg removal trigger (ignoring costs already regularly absorbed by existing industry transfer programs) to be as follows: (Ex. 439A, table 7.1).

Industry	Direct costs assuming transfers occur
Battery Manufacturing	$500,555
Primary Lead Smelting	44,209
Secondary Lead Smelting	300,018
Inorganic Pigments Manufacturing	359,100

The Center for Policy Alternatives termed these costs "so low on an absolute scale that it is ulikely that the 80/60 (first year 80 μg) regulation will have a significant impact on any of the industries which have been examined in this report." (Ex. 439A, p. 7-3.) No industry representative disagreed with this conclusion nor do we. And, to the extent that any costs exist, it is important to emphasize that these costs are largely a function of noncompliance with the prior 200 μg/m³ standard.

70 μg blood lead level removal trigger 1 year after the effective date of the standard.—One year after the effective date of the standard, a 70 μg blood lead level removal trigger comes into force. To avoid any economic impact from this requirement, employers need only accomplish minor declines in the blood lead levels of some of their employees. Those workers having blood lead levels between 70 μg and 79 μg need decline no more than 10 μg in 12 months—less than 1 microgram per month. As explained earlier, major blood lead level declines are to be anticipated soon after the effective date of the standard provided employers comply with the new standard. Due to this, OSHA is convinced that few workers should have to be removed once the 70 μg removal trigger comes into force. Since few removals should occur and numerous transfer opportunities will exist, the economic impact of this trigger should be insignificant.

The reasonableness of the conclusion that few workers should have blood lead levels in excess of 70 μg 1 year after the effective date of the standard is borne out by the dynamic air lead/blood lead modeling of MIT's Center for Policy Alternatives. First, it

is clear that those workers initially removed with blood lead levels in excess of 80 μg should all have declined to below 60 μg before the end of the first year of the standard, thus will be unaffected by the 70 μg removal trigger. The Center for Policy Alternatives modeled the consequences of exposing workers having blood lead levels betwen 70 μg and 79 μg (average 75 μg blood lead level) to 50 μg/m³ of lead for 1 year after the effective date of the standard. (Ex. 439B, addendum to p. 4-29.) This scenario parallels the minimum that should occur in reality. The following average blood lead levels (depending on job tenure) resulted after 1 year of 50 μg/m³ air lead exposure: (Ex. 439B, Table Ad. 2).

JOB TENURE (yrs)	0-1	1-5	5-10	10-20	Over 20
AVERAGE PbB	41.3	49.9	54.4	56.7	58.6

Assuming a normal distribution about each of these averages with a standard deviation of 9.5 μg, one can apply standard statistical tables to achieve the percentage of each job tenure group which would equal or exceed 70 μg 1 year after the effective date of the standard:

JOB TENURE (yrs)	0-1	1-5	5-10	10-20	OVER 20
PERCENTAGE OVER 70 ug	0.2%	1.7%	5.1%	7.5%	11.5%

Finally, one can form a weighted average for a total population by multiplying each job tenure by its relative proportion of the typical manufacturing industry work force. (Job tenure distribution for all manufacturing industries in 1973: 0-1 yr. (19.6 percent); 1-5 yr. (28.4 percent); 5-10 yr. (18.9 percent); 10-20 yr. (17.6 percent); greater than 20 yrs. (15.5 percent), from "Job Tenure of Workers—1973", Special Labor Force Report No. 172, BLS Monthly Labor Rev. (Dec. 1974); See also, Ex. 439A, p. 3-18.) A weighted average slightly less than 5 percent results. This 5-percent figure means the following: Of 100 workers with blood lead levels between 70 μg and 79 μg at the effective date of the standard, less than 5 workers should continue to exceed 70 μg 1 year later. Tables C-1 to C-3 indicate that workers with blood lead levels between 70 μg and 79 μg probably comprise less than 20 percent of each segment of the lead industry. As a result, 1 year after the effective date of the standard less than 1 percent of the existing work force should exceed 70 μg.

60 μg blood lead level removal trigger 2 years after the effective date of the standard.—Two years after the effective date of the standard, a 60 μg blood lead level removal trigger comes into force. To avoid any economic impact from this requirement, employers can take advantage of 24 months given to improve working conditions so that current worker blood lead levels between 60 μg and 80 μg will decline to below 60 μg. Two years is an adequate period of time to accomplish this goal. Tables C-1 to C-3 indicate that numerous individual companies in the highest impacted segments of the lead industry already have effected such declines. Two years are thus provided for all employers to achieve results comparable to what some employers have today.

The Center for Policy Alternatives modeling demonstrates the reasonableness of the conclusion that few workers should exceed 60 μg 2 years after the effective date of the standard. Workers originally having blood lead levels at or above 80 μg should have declined to below 60 μg within the first year of the standard's effect. Workers originally having blood lead levels between 70 μg and 79 μg should have declined within the first year of the standard such that 95 percent are below 70 μg. As noted earlier, the average blood lead level of each job tenure subgroup should have declined to below 60 μg. As a result, workers originally having blood lead levels between 70 μg and 79 μg should decline during the second year of the standard even more so than workers originally having blood lead levels between 60 μg and 69 μg (average 65 μg) declined during the first year of the standard. The Center for Policy Alternatives modeled the consequences of exposing workers having blood lead levels between 60 μg and 69 μg (average 65 μg blood lead level) to 50 μg/m³ of lead for 1 year after the effective date of the standard. The following average blood lead levels (depending on job tenure), and percentages over 60 μg (using statistical tables as before), resulted from this simulation: (Ex. 439B, table Ad. 2)

JOB TENURE (yrs)	0-1	1-5	5-10	10-20	OVER 20
AVERAGE PbB	41.3	47.2	50.7	52.6	54.2
PERCENTAGE OVER 60 ug	2.4%	8.9%	16.4%	21.8%	27.1%

The weighted average for a total worker population (using job tenure distribution as before) would be 14 percent. The foregoing suggests that of all workers originally having blood lead levels between 70 μg and 79 μg at the start of the standard, much less than 14 percent would exceed 60 μg 2 years later. Since workers now between 70 μg and 79 μg comprise less than 20 percent of the existing work force in each of the most heavily impacted industry segments, after 2 years of the standard much less than 3 percent of the existing work force of even those segments would be expected to exceed 60 μg.

The preceding "much less than 3 percent" figure would only be affected slightly by workers originally having blood lead levels between 60 μg and 69 μg. The previous paragraph's calculations indicated that less than 14 percent of these workers would exceed 60 μg after 1 year. One would expect most of these workers still above 60 μg to be very close to having a 60 μg blood lead level. One extra year should be sufficient for these few workers to decline one or two additional micrograms so that only extremely rare individuals would still exceed 60 μg.

Six-month 50 μg average blood lead level removal trigger 4 years after the effective date of the standard. The standard provides that the 6-month 50-μg average blood lead level removal trigger comes into force 4 years after the effective date of the standard. In essence, this gives employers 2 additional years after the 60-μg removal trigger comes into force to shift the blood lead levels of employees still between 50 μg and 60 μg down below 50 μg. Two years were provided instead of some shorter period since the rate of worker blood lead level declines between 60 μg and 50 μg will likely be somewhat slower than the rate of declines between 70 μg and 60 μg. In total, however, a full 4 years is provided for all segments of the lead industry to achieve worker blood lead level distributions which numerous employers in the most heavily impacted segments are close to achieving already.

Impact of ultimate blood lead level removal criteria. The preceding paragraphs explain that MRP costs should be insignificant during the first 4 years of MRP's existence. MRP costs in subsequent years should decline even further. Air lead exposures will drop with implementation of new engineering controls, and with increasing

employer experience in comprehensively controlling occupational lead exposure from all sources. Long term lead workers will gradually retire from the industry so that plant blood lead level distributions become less and less a function of previous lead exposure in excess of 50 $\mu g/m^3$. All of these factors will result in fewer and fewer removals due to elevated blood lead levels. Employers will be able to guarantee the availability of transfer opportunities to most removed workers so that the costs of a layoff with MRP benefits need rarely be incurred.

The Center for Policy Alternatives dynamic air lead/blood lead modeling included long run projections of what percentages of a work force would exceed 50 μg and 60 μg if all workers were exposed to air lead levels comparable to compliance with the 50 $\mu g/m^3$ PEL (supra, att. A(6).) This modeling suggests that 5.5 percent of a population would have blood lead levels between 50 μg and 60 μg, while 0.5 percent would have blood lead levels in excess of 60 μg. Thus an employer in total compliance with the final standard should never have more than 6 percent of the work force subject to removal at any time. In reality, much smaller percentages will be involved for two simple reasons. First, air lead levels within lead plants necessarily vary from department to department. When all departments are below 50 $\mu g/m^3$, many departments will be significantly below this figure. Many workers will have an exposure to lead far less than 50 $\mu g/m^3$, and overall plant blood lead level distributions will reflect this fact. Second, employers will likely want to avoid temporary medical removals where possible, therefore they will closely follow each employee's blood lead level. Special attention will be paid to workers whose blood lead levels are approaching the removal triggers. Engineering or work practice controls might be strengthened, respiratory protection might be invoked where appropriate, or other industrial hygiene measures might be applied so as to reduce a worker's effective exposure to lead. With experience, employers will have both the ability and opportunity to preclude most temporary medical removals; consequently the economic impact of MRP should be trivial.

Immediate removal due to physician determinations. No phasing-in period is provided for temporary medical removals initiated by physician determinations; this MRP trigger comes into effect immediately. OSHA is convinced that this trigger for temporary medical removal will not impose substantial economic burdens on any of the segments of the lead industry because few workers should have to be removed due to a physician determination. Some fraction of the lead industry work force is currently at risk of material impairment due not only to elevated blood lead levels, but to the development of specific lead-related diseases or health impairments. The medical surveillance provisions of the standard, however, will serve to prompt the temporary medical removal of only some of these workers, since developing lead-related diseases such as nephropathy and peripheral neuropathy will often prove impossible or extremely difficult to detect. Essentially, OSHA anticipates that few workers will be removed during the first few years after the effective date of the standard due to a physician determination. And, as working conditions in the lead industry improve, even fewer such removals should occur.

TABLE C-5

Industry	Number of production employees	Number of lead-exposed employees	Average annual straight time earnings and benefits	Average annual hours worked	Annual cash flow (1977 dollars)
Primary lead smelting	2,500	2,216	$14,978	1,960	$118,226,000
Secondary lead smelting	3,336	3,166	13,820	2,024	146,527,000
Battery manufacturing	17,300	16,727	13,269	1,938	338,416,000
Inorganic pigments manufacturing	2,945	2,000	15,605	2,036	101,058,000

Quantification of potential MRP costs. MRP costs both in the short term and the long term should be small since employers will have the opportunity and ability to prevent most removals. It is reasonable to project that beginning in the second year following the effective date of the standard and continuing thereafter, no more than 2 percent of the lead exposed work force should be on removal status at any one time. The annual direct costs to the most heavily impacted segments of the lead industry of a 2-percent removal rate can be quantified using data contained in the Center for Policy Alternatives economic study. This study tabulated the number of production employees (Ex. 439A, p. 6-3), the number of lead exposed employees (Ex. 439A, p. 6-3), the average annual straight time earnings and benefits (Ex. 439A, p. 6-7), the average employee hours worked (Ex. 439A, p. 6-16), and the annual industry cash flow in 1977 dollars (Ex. 439A, pp. 7-6, 8-14) for several major segments of the lead industry. This data is contained in table C-5. The direct annual costs (before taxes) that four of these industries would incur if 2 percent of their work force were continuously removed can be computed from table C-5 in conjunction with the Center for Policy Alternatives' estimate that the transfer of a worker will cost an employer approximately $0.96 per hour: (Ex. 439A, p. 6-19.)

Direct annual costs if 2 percent of work force is continuously on transfer status

Industry	Total cost	Cost per exposed employee	Cost per production employee	Percentage of annual cash flow (percent)
Primary lead smelting	$84,672.00	$38.21	$33.87	0.072
Secondary lead smelting	124,354.56	39.28	37.28	.085
Battery manufacturing	623,260.80	37.26	35.01	.184
Inorganic pigments manufacturing	78,182.40	39.09	26.55	.077

Similar calculations can be performed of the direct annual costs (before taxes) if half of the removals of 2 percent of the work force took the form of layoffs with the remainder being transfers:

Direct annual costs if 1 percent of work force is continuously laid off, and 1 percent is continuously on transfer status

Industry	Total cost	Cost per Exposed employee	Cost per production employee	Percentage of annual cash flow (percent)
Primary lead smelting	$374,847.60	$169.16	$149.94	0.32
Secondary lead smelting	500,271.28	158.01	149.96	.34
Battery manufacturing	2,531,534.10	151.34	142.22	.75
Inorganic pigments manufacturing	351,191.20	175.60	119.25	.35

The foregoing data demonstrates that MRP should present only modest expenses to the lead industry; expenses which are minor in comparison to the expenditures which must be made to adequately reduce existing lead exposures. The per employee costs of MRP will be small, as will the percentage demand on the industry's cash flow resources. In addition, the actual industry costs of MRP will be halved by the Federal corporate income tax rate of 48 percent. The resulting overall costs of MRP will be small, and will be outweighed by the health benefits afforded by the MRP program.

Economic impact of MRP on less heavily impacted segments of the lead industry. The phasing-in periods for MRP are applied equally across all segments of the lead industry. The phasing-in of MRP has been designed so that even the segments of the lead industry most heavily impacted by the new lead standard—battery manufacturing, primary and secondary lead smelting, and pigment manufacturing—should not be appreciably disrupted by MRP. An immediate consequence is that the remainder of the overall lead industry should experience trivial MRP costs over time. These industries have substantially lower air lead levels with resultant lower blood lead level distributions. Few workers will be subject to blood lead level removal triggers in either the short or long term, thus causing little MRP expense to these firms. All employers are given a fair and reasonable opportunity to avoid practically all MRP costs by complying with the new lead standard.

MRP is a new program which incorporates preventive health concepts not present in the prior 200 $\mu g/m^3$ standard. The new standard will hopefully foster a higher level of concern for worker health by the overall lead industry. In this spirit, MRP is phased-in consistent with anticipated worker blood lead level declines so that MRP costs cannot be viewed as a penalty for past occupational health practices. All segments of the lead industry are thus provided the same periods of time to accomplish blood lead level declines, regardless of the percentages of an industry's work force in the higher blood lead level ranges. Though OSHA certainly encourages companies to implement the ultimate MRP blood lead level removal triggers immediately if possible, we have chosen not to make this a legal requirement.

Economic impact on small manufacturers. Several participants in the lead proceeding argued that MRP will have a far greater economic impact on smaller lead firms (Tr. 7460-61; Ex. 354(0), p. 4; Ex. 385, pp. 11-12; Ex. 397A, p. 5.) We would agree that in some instances companies such as small battery manufacturers might have less flexibility in creating transfer opportunities for removed workers than would larger firms. This does not, however, necessarily imply higher MRP costs for the small firm. In many respects the management of a small firm is in much closer contact with production operations and workers than comparable management in a large firm. A small firm thus has great opportunities to correct factors which might cause the elevated blood lead level of a particular worker. OSHA does not agree that small companies by virtue of their size are incapable of protecting worker health. And, the level of health protection an employer provides, not size, is the prime determinant of any firm's MRP costs.

The ability of small firms to accommodate MRP can best be seen in terms of the blood lead level distributions currently accomplished by the most diligent small firms. Dynalite Corp., a small battery company having about 20 employees, uses no respiratory protection but nonetheless maintains such low lead exposure that only an odd blood lead level slightly exceeds 60 μg. (Tr. 1240-41, 1245.) Keystone Resources, Inc., a secondary lead smelter employing 37 people at one of its plants, reports having no workers with blood lead levels in excess of 60 μg. (Ex. 430G(1).) These small firms will likely experience no MRP costs for at least 4 years, and other small firms which comply with the new lead standard can achieve the same result. Consequently, we reject suggestions that MRP will necessarily have a greater economic impact on small employers than on large employers.

3. *Summary and Explanation of the Medical Removal Protection Sections of the Standard.*

a. *Temporary medical removal and return criteria.* The standard establishes explicit removal and return criteria for the temporary medical removal of workers at risk of sustaining material impairment to health due to continued exposure to lead. Removal and return criteria apply to elevated blood lead levels and medical opinions.

Elevated blood lead levels. The standard establishes two ultimate blood lead level removal criteria which are phased-in over a multiple year period. Ultimately, temporary removal is required for any worker having a blood lead level at or above 60 $\mu g/100$ g of whole blood, and for any worker having an average blood lead level at or above 50 $\mu g/100$ g of whole blood over at least the past 6 months. These criteria are phased-in over a period of 4 years, with an immediate 80 $\mu g/100$ g of whole blood lead level removal criteria. A 70 $\mu g/100$ g of whole blood removal criteria becomes effective 1 year after the effective date of the standard. The 60 $\mu g/100$ g of whole blood removal trigger comes into force 2 years after the effective date of the standard, with the 6 month 50 $\mu g/100$ g of whole blood average removal trigger coming into force 2 years later. The decisionmaking involved in this lengthy phasein period is explained in the prior section of this attachment concerning the feasibility of MRP.

The ultimate blood lead level removal triggers are a function of the risks to health presented by elevated blood lead levels. The preamble's discussion of the numerous adverse effects of lead on the human body demonstrates that long-term blood lead levels in excess of 40 μg must be avoided. Blood lead levels in excess of 40 μg are a matter of concern, but long-term blood leads in excess of 40 μg are the main focus of concern. Convincing evidence demonstrates that long-term blood lead levels of approximately 40-60 μg yield serious health impairments in some workers. Such impairments are slowly acquired, although it is impossible to precisely quantify the minimum duration of elevated blood lead levels needed for the onset of material impairments in occupationally exposed workers. It is clear, however, that this minimum duration is in terms of months as opposed to days and weeks. OSHA does not feel that a short-term blood lead level elevation above 40 μg, in and of itself, merits the temporary medical removal of a worker. But, since many workers will spend much of their working years in lead exposed occupations, OSHA has determined that temporary medical removals are essential in situations where long-term blood lead levels are likely to significantly exceed 40 $\mu g/100$ g of whole blood.

The standard mandates that a worker immediately be removed from exposure to lead whenever a followup measurement indicates a blood lead level at or above 60 μg/100 g of whole blood. Although a blood lead level of 60 μg may not automatically be dangerous, such a level has serious implications. Blood lead levels slowly rise in response to moderate increases in lead intake. In general, several months would be needed for a worker's blood lead level to rise from 40 μg to 60 μg, unless the worker was exposed to lead levels grossly in excess of the standard's requirements. Also, blood lead levels are often likely to decline at a rate even slower than they previously increased. As a rough example, if it took a worker 2 months to rise from 40 μg to 60 μg, then it might take 3 or 4 months after removal for the worker's blood lead to return to 40 μg. Also, there is considerable individual variability in the rate of excretion of absorbed lead. Some workers will take many months to decline from 60 μg to 40 μg even though the higher blood lead level was quickly acquired.

The crucial result of a blood lead level of 60 μg is the high probability that it will represent numerous months of a blood lead level in excess of 40 μg during the overall period of absorption and excretion. Due to this, the agency has concluded that removal must be automatic whenever a worker's blood lead level equals or exceeds 60 μg/100 g of whole blood. Postponing mandatory removal until some higher blood lead level is reached merely invites the occurrence of material impairment. Each worker's health must be protected over an entire lifespan, and the goal of minimizing the duration of any and all blood lead level elevations in excess of 40 μg must be paramount. Removal is thus automatic at or above a blood lead level of 60 μg/100 g of whole blood.

As previously explained, prevention of material impairment to worker health and functional capacity dictates that long-term blood lead levels be maintained at or below 40 μg/100 g of whole blood, with elevations above 40 μg to be minimized. Removal is essential for workers whose blood leads are slowly but steadily increasing above 40 μg, and for workers whose blood lead levels are stabilizing appreciably above 40 μg. To respond to these and similar possibilities, the standard ultimately provides for the temporary removal of any worker having an average blood lead level of 50 μg/100 g of whole blood over the last three measurements, or the last 6 months, whichever is longer. If the average of these measurements equals or exceeds 50 μg, then there is the serious danger that the worker's long-term average blood lead level will significantly exceed 40 μg, and material impairment result. In light of such a 6-month average, removal is essential to protect worker health, unless the most recent blood sampling test indicates a blood lead level at or below 40 μg. Such a measurement would strongly suggest that whatever caused the worker's several most recent elevated blood lead levels no longer was in operation.

In setting the blood lead level removal criteria, OSHA has sought to minimize the effect of blood lead level measurement variability. Blood lead level measurements under the standard need only have an accuracy of plus or minus 15 percent (to a confidence level of 95 percent). Due to this factor, blood lead level removal triggers are set on the basis of more than one measurement. The standard provides that the 60 μg/100 g of whole blood removal criteria (and the phase-in 80 μg and 70 μg triggers) only comes into force when an initial blood sampling test, and a second follow-up blood sampling test conducted within 2 weeks after receipt of the results of the first test, both indicate that the removal trigger has been reached. Similarly, the standard provides that the 6-month 50 μg/100 g of whole blood average removal criteria only comes into force when the average of at least three measurements equals or exceeds 50 μg. By relying on multiple measurements before mandating the temporary removal of a worker, the standard greatly reduces the statistical probability that a worker's apparent blood lead level is largely a function of measurement error.

OSHA considered including in the standard a 40 μg/100 g of whole blood 6-month average removal trigger. At first glance, this would appear to be dictated by OSHA's goal to maintain long term blood lead levels at or below 40 μg. The practical operation of such a removal trigger, however, militates in favor of a higher figure for removal. For example, three blood lead level measurements of 46 μg, 34 μg, and 41 μg taken over 6-months would average over 40 μg/100 g of whole blood, but would indicate little about the likelihood of a worker's blood lead level continuing to average over 40 μg. Any or all three of the measurements could have been in error by plus or minus six micrograms and still be considered accurate in light of the inherent variability of blood lead measurement. As a result, a three measurement, 6-month-average blood lead level figure of 40 μg contains an appreciable margin of error, and may not be significant in terms of a worker's long term blood lead level. Removing a worker on the basis of such an average would often be overprotective and premature. Postponing removal until a worker's blood lead level averages 50 μg over 6 months virtually eliminates any possibility that the worker's true blood lead level is somewhat less than 40 μg. Waiting until an average of 50 μg is reached also guarantees that a blood lead level trend in excess of 40 μg has been established. For these reasons, the standard mandates removal based off of a 6-month 50 μg average removal trigger as opposed to some lower figure.

The standard provides that the return of a worker removed due to an elevated blood lead level is also governed by the worker's blood lead level. During the years that the ultimate removal criteria are being phased in, the return criteria have been set to assure that a worker's blood lead level has substantially declined during the period of removal. A worker removed due to a blood lead level at or above 80 μg must be returned when his or her blood lead level is at or below 60 μg/100 g of whole blood; if removed due to a level at or above 70 μg, return shall follow when a level of 50 μg/100 g of whole blood is achieved. Once the ultimate removal criteria have been phased in, return depends on a worker's blood lead level declining to 40 μg/100 g of whole blood. Any higher return criteria that 40 μg would be inconsistent with the goal of maintaining long term blood lead levels below 40 μg/100 g of whole blood. A lower return criteria than 40 μg could easily be justified to provide a margin of safety in case a worker's blood lead level began to climb anew. OSHA, however, does not presume that a worker's blood lead level will automatically begin to climb again once the worker is returned to his or her former job. Conditions may well have changed such that the reasons for the worker's elevated blood lead level no longer exist. If conditions have not changed, then the worker will likely soon be removed again due to an elevated blood lead level.

The standard permits return only when two consecutive blood lead level measurements indicate a blood lead level at or below 40 μg. The two-measurement restriction was chosen so as to demonstrate that the worker's blood lead level had stabilized at or below 40 μg, and to reduce the effect of possible measurement variability. Relying on one measurement at or below 40 μg runs the risk that the worker's true blood lead level is appreciably in excess of 40 μg. Relying on more than two measurements separated by time further reduces this risk, but adds the new risk of keeping a worker on removal status longer than is medically justified. The agency has attempted to balance the need to avoid premature return against the need to avoid unjustified burdens due to unnecessarily long periods of re-

moval. The two-measurement restriction was chosen with these interests in mind.

In structuring the standard's provisions requiring the temporary medical removal and return of workers with elevated blood lead levels, OSHA has closely examined existing industry practices. The lead record reveals that many of the employers in the primary sectors of the lead industry already include temporary medical removals due to elevated blood lead levels as part of their medical surveillance programs. (Ex. 26, p. 5-11.) Many firms remove workers having blood lead levels in excess of 80 μg/100 g of whole blood (Ex. 453, p. 15), while some companies have blood lead level removal criteria of 75 μg (Tr. 7846-47, 8458; Ex. 354(HH), p. 8) and 60 μg (Tr. 6202, 7629, 7706-07, 7901). One large paint manufacturer endorsed a blood lead level removal trigger of 40 μg. (Ex. 3(97), p. 2.) Although companies differ as to when they remove a worker due to an elevated blood lead level, it is clear that the concept of temporary medical removal and return due to elevated blood lead levels is a protective mechanism both known by and acceptable to management as well as labor. (See e.g., Ex. 354(AA), p. 12; Ex. 452, pp. 52-56; Ex. 453, pp 12-15.) Due to this, OSHA is confident that employers can quickly and easily implement the mechanics of temporary medical removals and returns.

The 1975 proposed lead standard contained a requirement that workers having a followup blood lead level at or above 60 μg/100 g of whole blood be provided within a week with a detailed medical examination to determine whether the employee was experiencing symptoms of lead intoxication. (40 FR 45934 (1975), to be codified in 29 CFR Section 1910.1025(K)(2)(ii)(B)(*l*).) A similar exam was required every other month until the employee's blood lead level declined below 60 μg/100 g of whole blood. (40 FR 45934 (1975), to be codified in 29 CFR section 1910.1025(K)(3)(ii)(c).) The final lead standard drops these requirements because OSHA no longer feels that they are necessary. Workers with elevated blood lead levels may or may not be experiencing symptoms of lead intoxication. Those who believe they are may immediately obtain a complete medical examination pursuant to the standard. To automatically provide repeated detailed medical examinations on the basis of an elevated blood lead level alone serves no substantial purpose. The blood lead level removal criteria were established due to their longrun implications for worker health, not due to an expectation that any particular blood lead level correlates with specific immediate symptoms of lead poisoning. The removal criteria are preventive-oriented in the hope that few workers will actually develop lead-related disease before removal occurs.

Final medical determinations. The standard mandates that an employee be removed whenever a final medical determination results in a medical finding, opinion, or recommendation that the employee has a detected medical condition which places the employee at increased risk of material impariment to health from exposure to lead. This removal criteria is tied to the medical surveillance provisions of the standard which require that such a medical judgment be made a part of written medical opinions. The term "final medical determination" refers to the outcome of the multiple physician review mechanism, or alternate physician mechanism, used pursuant to the medical surveillance provisions of the standard. These provisions also provide that written medical opinions contain any recommended limitation upon the employee's exposure to lead or use of respirators. Accordingly, the temporary medical removal and return portion of MRP mandates that an employer implement and act in accordance with these limitations so as to protect worker health. The requirement that an employer follow such recommendations was included as a part of the MRP portion of the standard since some limitations on an employee's exposure to lead will result in an employer having to provide MRP benefits.

Removal based on medical determinations was included in MRP as a necessary complement to removal based on elevated blood lead levels. Most temporary medical removals under the standard will occur due to elevated blood lead levels, but exceptions will arise. During the multiple year phasing-in of MRP, some workers will continue to have highly elevated blood lead levels and some of these workers will experience recognized symptoms of lead poisoning. Employees experiencing lead poisoning in any of its many forms deserve a temporary medical removal despite the fact that their blood lead levels do not yet require a removal. Even after MRP has been fully phased-in, situations may arise where, for example, lead poisoning occurs in a worker having a blood lead level below the removal criteria, or a worker acquires a temporary nonwork related medical condition which is aggravated by lead exposure. Temporary medical removal of these workers will also be necessary.

In addition, temporary medical removal may in particular cases be needed for some workers desiring to parent a child in the near future. Some males may need a temporary removal so that their sperm can regain sufficient viability for fertilization; some women may need a temporary removal to slightly lower their blood lead levels so that prior lead exposure will not harm the fetus. Some participants in the lead proceeding urged that OSHA provide voluntary transfers at the request of any male or female worker desiring to parent a child in the near future. (Tr. 691-92, 1170-71, 4139-40, 8224; Ex. 148, pp. 15-16; Ex. 452, p. 60.) Instead of providing an automatic transfer opportunity, OSHA has determined that questions concerning reproduction can best be addressed first by primary control measures protective of reproductive capability (*see* attachment B), and second, by the flexibility and informed medical judgment which will result from the medical surveillance and MRP provisions of the standard. Where medically indicated, temporary removal of workers intending to parent can be provided pursuant to a medical determination. Temporary removal is only one of several alternatives, however. For example, a physician might first recommend that a particular employee be provided with a powered air purifying respirator to use several hours each day, even though the worker would otherwise have no opportunity under the standard to obtain this form of respirator. If this respirator usage proved inadequate, the physician could later recommend complete removal of the worker from lead exposure.

The preceding use of special protective measures or temporary medical removals for those intending to parent equally applies to pregnant employees. The preamble's discussion of lead's effects on the reproductive system indicates that in many cases it would be unacceptable for a pregnant employee having an elevated blood lead level to continue to experience substantial lead exposure during the pregnancy. If the employee's blood lead level were only slightly elevated, and the employee's normal lead exposure were low or moderate, then removal of the employee might not be essential—perhaps the provision of a powered air purifying respirator would afford adequate protection to both the mother and the fetus. In other cases, physical removal of the employee from all significant lead exposure might be imperative due to such factors as (1) the employee's blood lead level, (2) the extent of the employee's prior exposure to lead, (3) the nature of the employee's present exposure to lead, or (4) the lack of other protective alternatives. As with other medical conditions, the nature of special protective measures which should be provided to pregnant employees will depend on the circumstances of each case. Temporary medical removal with MRP

benefits is certainly one option, however.

The preceding situations illustrate why OSHA has included removal based on medical determinations as part of the overall MRP regulation. Both worker and industry participants in the lead proceeding endorsed the concept of removal from lead exposure based on medical determinations (Tr. 7249-51; Ex. 354(AA), p. 13; Ex. 452, pp. 63-64; Ex. 453, p. 31), and the lead record reveals no controversy concerning the 1975 proposed standard's requirement that "In no case shall an employee be placed at increased risk of material impairment of health from such (existing) exposure" to lead. (40 FR 45934 (1975), to be codified in 29 CFR Section 1910.1025(k)(4)(iii).) The final standard does not explicitly define the term "material impairment to health" since no comprehensive definition is possible. OSHA relies on informed medical judgment due to the innumerable contexts in which a particular lead exposure may be unusually hazardous to a particular worker. OSHA's approach parallels that of ESB, Inc., a large battery manufacturer (Ex. 354(U), p. 1), who submitted the following comment:

> * * * (B)lood lead levels are the primary criterion for removal under ESB's present medical surveillance program. That program also provides, as any effective program must provide, for removal from lead exposure of individuals who develop diseases which may make them more susceptible to the effects of lead exposure. For instance, if a person develops a medical condition such as anemia from a bleeding ulcer or iron deficiency, the person should be removed from lead exposure even though his blood lead level may be below that which is known to cause anemia. It is neither possible nor advisable to attempt to identify all possible medical criteria and itemize them in a regulation or surveillance program description. The better approach is to acknowledge that such situations may arise and to place the determination as to whether an individual should be removed from his job in the judgment of a knowledgeable physician. (Ex. 354(U), p. 4.)

OSHA is confident that the physician determination mechanism provided by the final standard will result in an application of the term "material impairment to health" such that lead workers do not suffer diminished health, functional capacity, or life expectancy as a result of their work experience.

During the period of time that a worker is removed due to a medical determination, appropriate followup medical examinations are provided by the standard. When a final medical determination results in a medical opinion that the employee no longer has a detected medical condition which places the employee at increased risk of material impairment to health from exposure to lead, then the employer is required to return the worker to his or her former job status. Similarly, the employer must remove any other limitation placed on an employee when a final medical determination indicates that the limitation is no longer necessary.

As noted earlier, the MRP provisions require employers to implement medical opinions resulting from the standard's medical surveillance program. In so doing, however, the standard neither legitimizes nor authorizes the categorical exclusion of any class of people from lead-exposed jobs. The lead record demonstrates that numerous employers systematically exclude women of childbearing capacity from lead-exposed jobs. (Tr. 678-81, 1629-30, 1673-74, 4731-32, 4997, 5245, 5905, 7797-99; Ex. 3(71), p. 10; Ex. 3(105), pp. 6-7; Ex. 3(114), p. 3; Ex. 28(26), pp. 4-6; Ex. 29(30), p. 1; Ex. 86(D), pp. 2, 6; Ex. 164.) The PEL section of the preamble discusses why such exclusionary practices are not justified in the context of the new lead standard. Continuation of these exclusionary practices after the effective date of the standard will raise possible questions of compliance with the OSH Act, Title VII of the Civil Rights Act of 1964, and Executive Order 11246 concerning the equal employment opportunity practices of Federal contractors. This attachment is not the appropriate forum for the discussion and resolution of these issues, but explicit reference to the matter is appropriate. MRP is intended to provide special health protection to those employees who temporarily need it, and is not intended to insulate employers from the consequences of blanket exclusions of certain classes of workers.

b. *Removal from work at or above the action level.* In most cases where a worker is removed due to an elevated blood lead level or a medical determination, the standard provides that removal be from work having an exposure to lead at or above the action level. The standard, however, permits somewhat higher air lead exposure to workers removed due to elevated blood lead levels during the first 2 years of the phasing-in of the ultimate blood lead level removal criteria. (See discussion of feasibility of MRP.) Work having an exposure to lead at or above the action level refers to the worker's daily 8-hour time weighted average (TWA) exposure to lead. As in all cases where the term "action level" is used, exposure is to be computed without regard to the use of respirators. OSHA's choice of this job placement limitation for most removals was based on two objectives: First, to assure that a worker not be removed to work having lead exposure high enough to further increase risks to health; and second, to assure that a worker be removed to work having lead exposure low enough to enable the gradual excretion of excess lead so as to permit return of the worker to his or her former job.

Prohibiting placement of a removed worker in work at or above the action level is consistent with OSHA's choice of the standard's specific action level. The 30 $\mu g/m^3$ PbA TWA action level represents a point at which an employer's operations might begin to pose risks to some exposed employees. Medical surveillance of employees (along with other protective measures) begins at that point due to an expectation that adverse health effects from lead exposure might be found. We do not believe adverse health effects are to be expected in workers consistently exposed to lead at levels below the action level, and at such low exposure neither periodic medical surveillance nor other protective measures must be provided. Having carefully chosen the 30 μg action level on the basis of health considerations, we feel confident in concluding that the vast majority of workers at risk of material impairment to health would not face heightened risks if removed from exposure at or above the action level. Removal to any higher exposure would be unacceptable since lead exposure above the action level poses some health risk even to workers not already at risk of material impairment to health. Workers removed as a matter of medical necessity merit the protection that exposure below the action level provides, and thus the standard incorporates this limitation.

Removal to work below the action level is also essential to enable the gradual excretion of excess lead so as to permit the eventual return of the worker to his or her former job. In most cases of removal, one not only desires to prevent further risks to worker health, but more importantly desires to undo whatever damage has already been done to the worker's health. Chelation will rarely if ever be an appropriate means of treatment, thus reliance must be placed on the body's ability to gradually excrete previously absorbed lead. A rapid excretion rate is desirable since the worker is already at substantial risk of sustaining material impairment to health. The rate at which a worker naturally excretes absorbed lead would clearly be slowed, if not halted, by continued substantial exposure to lead. Since lead exposure below the action level should not yield unacceptable blood lead levels in the first instance, such low exposure will permit a worker's body to naturally excrete previously absorbed lead.

Elimination of all occupational exposure would maximize the rate of excretion, but would in practice mean that most removed workers would have to

be laid off as opposed to being transferred to a low exposure position. By choosing the action level, OSHA has sought a compromise between the conflicting goals of (1) assuring the rapid excretion of previously absorbed lead, and (2) enabling employers to transfer removed workers to lower exposure positions rather than lay them off. The dynamic air lead to blood lead modeling of MIT's Center for Policy Alternatives indicates that transfers to positions just below the action level would roughly double the period of removal as compared to laying off removed workers. (Ex. 439A, p. 4(13).) OSHA feels this probable result is not too great a price to pay in order to enable employers to transfer removed workers rather than lay them off.

OSHA recognizes that situations may arise where removal to lead exposure just below the action level is inadequate to protect worker health. These situations can and should be dealt with on an individual basis in the course of a thorough medical examination conducted pursuant to the standard. OSHA encourages physicians, where appropriate in individual cases, to recommend limitations on a worker's exposure to lead which are even more stringent than those mandated by the standard. An employer must implement such recommended limitations pursuant to the requirements of the standard. The standard implies no unnecessary restriction on a physician's ability to recommend individual actions more protective than the standard's requirements, but simply embodies the judgment that at a minimum, all removed workers must be removed from work having an exposure to lead at or above the action level.

c. *Return of an employee to his or her former job status.* The standard provides that once a period of removal or limitation has ended, an employee must be returned to his or her former job status. Former job status refers to the position the worker would likely be occupying if he or she had never been removed. In most instances, an employer must return a worker to the same job held just before removal occurred, but this will not always be so. The MRP program seeks to assure that a worker's rights and benefits are not damaged due to the need to temporarily remove the worker from lead exposure in order to protect his or her health. MRP in no way seeks to give a worker job security beyond that held in the absence of temporary medical removal. The standard explicitly states that the requirement that an employer return an employee to his or her former job status is not intended to expand upon or restrict any rights an employee has or would have had, absent temporary medical removal, to a specific job classification or position under the terms of a collective bargaining agreement. If, but for a temporary medical removal, a worker would now be working at the same position held just before removal, then the employer must return the worker to that job. Otherwise, the employer may return the worker consistent with whatever job assignment discretion the employer would have had if no removal had occurred.

d. *The implementation of temporary medical removals.* It is OSHA's intention that employers implement each temporary medical removal in a manner consistent with existing collective bargaining agreements. MRP is meant to override existing contractual obligations only to the extent that specific contract provisions directly conflict with the terms of MRP. MRP has been structured to guarantee the maximum employer flexibility in effectuating MRP while minimizing the possibility of conflicts with existing collective bargaining agreements or other relationships. The standard does not specify what an emloyer must do with a removed worker; practically any action is permisssible so long as the worker is not exposed to lead at or above the action lead. In most cases OSHA expects that a removed worker will be transferred to a low lead exposure position during the period of removal. As urged by the LIA, OSHA intends that a transfer be to work the employee is capable of performing located in the same geographical area as the employee's normal job. (Ex. 354(AA), pp. 19-20) Alternatively, the worker might work shorter hours at his or her normal job such that the daily time weighted average exposure is below the action level. The worker might even be temporarily laid off or arrangements might be made for the removed worker to temporarily work at a nonlead related facility (this being a form of transfer). OSHA's intention is that the choice between these or other alternatives be a prerogative of the employer unless this flexibility is altered by some countervailing obligation. A removed worker is provided no automatic right to veto an emloyer choice which meets the standard, but similarly, the standard provides an employer no right to override existing contractual commitments to either removed employees or to other employees.

The mechanics of each removal is a matter for the employer, the removed employee, and his or her collective bargaining representative if any, to work out in the context of the preceding principles. Some employers and unions may decide to modify their contractual agreements to specify how each removal will be accomplished. The 4-year period during which the overall MRP program is phased in will provide ample opportunity for companies and unions to negotiate as to the implementation of MRP. Most collective bargaining agreements are of short-term duration—typically 2 or 3 years. Of 33 collective bargaining agreements in the lead record with identifiable durations, 3 have a 1-year term (Ex. 381(C); Ex. 300B (Texaco); Ex. 426), 8 have a 2-year term (Ex. 381(H); Ex. 400B (Amoco); Ex. 400B (Atlantic Richfield); Ex. 400B (Mobil); Ex. 415(C); Ex. 430(D) (Gould); Ex. 430(D)(6); Ex. 430(D)(14)), and 22 have a 3-year term (Ex. 157; Ex. 158; Ex. 261; Ex. 368; Ex. 369; Ex. 381(I); Ex. 388; Ex. 389; Ex. 400B (GATX); Ex. 400B (Kawecki); Ex. 401(B); Ex. 404B(D)(1); Ex. 404B(D)(4); Ex. 404B(D)(5); Ex. 423; Ex. 424; Ex. 425; Ex. 427; Ex. 415(A); Ex. 415(B); Ex. 430(D)(1); Ex. 430(D)(28)). If negotiations are deemed necessary, OSHA is confident that employers and affected unions can resolve the situation so that temporary medical removals will be implemented in an efficient and mutually agreed upon fashion. The collective bargaining process is well suited to accomplish this object. (See, e.g., *United Steelworkers of America* v. *Warrior and Gulf Navigation Co.*, 363 U.S. 574, 578-81 (1959).)

During the course of the lead proceeding, several industry representatives argued that MRP posed substantial conflicts with collective bargaining agreements. Statements were made that MRP could clash with established job bidding and transfer rights (Tr. 7462, 7471-73, 7511-12; Ex. 354(F), p. 2; Ex. 354(W), p. 2; Ex. 354(Y), p. 3; Ex. 385, pp. 15, 18, 21-22; Ex. 391, p. 4; Ex. 402, p. 9; Ex. 453, pp. 36-41; Ex. 457, pp. 29-30), and several industry spokesmen expressed skepticism that collective bargaining would be able to resolve difficulties presented by MRP. (Tr. 7534, 7789-90; contra, Tr. 8222, 8234-35). The Lead Industries Association argued that "earnings protection matters are among the most complicated and difficult issues to resolve at the negotiating table" (Ex. 453, p. 38), although the specific transcript references cited to support this proposition are not on point. (The failure of two parties to agree on an issue does not support any logical inference that the issue is necessarily complicated or difficult to resolve.) Union representatives, however, consistently expressed optimism that MRP would not significantly disrupt industrial relations. Tr. 7201-02, 7272; Ex. 452, pp. 31-38.)

Due to the controversy on this matter, OSHA carefully considered the evidence in the record and concludes that MRP will not unduly interfere with established employer-employee relations. Admittedly, there may be situations where collective bargaining agreements have to be altered

to smoothly accommodate MRP. But, OSHA has seen no evidence which would support claims that conflicts will likely not be easily resolved. The weight of the evidence directly contradicts such claims.

First, it is difficult to overstate the extent to which many OSHA and other Federal regulatory actions infringe upon traditional subjects of collective bargaining. Minimum wage guarantees (29 U.S.C. sections 201-219 (1976)), equal employment opportunity regulations (5 U.S.C. sections 5108, 5314-5316 (1976); 42 U.S.C. sections 2000e, 2000e-1 to 2000e-6, 2000e-8 to 2000e-9, 2000e-13 to 2000e-17 (1976)), pension reform legislation (29 U.S.C. sections 441, 1001-1003, 1021-1031, 1051-1061, 1081-1086, 1101-1114, 1131-1144, 1201-1204, 1221-1222, 1231-1232, 1241-1242, 1301-1309, 1321-1323, 1341-1348, 1361-1368, 1381 (1976)), and Fair Labor Standards Act provisions (29 U.S.C. sections 201-219 (1976)), all directly conflict with and restrict collective bargaining freedom to negotiate wages and other terms and conditions of employment.

The collective bargaining problems posed by MRP, to the extent any problems exist, are trivial in comparison to the complex problems which are resolved in the course of effectuating remedial EEO affirmative action plans, (Lussier, "Academic Collective Bargaining: Panacea or Palliative for Women and Minorities," 27 Lab. L.J. 565 (1976); Wood, "Equal Employment Opportunities and Seniority: Rights in Conflict," 26 Lab. L.J. 345 (1975)) and the new Pension Reform Law. (Jett, "Employer Contingent Liabilities Under Union Pension Plans: ERISA Fact or Fiction," 27 Lab. L.J. 361 (1976).)

Actions taken pursuant to the Occupational Safety and Health Act directly conflict with collective bargaining matters. Occupational safety and health issues have been recognized topics of collective bargaining for decades. (*In re: Clinton Foods, Inc. and Local 514, International Chemical Workers Union*, 112 NLRB 239 (1955), *Fibreboard Paper Products Corp.* v. *NLRB*, 379 U.S. 203, 221-222 (1964) (Stewart, J., concurring), *In re: Gulf Power Co. and Local Unions 1055 and 624, International Brotherhood of Electrical Workers*, 156 NLRB 622, 625 n. 1 (1966)). The detailed 1976 U.S. Department of Labor Bureau of Labor Statistics study of safety and health provisions in major collective bargaining agreements highlights thousands of collective bargaining clauses which address issues affected by OSHA standards, such as hygiene facilities, personal protective equipment, plant inspection and compliance responsibilities, medical surveillance provisions, and hazard pay. (Ex. 365.) There are innumerable opportunities for an OSHA standard to legitimately require a specific protective mechanism which would be contrary to an established collective bargaining agreement. To OSHA's knowledge these conflicts have been satisfactorily resolved in the past, and there is no evidence suggesting that the implementation of MRP will proceed any differently.

OSHA is convinced that MRP presents no major conflict with collective bargaining in view of the long-term use of MRP-type programs by industry itself. Industry claims that MRP conflicts with specific collective bargaining clauses typically point to job bidding and assignment practices as the areas of greatest conflict. Industry arguments that collective bargaining cannot resolve these matters lack credibility since many lead firms already have temporary medical removals as part of their medical surveillance programs. OSHA has in large part adopted MRP because of the demonstrated value of temporary medical removal as a protective mechanism. There is no evidence in the lead record that existing industry medical removal programs have proved difficult to structure or administer. (Tr. 8231, 8234-35; Ex. 452, p. 37; cf., industry recommendations that the creation of an MRP program can and should be left to collective bargaining, Tr. 7756; Ex. 354(F), p. 1; Ex. 354(P), pp. 1-2; Ex. 354(DD), pp. 1-2; Ex. 354(EE), p. 3; Ex. 354(GG), pp. 1-2; Ex. 354(HH), p. 8; Ex. 391, p. 1; Ex. 397(A), p. 1; Ex. 465.)

More importantly, worker transfer programs with economic protection have had long-term use throughout industry in a variety of contexts. Mr. Roger Sonnemann, vice president of corporate administration and employee relations for Amax, Inc., (Ex. 391, p. 1) discussed the history of transfer programs having economic protection as follows:

> It is more often called protection of rate, and has been the subject for negotiation in our company, in our industry and other industries dating back before the National Labor Relations Act, and in some cases, before there were unions in the industry. Rate protection has been applied, not only for reasons of health but for other reasons such as job re-evaluation, crew reductions, partial closings, temporary transfers for special assignments, and many other reasons. (Ex. 391, p. 3.)

Organization Resources Counselors, Inc., one of the industry representatives expressing the greatest skepticism about the ability of collective bargaining to accommodate MRP, (Tr. 7534) stated the following concerning transfer programs:

> There are in fact many possible reasons for transfer from a job. These include employee request, equipment breakdown, reduction in force, job combinations, changes of methods or equipment, or other changes inherent in the dynamics of the normal workplace. Industry as a whole has already accumulated many years of experience with job removal necessitated by medically related causes, both occupational and nonoccupational. The large bulk of these situations are already covered in varying degrees by existing laws, collective bargaining agreements, or employer policies and established practices. (Ex. 385, p. 14-15.)

Representatives from both the United Steelworkers of America and the United Automobile Workers testified that rate retention provisions resulting from some form of transfer were to be found in practically every major collective bargaining agreement they administered. (Tr. 7666-69, 7676-77, 8231.) Having considered all of the above, OSHA is certain that the collective bargaining process can effectuate temporary medical removals which occur as a result of the MRP provisions of the standard.

MRP benefits must be provided to removed workers who are temporarily laid off as well as those transferred to an available position. The LIA has properly raised the question of whether a laid off worker is obligated to look for work elsewhere so as to reduce the employer's MRP costs. (Ex. 354(AA), pp. 22-23.) The standard does not expressly address this issue, but OSHA intends that a rule of reason apply in individual cases. No explicit worker obligation to seek alternative work is established for several reasons. First, few removed workers are likely to secure new employment due to the indefinite duration of their removal. Removal could continue for a month, a year, or even stop the next day if the employer creates a transfer alternative and calls the removed worker back to work. Removed workers therefore will be particularly undesirable job applicants due to the impossibility of committing themselves to continued employment with a new employer.

A second reason against requiring all laid off workers to seek alternative employment is the likelihood that suitable jobs for removed workers would also involve substantial lead exposure. OSHA does not intend that laid off workers be compelled to accept alternative employment greatly different from work normally performed or employment located in a different geographical area. Thus, a laid off skilled electrician or heavy equipment mechanic could not be forced to accept indefinite employment as a trench worker or dishwasher. Alternative comparable employment for laid off workers would necessarily often involve lead exposure similar to that which necessitated removal. For example, workers such as lead burners or battery plate pasters would have job skills peculiar to lead-related industries, and thus would likely not find al-

ternative employment unrelated to substantial lead exposure. No prospective lead-related employer would hire a worker already on removal status due to the adverse effects of prior lead exposure, thus few if any suitable jobs will be available for laid off workers having specialized lead-related job skills.

The standard does not mandate that laid off workers on removal status actively seek alternative employment, but other reasonable requirements may be fully consistent with MRP. Employers are in the best position to communicate with neighboring employers and make arrangements whereby employer B would offer temporary comparable employment (without lead exposure) to employees of employer A who are temporarily removed from lead exposure. A wide variety of employer relationships are imaginable which would secure alternative employment for removed workers. It is MRP's intent that laid off workers accept offered comparable alternative employment. Laid off workers not offered alternative employment will in some instances be clearly eligible for unemployment compensation. It would be consistent with MRP for an employer to require a laid off employee in this circumstance to apply for unemployment compensation, and satisfy any applicable requirements for this form of payment.

As explained earlier, it is OSHA's intention that employers effectuate temporary medical removals in a manner consistent with collective bargaining agreements. In some instances an employer might succumb to the temptation to violate a collective bargaining agreement so as to ease compliance with MRP. For example, an employer might fire worker A without required just cause in order to make a transfer position available for removed worker B. If worker B is in fact transferred with MRP benefits, then the lead standard has been complied with. OSHA does not intend to become involved with the enforcement of collective bargaining agreements, thus workers and worker representatives will have to rely on applicable dispute resolution mechanisms, such as grievance and arbitration procedures, to redress situations where employers violate collective bargaining agreements to comply with MRP.

e. *Employer flexibility as to removal and return pending a final medical determination.* OSHA expects that in some instances a conflict will arise between an initial physician and a second physician as to the removal or return of a particular worker. The standard requires an employer to implement and act in accordance with findings, opinions, or recommendations resulting from a final medical determination. The issue arises, however, as to what the employer can or should do pending the outcome of the multiple physician or other medical determination mechanism. Rather than permit uncertainty in this area, the standard provides that an employer may remove an employee from exposure to lead, or place limitations upon an employee, in accordance with the medical findings, opinions, or recommendations of any of the physicians who have examined the employee. The standard gives the employer equal flexibility with respect to the return of the employee or the removal of limitations placed upon an employee, with two exceptions.

The first exception applies to situations where an employee was removed from exposure to lead or otherwise limited due to a final medical determination which differed from the opinion of the examining physician chosen by the employer. In such cases there was a justified controversy as to whether the worker should have been removed or limited. It is reasonable to presume there may be legitimate controversy as to the propriety of returning the worker or removing limitations placed upon the worker. Under these circumstances, delaying the return of a worker or the removal of limitations until after a final medical determination has been reached on these issues is appropriate. The final standard incorporates this requirement.

The second exception applies to situations where an employee has been on removal status for the preceding 18 months due to an elevated blood lead level. As explained later in this attachment, a final medical determination is then obtained which will decide whether to continue removal, permit return, or even decide that return of the employee to former job status can never occur. Only a very small number of long-term lead workers should ever reach the position where this form of final medical determination becomes necessary. The medical determination to be reached in this situation, however, presents unique circumstances and will be of extreme importance to the future health protection of these workers. The standard therefore maintains the status quo—i.e., continued removal—until the full physician determination mechanism has had an opportunity to form a final medical determination concerning one of these workers.

f. *Definition of medical removal protection benefits.* The standard requires an employer to provide MRP benefits to a worker on each occasion that a worker is removed from exposure to lead or otherwise limited. This requirement is defined as meaning that the employer must maintain the earnings, seniority, and other employment rights and benefits of a worker as though the worker had not been removed or otherwise limited. In most cases this will simply mean that an employer must maintain the rate of pay of a worker transferred to a low lead exposure job. The standard, however, uses the all-encompassing phrase "earnings, seniority, and other employment rights and benefits" to assure that a removed worker suffers neither economic loss nor loss of employment opportunities due to the removal. The United Steelworkers of America urged that the standard include a detailed definition of the term "earnings," listing all the possible forms of direct and indirect compensation which an employer might have normally given a worker in the absence of a removal. (Ex. 452, p. 44.) OSHA rejected the adoption of such a detailed definition because it would likely be confusing to some employers in light of the many contexts in which the standard will apply. To comply with the standard, an employer need only maintain the removed worker as though no removal had occurred. In situations where the earnings of a removed worker had been in part based on a piece work rate of pay, the standard necessarily obligates the employer to make a good faith estimation of the worker's likely earnings but for the removal, and maintain those earnings during the period of removal.

The standard's broad economic protection for removed workers results from the reasons for MRP's adoption. MRP is an integrated preventive health program combining temporary medical removals with economic protection for removed workers. MRP is essential to effectuate meaningful worker participation in the standard's medical surveillance program and MRP is also an appropriate means of allocating the control costs of temporary medical removals. From these considerations flow the standard's minimization of economic loss to removed workers. Similarly, MRP benefits are provided to workers limited in a manner short of removal. For example, an examining physician might recommend that an employee should be limited to a 6-hour workday, or a 4-day workweek to prevent material impairment to health. These limitations would not literally constitute a removal of the worker from his or her normal job. but would have economic consequences identical to a transfer (removal) with a major cut in pay. Many medically imposed limitations on a worker will have no economic consequences, but those that do merit the provision of MRP benefits for the same reasons applicable to removals.

The standard explicitly requires that an employer maintain the seniority of a removed worker. The reference to se-

niority is somewhat redundant since the phrase "earnings and other employment rights and benefits" would be adequate to encompass all possible seniority interests. OSHA decided to expressly include a reference to seniority in the standard since there was controversy during the lead proceeding as to whether OSHA should include seniority as a part of MRP. Several industry representatives testified that the area of seniority was extremely complex with seniority interests arising in innumerable contexts. Concern was expressed that OSHA would inevitably become overwhelmed by disputes arising out of any attempt to maintain a removed worker's seniority. (Tr. 7756-57, 7770-73, 7785, 7787; Ex. 354(AA), p. 26; Ex. 354(GG), pp. 3-7; Ex. 385, p. 17; Ex. 453, pp. 41-43.) Claims that MRP and seniority cannot mix are another form of the general argument that MRP will pose irreconcilable conflicts with collective bargaining agreements. As discussed earlier in this attachment, this general argument is not persuasive, particularly in light of the many existing transfer programs which have meshed quite well with seniority interests.

The standard includes seniority as part of MRP benefits due to the importance of seniority to a removed worker. The evidence in the lead record indicates that many if not all economic benefits that workers receive could arise only as a function of a worker's seniority. (Tr. 7770-73; Ex. 416(D), p. 1.) Accordingly, if seniority rights are not preserved during the period a worker is removed, then major economic benefits or opportunities could be lost. Mr. John Tomayko, assistant to the president of the United Steelworkers of America, (Tr. 7649) and for 25 years director of the union's pension and insurance departments (Tr. 7664), also testified as to the importance of seniority to workers:

> * * * (O)ut of seniority rights generally flows the man's right to employment, generally it dovetails with his right to a lot of other benefits, generally his continuous services deeply involved. His seniority rights, his vacation rights, his level of insurance rights, his—many instances his level for pension rights. (Tr. 7671.)
>
> I think that is probably one of the most important things that is safeguarded by the fact that there is a union. First comes the money and second comes the right to the job. I am not sure which comes first. They want two things, they want preservation of their right to a job, and they want preservation of decent money. Maybe they go hand in hand down the road together, because that too determines a man's rate of pay, his seniority rights. (Tr. 7671-72.)

MRP cannot be an effective program in the absence of protecting seniority interests, and the standard's inclusion of seniority reflects this fact.

OSHA is convinced that MRP and seniority interests can be smoothly integrated by affected employers, workers and collective bargaining representatives. Some supplementary language in new collective bargaining contracts may prove desirable, but major revisions should be unnecessary. Hearing participants agreed that at any given moment an employee is working, both the company and the worker know what the worker's seniority rights are. (Tr. 7529-30, 7670-71.) Mr. Tomayko verified that due to their importance seniority rights are clearly spelled out in collective bargaining agreements. (Tr. 7669-71.) Since the standard provides that a removed worker's seniority rights must be maintained as though the worker had not been removed, there should be little difficulty in computing a removed worker's rights. If disputes arise, they will often be resolved without intervention by OSHA. Safety and health matters are enforceable obligations and subject to the dispute resolution mechanisms under many collective bargaining agreements, (Ex. 365, pp. 3, 12-13, 15-17, 49, 52-53), including many of the agreements contained in the lead record. (Ex. 157, pp. 43-44; Ex. 158, p. 66; Ex. 261, p. 28; Ex. 368, pp. 6-7; Ex. 369, p. 88; Ex. 381(C), Art. XII; Ex. 381(D), Art. XXII; Ex. 388, p. 129; Ex. 389, pp. 46-47; Ex. 400(B), pp. 54-55; Ex. 401(B), p. 29; Ex. 404(B)(D)(1), p. 63; Ex. 404(B)(D)(2), p. 15; Ex. 404(B)(D)(4), pp. 68, 70; Ex. 404(B)(D)(5), pp. 49, 68; Ex. 415(C), p. 23; Ex. 423, pp. 33-34; Ex. 424, p. 31; Ex. 425, p. 5; Ex. 426, p. 32; Ex. 430(D)(2), pp. 7, 30-31; Ex. 430(D)(6), pp. 21-22; Ex. 430(D)(7), p. 12; Ex. 430(D)(17), p. 17; Ex. 430(D)(23), p. 48; Ex. 430(D)(25), p. 21; Ex. 430(D)(26), p. 48; Ex. 430(D)(28), Art. XII (p.), Art. XIII.) As a result, employers and removed workers will likely resolve questions involving seniority through established grievance procedures. If OSHA intervention is necessary in particular situations, we are confident that the agency can competently deal with these questions. In the course of processing section 11(c) discrimination complaints under the Act, OSHA already investigates and operates in the context of the full range of industrial relations questions. Also, as part of the Department of Labor, OSHA has immediate access to a broad range of experts in seniority and all other facets of collective bargaining. For all of the preceding reasons, the inclusion of seniority in the definition of MRP benefits should not unduly complicate either the implementation or the enforcement of MRP.

The standard requires that MRP benefits be provided to a removed worker irrespective of what happens to the worker after he or she is removed. By necessary implication, this rejects suggestions by the lead industry that the provision of MRP benefits should be contingent upon an employer's ability to locate an available transfer position. (Ex. 354(AA), pp. 20-21.) Arguments in favor of this available position precondition are founded primarily on feasibility considerations. One conclusion of the discussion of MRP's feasibility is that in most instances, employers should be able to locate available positions to which they can transfer removed workers since the number of temporary removals will be small. As a result, an available position precondition is not necessary in order to render MRP feasible as an economic matter.

OSHA's rejection of an available position precondition is not primarily based on economic considerations, but rather on several other factors including the adverse practical effect such a provision would have. MRP is essential as a means of effectuating medical surveillance, but it cannot possibly serve this purpose if the provision of MRP benefits is uncertain. MRP is directed toward worker reluctance to meaningfully participate in medical surveillance due to fear of economic loss. This fear will be little affected by a mere possibility that MRP benefits might be provided. After the first instance a worker was removed and no available position found, MRP would have no impact upon subsequent workers' willingness to take advantage of medical surveillance.

In addition to the above, the inclusion of an available position precondition would end MRP's role as an economic incentive for employers to fully comply with the new lead standard. As discussed earlier in this attachment, employers who make serious attempts to comply with the standard will experience only small numbers of temporary medical removals—removals which likely can be absorbed by available transfer alternatives. Those who make only half-hearted attempts to comply will discover that the greater the degree of noncompliance, the greater the worker blood lead levels thus the greater the number of temporary medical removals with associated MRP costs. The absence of an available position precondition serves as an economic stimulus for employers to protect worker health. With an available position precondition, MRP would in fact operate as an economic incentive against an employer reducing worker exposure to lead. MRP costs would grow only to the point that an employer exhausted available transfer positions. Furthermore, an employer could eliminate the possibility of ever having to pay any MRP costs by guar-

anteeing that all positions had an air lead exposure at or above the 30 μg action level. Some lead firms would achieve this result simply by waiting for OSHA to compel the implementation of engineering controls.

g. *Duration of medical removal protection benefits.* The standard requires that up to eighteen (18) months of medical removal protection benefits be provided to a worker on each occasion that he or she is removed from exposure to lead or otherwise limited pursuant to the standard. In the vast majority of removals, a much smaller period of MRP benefits will be needed due to a shorter period of removal. The prime determinant in the choice of the 18 month maximum was OSHA's best estimate of the rate at which workers will naturally excrete lead once removed from significant exposure. As noted by industry representatives, establishing such a figure is a difficult endeavor (Ex. 354(E), pp. 2-3; Ex. 354(P), pp. 2—3), particularly in light of the limited research performed on this subject. The evidence in the lead record, however, indicates that except for a few instances 18 months is a reasonable maximum.

The lead record contains considerable evidence concerning the dynamics of blood lead level declines from in excess of 80 μg/100 g to 60 μg/100 g. Evidence from a variety of sources indicates that most workers transferred to low exposure jobs could accomplish such declines in 3 to 5 months. (Ex. 354(U), pp. 2-3; Ex. 354(AA), pp. 28-29; Ex. 354(DD), pp. 3-4; Ex. 354(HH), p. 10; Ex. 397(A), pp. 6-7; Ex. 453, pp. 52-53.) As noted by at least one large battery manufacturer, however, the periods of removal are likely to be considerably longer when workers are removed at blood lead levels in excess of 60 μg/100 g and returned at blood lead levels below 40 μ/100 g. (Ex. 354(U), p. 5.) Removal at 60 μg/100 g with return at 40 μg is one of the standard's most sensitive ultimate temporary medical removal criteria. Estimating a typical period of removal under these circumstances is difficult since there is no history of industry experience to use as a guide. Our understanding of the dynamics between air lead levels and blood lead levels suggests that crucial determinants include the length and severity of a worker's prior lead exposure. (See discussion in attachment A concerning air lead to blood lead relationship.)

Several research efforts in the lead record shed light on the issue of how long it will take for the blood lead levels of removed workers to decline from 60 μg/100 g to 40 μg/100 g. One Canadian study authored by Dr. Clement Richer looked at the blood lead levels of lead factory workers before and after a 4-month strike. (Ex. 371.) No data was reported concerning the typical exposure levels before the strike, but blood lead level results (in μg/100 ml) can be tabulated as follows: (Ex. 371, tables 1 and 2).

	Mean PbB before strike	Mean PbB after 4 mo.	Average PbB decline per month (μg/100 ml)
Years exposed to lead:			
More than 20 (N=17)	68.4	55.8	3.2
Less than 20 (N=21)	67.5	47.1	5.1
PbB before strike:			
Less than 60 (N=10)	48.4	37.4	2.8
Between 60 and 80 (N=19)	70.6	55.6	3.8
Over 80 (N=9)	88.2	60.3	7.0

These results indicate that the rate of decline of blood lead level after removal is slower among long-term lead workers. Also, the lower the blood lead level at the start of removal, the smaller the rate of decline during removal. To make some conservative extrapolations from these data, OSHA assumes that the blood lead level of long-term lead workers upon complete removal from lead exposure would decline on the average at a rate of approximately 3 μg/100 g per month. Some 7 months would thus be needed for blood lead levels to decline from over 60 μg to below 40 μg, with absolutely no intervening occupational lead exposure. Since this is an average figure, some variability of response would be expected with some workers requiring more than 7 months, others requiring less. The spread of this variability is unclear since the Canadian study did not report individual blood lead level data.

In extrapolating from the Richer study, it is important to note that the lead workers there were on strike, thus not occupationally exposed to lead during the 4 months of the study. Most of the workers removed pursuant to the lead standard will be transferred to positions having extremely low air lead levels, but nonetheless having some continued exposure to lead. This continued exposure will lengthen the period of removal since the workers' bodies will continue to absorb some lead which will offset apparent declines. The air lead/blood lead dynamics modeling of MIT's Center for Policy Alternatives suggests that transferring workers to positions just below the action level would roughly double the duration of removal as compared to permitting no occupational exposure during the period of removal. OSHA believes this doubling factor to be a reasonable calculation. Applied to the results of the Richer study, it would suggest that long-term lead workers would require 14 months to decline from 60 μg/100 g to 40 μg/100 g, plus or minus appreciable periods of time due to individual worker variability of excretion of lead.

The center for policy alternatives work modeled the dynamics of worker blood lead level declines from 60 μg/100 g to 40 μg/100 g after removal, and the results of that modeling is consistent with the results of the Richer study. Some of these MIT projections can be tabulated as follows: (Ex. 439A, p. 4-13.)

Number of years of lead time required to decline from 60 μg/100 g to 40μg/100 g exposure prior to removal	Transfer to low exposure position		Layoff with no occupational exposure	
	Months of removal*	Average PbB decline/month	Months of removal*	Average PbB decline/month
0.95	0.7	28.6	0.5	40.0
3.4	3.3	6.1	1.4	14.3
9.0	7.7	2.6	3.5	5.7
16.0	11.0	1.8	5.4	3.7
28.5	14.2	1.4	7.2	2.8

*Assuming 30 days/month.

As with the Richer data, the MIT projections indicate that 14 months of removal in the form of transfer would be typical for long-term lead workers, with even greater periods of time expected for workers having more than 28.5 years of prior lead exposure.

OSHA believes that the Richer study and the MIT center for policy alternatives work provide an adequate basis for the 18 months maximum duration of MRP benefits. Very few workers should require longer than 18 months to decline to acceptable blood lead levels, and 18 months is not in excess of what some long-term lead workers may require. Special procedures have been established for the very few workers who might exceed 18 months of removal without achieving acceptable blood lead level declines.

h. *Employees whose blood lead levels do not adequately decline within 18 months of removal.* The standard establishes special procedures to apply in those rare situations where an employee's blood lead level has not adequately declined during 18 months of removal. A medical examination must be made available to obtain a medical determination as to whether or not the worker may be returned to his or her former job status. The standard also requires that in situations where the worker may not yet be returned to his or her former job status, the employer shall continue to provide medical removal protection benefits until either the worker is returned to former job status, or a final medical determination is made that the employee is incapable of ever safely returning to his or her former job status. In situations where the worker is returned to his or her former job status despite what would otherwise be an unacceptable blood lead level, later questions concerning removing the worker again are to be decided by a final medical determination, with no automatic removal occuring due to an elevated blood lead level.

The above procedures were established due to the likelihood that a very small percentage of long-term lead workers will not be able to adequately excrete the immense quantities of lead accumulated in their bodies. As suggested by Dr. Paul Hammond, some workers may have accumulated such large body burdens of lead that their blood lead level will never again reach safe levels. (Tr. 310-12.) The possibility of this happening is reflected in the results of a 1976 Swedish study authorized by Ahlgren, et al., which looked at the blood lead levels of five retired workers, all of whom had had years of lead exposure in a metal industry. (Ex. 99(D).) The mean blood lead level of each worker during his last 5 years of employment was reported, along with a blood lead level value obtained some 0.5 to 4 years after retirement. The data can be presented as follows (PbB in μg/100 g): (Ex. 99(D), p. 83.)

Subject	Age	Years of prior lead exposure	PbB prior to retirement	PbB after period of retirement	Years on retirement when PbB measured	Average PbB decline/year
ON	66	17	68	43	2	12.5
JB	71	23	65	41	4	6.0
MB	64	22	73	32	4	10.3
AS	67	27	63	51	1	12.0
IL	67	22	68	42	0.5	52.0

The first four retirees excreted lead at a rate much slower than the average rates of decline reflected in either the Richer study or the work done by the Center for Policy Alternatives. If an American lead worker with the characteristics of subject JB were transferred to a low lead exposure job due to a blood lead level of 60 μg/100 g, he or she might well require over 7 years of removal to decline to below 40 μg/100 g. If, however, the same American lead worker excreted lead comparably to subject IL, a transfer of perhaps 9 months would be sufficient to permit return of the worker to his or her former job status.

The preceding data illustrates that some long-term lead workers will excrete lead at an extremely slow rate, while other workers with comparable prior exposures will rapidly excrete lead upon removal. OSHA is convinced that there is no possibility of determining in advance how any particular worker will respond to a removal. At some point, however, it should become clear to what extent the blood lead level of a removed worker is likely to soon decline to acceptable levels. OSHA believes that at this point a medical determination should be made as to the propriety of continuing the worker's removal. With this in mind, the standard provides a medical examination for workers whose blood lead levels have not adequately declined within 18 months of removal.

The standard is not intended to preclude all final medical determinations formed prior to the end of 18 months of removal which decide that a particular worker's condition will never permit a return to a lead-exposed job. Determinations of this nature might arise with respect to permanent, irreversible neurological impairment, and kidney disease. The standard does, however, embody the judgment that such medical determinations cannot be quickly made with respect to blood lead level declines. Little is firmly known about the complicated dynamics of individual worker lead excretion. It would be premature to attempt to quickly assess the nature of a specific long-term worker's future blood lead level declines. The standard requires 18 months of removal before this medical determination is attempted so that the nature of a specific worker's excretion of lead has been documented and thus can be evaluated without concern for such confounding factors as recent substantial lead exposure.

Workers whose blood lead levels will not decline to acceptable levels present unique circumstances. Informed medical judgment can much better respond to these circumstances than an inflexible regulation. In some of these cases, continued removal of a worker will serve no major purpose since the damage done to the worker's body is beyond the point of correction. The worker may yet have no lead-related disease, but his or her fixed body burden of lead continuously presents a risk of material impairment to health no matter how the worker is treated. Return of the worker to his or her former job status may not present further risks to the worker's health so long as the worker's blood lead level remains fairly constant. Furthermore, the worker may be close to retirement age and may wish to spend the remaining months on the job working at his or her former job.

The standard provides that if a final medical determination returns a worker to his or her former job status despite what would otherwise be an unacceptable blood lead level, then any subsequent questions concerning removing the worker again are to be decided solely by a final medical determination.

Automatic temporary medical removal due to an elevated blood lead level is no longer afforded due to the special circumstances presented by such a worker. The flexibility of a final medical determination can afford far better protection to the worker. In this context, physicians participating in the formation of a final medical determination contemplating the return of such a worker are urged to: (1) Recommend appropriate followup medical surveillance subsequent to return, and

(2) recommend explicit protective measures in response to the possibility that the worker's blood lead level begins to climb subsequent to return.

In other situations where a worker's blood lead level has not declined to an acceptable level within 18 months, it may nonetheless be steadily declining toward this point. In this circumstance, several additional months of removal may be all that is needed to achieve an acceptable blood lead level. In rare situations, a worker's blood lead level: (1) May not have declined appreciably during 18 months of removal, and (2) may still be at such a high level as to preclude considering returning the worker to prior exposure. In extremely rare situations it may even be possible to conclude that the worker will never be able to safely return to prior lead exposure.

All of the preceding situations can best be evaluated and resolved by a final medical determination obtained pursuant to the standard. The physician determination mechanism will enable the return of a worker to his or her former job status, or the continuance of the period of removal. If the period of removal is prolonged for some period, either pursuant to a final medical determination, or pending the formation of a medical determination, then MRP benefits must continue to be provided by an employer for the same reasons that they were provided originally. If, however, a final medical determination is made that the worker is incapable of ever safely returning to his or her former job status, then the provision of MRP benefits may cease. At that point, a worker would have to turn to the State workers' compensation system for possible relief, since continued removal would no longer constitute a temporary medical removal under this lead standard. In some circumstances a worker might be eligible for a permanent partial or permanent total disability workers' compensation award, but this is solely a matter for State law. In this regard, no aspect of MRP intends to define, supersede, enlarge upon, or affect in any manner any State workers' compensation law.

i. *Followup medical surveillance during the period of employee removal or limitation.* The standard provides that during the period of time that an employee is removed from exposure to lead or otherwise limited, the employer may condition the provision of MRP benefits upon the employee's participation in reasonable followup medical surveillance. MRP is a comprehensive program combining temporary medical removal with economic protection only for so long as removal is needed to protect worker health. The program's operation depends upon followup medical surveillance after removal to determine when a worker may be safely returned to his or her former job status. Consequently, the standard conditions the provision of MRP benefits after removal upon a worker's willingness to participate in procedures necessary for MRP's smooth operation. The standard does not mandate worker participation in followup medical surveillance, but rather permits the denial of economic protection to those unwilling to help MRP work as intended.

The Lead Industries Association and other industry representatives urged that MRP benefits be denied to any worker who refused any biological monitoring or medical examination offered during the 12 months prior to removal. (Ex. 453, pp. 43-47; see, also, Tr. 7541-43; Ex. 457(A), p. 15.) This condition was urged as essential to enable employers to detect conditions in advance which threatened a worker's health, and to correct these conditions before removal became necessary. (Ex. 354(AA), p. 31; Ex. 453, pp. 44-45.) OSHA decided against the inclusion of such a 12-month disqualification clause for several reasons.

First, such a clause would be counterproductive and punitive in nature. A variety of situations could arise where a worker for understandable reasons refused to participate in a particular blood sampling test or medical examination. A worker may not as yet have received adequate information and training on the hazards of lead, thus fails to appreciate the need for participation. A bona fide dispute may have arisen at a plant concerning the accuracy of a company's blood lead level monitoring, or the objectivity of a company-retained physician. The company might even be under an OSHA citation on any of these matters. Workers might understandably decline to participate until the dispute is resolved. Finally, a worker may have over the years demonstrated an ability to maintain his blood lead level exactly at 40 μg/100 g, and thus participates in blood lead level monitoring every 3 or 4 months rather than every other month as the standard makes possible. In any of the above situations, it would be highly punitive to bar a worker from any participation in MRP in the distant future because of understandable past actions. Such a punitive bar to MRP eligibility would likely have the counterproductive effect of reducing worker willingness to participate in medical surveillance in situations where a worker once declined to participate in a particular test or exam. When participation was most needed to protect a worker's health, the worker would often resist participation since removal without economic protection was assured.

Second, the 12-month disqualification clause suggested by the LIA differs little from a requirement mandating worker participation in medical surveillance. As explained in detail earlier in this attachment, OSHA rejected a mandatory participation provision in the MRP program. Maximum meaningful worker participation can better be assured through cooperation and education than through coercion. We reject the LIA 12-month disqualification clause on this basis even though we expect that many employers will nonetheless continue to mandate worker participation in medical surveillance as has been the practice in the past.

The LIA argues that its suggested precondition is needed for an employer to know a worker's condition so as to be able to take affirmative action to reverse circumstances carrying a worker toward removal. This employer concern is a legitimate one, and the ultimate MRP blood lead level removal criteria were designed with this in mind. Once MRP has been fully phased in, the most sensitive removal criteria will require removal when the average of the last three blood sampling tests (or the average of any tests conducted over the previous 6 months, whichever is longer) indicates a blood lead level at or above 50 μg/100 g of whole blood. Removal will be predicated upon at least three blood sampling tests conducted over a minimum of 6 months. The first two tests will tell an employer what is happening with a worker's blood lead level, and the employer will have several months to reverse any apparent increase before removal could be required by a third test. Consequently, the structure and operation of this ultimate blood lead level removal criteria meets the objective of LIA's suggested precondition without being punitive, coercive, or counterproductive in nature.

j. *Medical removal protection and workers' compensation claims.* The standard contains provisions addressing those situations where a removed worker is eligible for and is awarded workers' compensation payments for earnings lost during the period of removal. Before explaining these provisions, it is appropriate to respond to industry arguments that MRP somehow supersedes, replaces, or enlarges upon workers' compensation law. (Ex. 354(V), pp. 3-8; Ex 354(AA), pp. 4, 6; Ex. 354(EE), p. 3; Ex. 402, p. 6; Ex. 453, pp. 2, 5, 10.)

Arguments that MRP and workers' compensation are essentially one and the same flow from the observation that both programs potentially involve the payment of lost wages to workers. This is the only similarity, for MRP and workers' compensation were formed for different reasons and serve

different ends. Workers' compensation law was established as an alternative to workers pursuing common law remedies for job-related injuries and diseases. In exchange for relatively rapid and equitable payments, workers no longer needed to prove employer negligence as the cause for job-related injuries and diseases. (Ex. 357, pp. 32, 34; Ex. 358, pp. 11, 13, 17.) Workers' compensation law as designed and implemented was and is compensatory in nature. Workers' compensation payments are activated only after a worker has been injured or has contracted a disease. Workers' compensation law is not structured as a preventive health mechanism. And, as recognized by a representative of Organization Resources Counselors, Inc., a major industrial consulting firm, workers' compensation has not proved preventative in practice. (Tr. 7524.)

MRP, in stark contrast to workers' compensation law, is solely a preventive health program. MRP is activated before a worker contracts a permanent lead-related disease. Temporary medical removals enable workers to either reverse effects of lead exposure before any form of disease is acquired, or check the beginnings of lead disease before irreversible conditions arise. Economic benefits to removed workers maximize the likelihood of removal being used where needed, and are an appropriate means of allocating the costs inherent in the use of temporary medical removal as a protective mechanism. Payments to removed workers are not intended to be and do not operate as compensation for injury sustained, but rather are associated with and essential to the overall operation of MRP as a preventive health program. Furthermore, MRP is in no manner intended to define, supersede, enlarge upon, or affect in any manner any State workers' compensation or other law concerning lead-related diseases.

Due to the differences between MRP and workers' compensation law, most lead workers temporarily removed under the standard could have no possible eligibility for workers' compensation payments. Workers' compensation law typically requires a showing of some concrete medical disability or impairment involving symptoms of disease. (Tr. 7119-22, 7167.)

In the vast majority of States, removal due merely to an elevated blood lead level without the presence of symptoms of lead poisoning would yield no workers' compensation benefits. (Tr. 7122-23.) The MRP blood lead level removal triggers have been designed to remove most workers before clinical signs of lead poisoning appear.

In some situations, however, workers will be removed as a preventive health matter who also happen to have specific clinical symptoms of lead poisoning, particularly in removals involving long term lead workers. In these cases there may be some eligibility for temporary partial disability workers' compensation payments for lost wages. (Ex. 376A, pp. 5, 7.) The lead standard contains provisions to deal with these situations. If a removed worker files a claim for workers' compensation payments for a lead-related disability, and an award is made to the worker for earnings lost during the period of removal, then the employer's MRP benefits obligation shall be reduced by that amount. The employer is requred to continue to provide MRP benefits pending disposition of any filed workers' compensation claim, subject to a credit or payback once an award is finally made. An employer receives no credit, however, for any workers' compensation payments made for other than earnings lost during the period of removal.

The foregoing provisions were designed to parallel widespread existing industry practices whereby monetary supplements are made pending and subsequent to the disposition of workers' compensation claims. The National Commission on State Workmens' Compensation Law conducted a detailed study of employer supplements to workers' compensation. ("Employer Supplementation of State-Required Workmen's Compensation," National Commission on State Workmen's Compensation Laws, Supplemental Studies for the National Commission on State Workmen's Compensation Laws (1972) (hereinafter cited as "Supplemental Study").) The lead record contains 19 such provisions appearing in collective bargaining agreements. (Ex. 158, p. 68; Ex. 368, pp. 44-45; Ex. 369, p. 4; Ex. 400(B), p. 56; Ex. 401(B), p. 39; Ex. 404(B)(D)(2), p. 17; Ex. 404(B)(D)(5), p. 47; Ex. 404(B)(D)(6), p. 30; Ex. 404(B)(D)(7), p. 36; Ex. 404(B)(D(8), pp. 18-20; Ex. 404(B)(D)(9), p. 250; Ex. 415(A), pp. 21-22; Ex. 415(B), p. 73; Ex. 426, pp. 47-78; Ex. 430(D)(4)(b), Art. XX, Sec. 99; Ex. 430(D)(14), amendment to Art. XXI, adding new sec. 4; Ex. 430(D)(16), Art. XIII, sec. 1; Ex. 430(D)(17), p. 45; Ex. 430(D)(25), Art. XX; see, also, Tr. 2211, 7662-63, 7835-37, 8220, 8235-37; Ex. 354(DD), p. 2; Ex. 365, pp. 33, 42-45, 62; Ex. 379(A)(5).) Both the Commission's study and the collective bargaining provisions in the lead record indicate that employers: (1) Maintain the wages of workers pending disposition of workers' compensation claims, (2) receive credits or paybacks once awards are made, and (3) supplement workers' compensation awards up to (maintain) 100 percent of a worker's lost earnings. (In particular, see Supplemental Studies, p. 293 (table III-A1).) There is no evidence in the lead record, in the National Commission study, in treatises or articles on workers' compensation law (2A. Larson, The Law of Workmen's Compensation, § 57.41 (1978)) or in the relevant case law. (See generally: *Mercury Aviation Co.* v. *Industrial Accident Commission*, 186 Cal. 375, 199 p. 508 (1921), *Tulsa Rolling Mills Co.* v. *Krejci*, 149 Okla. 103, 299 p. 225 (1935), *Tulley* v. *American Radiator and Standard Sanitary Corp.*, 183 N.Y.S. 2d 688 (1959), *Gonzales* v. *Coastal Wire Warehouse, Inc.*, 328 So. 2d 923 (La. App. 1976)) that any problems have ever arisen with respect to such employer practices. If MRP were structured such that the provision of MRP benefits was made totally without regard to worker compensation eligibility, then in some cases MRP could preclude workers' compensation awards. (Tr. 7139-41.) The standard, however, avoids this result by explicitly providing an employer credit for workers' compensation payments made.

In structuring the standard's provisions relating to workers' compensation law, OSHA rejected conditioning the provision of MRP benefits upon a removed worker filing and processing a claim for workers' compensation. Such a requirement would serve no useful purpose and would burden State workers' compensation administrations with frivolous claims since, as explained earlier, most workers removed under the lead standard will have no eligibility for workers' compensation payments. In situations where removed workers could receive workers' compensation payments, we expect that claims will be filed even though the lead standard does not require claims to be filed. Such workers would be experiencing clinical symptoms of lead poisoning, and would likely be under a physician's care to treat these symptoms. Neither MRP nor the lead standard require an employer to pay for treatment-related expenses of removed workers who have clinical lead poisoning. Workers' compensation laws universally provide for the reimbursement of such expenses (Ex. 358, pp. 145-149) thus workers will want to file and process workers' compensation claims to recover these expenses. Workers who believe that they are eligible for workers' compensation awards should promptly file for these payments irrespective of MRP due to the existence of State statutes of limitations.

k. *Other credits.* As explained above, an employer's obligation to provide MRP benefits is reduced to the extent that workers' compensation payments are made for earnings lost during the period of removal. An employer should not have to provide MRP benefits which duplicate compensation which a

removed worker is actually receiving from other sources for earnings lost during the period of removal. Accordingly, the standard explicitly provides that the employer's obligation to provide MRP benefits to a removed worker shall be reduced to the extent to which the worker receives compensation for earnings lost during the period of removal either from a publicly or employer-funded compensation program or from employment with another employer made possible by virtue of a worker's removal. Thus, an employer would receive no credit for the earnings received by a removed worker from a job already held prior to removal—e.g., evening or weekend part-time work. Examples of publicly or employer-funded compensation programs that might provide payments include unemployment compensation or sickness and accident benefits.

1. *Voluntary removal or restriction of an employee.* The standard provides that where an employer, although not required to do so, removes an employee from exposure to lead or otherwise places limitations on an employee due to the effects of lead exposure on an employee's medical condition, the employer shall provide MRP benefits to the employee. The purpose of this requirement is to preclude employers from evading the MRP program by voluntarily removing workers shortly before the standard would mandate removal. For example, absent some countervailing requirement, an employer could lay off or transfer a worker having a blood lead level of 58 μg/100 g without providing any economic protection. It is likely that some employers would do this in the expectation of avoiding removal with MRP benefits when the worker's blood lead level reached 60 μg/100 g. Even the Lead Industries Association openly predicts that employers will remove workers from exposure before blood lead levels reach the standard's removal trigger. (Ex. 453, p. 59.) If employers can evade MRP with such ease, then MRP will play no role in effectuating meaningful worker participation in medical surveillance. Without the protection of a comprehensive MRP program, the level of worker protection afforded by the standard will be reduced. MRP benefits must be provided so as to close this loophole.

In requiring that employers provide MRP benefits to workers voluntarily removed or limited, OSHA does not intend to unnecessarily reduce an employer's flexibility in removing or limiting workers due to occupational exposure to lead. Voluntary removals may occur, but they must be accompanied by MRP benefits. Few such removals should occur, since there is no evidence in the record that any lead company in this country utilizes removal criteria more protective than those established by this final standard. Some situations might arise, however, where a particular employer genuinely acts in a uniform and nondiscriminatory manner which is more conservative than the standard would require. Regardless of an employer's intent, MRP benefits must be provided, since any avoidance of MRP would seriously undermine the standard's medical surveillance program.

m. *Miscellaneous matters.* The remaining paragraphs of this attachment concern three additional issues raised by industry participants in the lead proceeding. The relationship of MRP to these three matters is discussed as follows.

Personal hygiene and work practice rules. The LIA argued that MRP should contain an explicit provision voiding an employer's obligation to provide MRP benefits in the event the employer is somehow prevented from establishing and enforcing reasonable personal hygiene and work practice rules. (Ex. 354(AA), pp. 32-35.) The Battery Council International (BCI) urged that MRP be denied to workers who violated established work rules. (Ex. 397(A), pp. 3-4.) The MRP provisions do not include either of these suggestions since they are neither necessary nor appropriate.

It is undisputed that employee personal hygiene and work practices are crucial to preventing harmful absorption of lead and the final standard contains numerous provisions specifically addressing these problems. OSHA fully expects that employers will establish reasonable personal hygiene and work practice rules and then enforce them in a fair and nondiscriminatory fashion. OSHA is in full agreement with the following statement by the LIA:

> In order to encourage workers to develop good hygiene habits and work practices, the employer should have the authority to promulgate reasonable rules and regulations concerning hygiene and work practices. Moreover, if an employer is or should be aware that an employee is disobeying such rules and that his poor hygiene or work practices, if unchecked, might eventually endanger the employee's health, the employer should have the authority to warn and then discipline the employee. (Ex. 354(AA), pp. 32-33.)

The United Steelworkers of America concurs:

> Obviously, the way of handling poor, personal hygiene practices is through education, the furnishing of clean, adequate hygiene facilities, and only as a last resort, disciplinary action. (Ex. 452, p. 82.)

The lead record reveals that employers have the ability both to establish and enforce these types of rules. (Tr. 1045-46, 7195-96, 7306-09, 7713, 7767-68; Ex. 365, pp. 16-17.) In view of this power, employers should be fully capable of assuring that employees understand and follow these rules. Permitting employers to deny MRP benefits to employees who have at some time in the past violated a work rule adds nothing to an employer's power, but carries the potential for abuse. The LIA recognized this fact when it stated:

> If, on the other hand, the employer does not take any disciplinary or corrective action at the time the violation of rules is discovered, he should not later be able to disclaim responsibility for paying rate retention after it becomes necessary to remove the worker from overexposure. (Ex. 354(AA), p. 33.)

As a consequence, the final standard does not permit an employer to deny MRP benefits to an employee on the ground that the employee violated a hygiene or work practice rule.

In addition, OSHA sees no need to condition MRP upon an employer's ability to establish and enforce these rules. LIA's sole support for arguing that employers might somehow be incapable of establishing and enforcing these rules is an arbitration decision voiding an employer's unilateral creation of a new smoking rule. (Ex. 354(AA), pp. 34-35.) The rule was voided, however, not because the employer was incapable of establishing reasonable rules, but because the employer had failed to negotiate the issue with the union as previously agreed. (Ex. 405.) OSHA does not view an employer's voluntary agreement to negotiate as to these rules as being in any way a reasonable basis for permitting an employer to deny MRP benefits to removed workers. This is especially true since personal hygiene and work practice rules have the best chances of complete success where they are created by a process of consultation and cooperation between an employer and its employees.

MRP and employee conditions "Not the Fault" of the employer. The LIA and other employers argue that MRP benefits should not be provided where the need to temporarily remove a worker arises from causes other than occupational exposure to lead. (Ex. 354(Z), p. 1; Ex. 354(AA), pp. 15-16, 29-30; Ex. 453, pp. 49-50; Ex. 457(A), pp. 19-20.) Under this approach, MRP would be denied to workers having special susceptibilities to lead, and to workers having sources of nonoccupational exposure to lead, and to workers who contract a temporary non-work-related medical condition which is sustantially aggravated by occupational exposure. The LIA offers no suggestions, however, as to how these situations could be administratively isolated from cases where occupational exposure to lead was the prime basis for the removal of a worker. This issue

was likely avoided since, as NIOSH has reported, the determination of whether a specific medical condition is of occupational origin is an extremely complex matter. (Ex. 376(C).) The issue is posed, though, as whether an employer should be "held responsible" for employee conditions not the fault of the employer. (Ex. 354(AA), p. 30; Ex. 453, p. 49.)

MRP applies irrespective of the combination of factors underlying the need to temporarily remove a worker since MRP is solely a preventive health mechanism. MRP in no fashion "penalizes" an employer, or "holds an employer responsible" for anything; MRP simply provides health protection to a worker who temporarily needs it. OSHA recognizes that there are potential sources of nonoccupational exposure to lead. (Ex. 376(C).) There is little evidence in the lead record, however, to indicate that these sources are of a magnitude comparable to the substantial occupational exposure faced by many lead industry workers. (Tr. 3104-06.) OSHA recognizes that some people may develop permanent medical conditions which absolutely preclude any lead exposure. These individuals will be handled not through MRP but through preemployment medical examinations and through disability pensions. OSHA also recognizes that some workers may at a point during their working careers develop a temporary medical condition which is substantially aggravated by continued exposure to lead. These workers, as well as those who, for example, have a medical condition in part caused by nonoccupational exposure to lead, merit the protection that MRP affords. MRP responds to the likely adverse effects of continued occupational exposure to lead, thus the underlying causes prompting the removal of a worker should not affect the worker's eligibility to participate in the MRP program.

Although the LIA believes that employer fault should somehow affect the MRP program, both the LIA and the BCI agree that an employer has the responsibility to protect a worker from harmful occupational exposure to lead even where the effects of nonwork-related exposures are what make continued occupational exposure to lead so harmful. (Tr. 3221-22, 3226-27; Ex. 137, p. 15; Ex. 335, pp. 84-85; see also, Ex. 335, p. 81.) As stated by the LIA:

> A second objection which has been raised * * * is that it would somehow be unfair to penalize the employer when the worker's elevated blood lead level may have been caused in part by factors (such as off-the-job exposure) over which the employer has little control (Ex. 335, p. 84). * * * We start from the proposition that no employer should allow an employee to work in a lead-exposed area—no matter how safe that area may be for others—if the employer has reason to believe that such exposure would materially endanger the employee's health. Whether an employee has a preemployment condition (such as anemia) or later develops high blood-lead levels through poor hygiene or outside activities, he should not be subjected to exposures which, although not harmful to others, are not safe for him. (Ex. 335, pp. 84-85).
>
> If the exposure in the work room, when added to the baseline exposure from other sources, endangers a worker's health, this is very much a matter for concern and action by OSHA. (Ex. 335, p. 85).

Several participants in the lead proceeding made various suggestions that MRP should include a "safe zone qualification" whereby MRP would not apply if, for example, an employee had not been exposed to lead above the permissible exposure limit for the past 6 months. (Ex. 354(W), p. 1; Ex. 354(AA), pp. 30, 35; Ex. 453, pp. 50-51.) MRP omits this form of qualification since its purported justifications parallel arguments advanced in favor of denying MRP due to hygiene and work practice violations, or due to factors not the fault of the employer. In addition, the 50 μg PEL is not claimed to be a safe level for all workers. Finally, the 30 $\mu g/m^3$ action level is in a sense a safe zone qualification, since the standard does not apply to employers who keep exposure below 30 $\mu g/m^3$.

Worker abuse of MRP. Several employers raised the spectre of workers abusing the MRP program. (Tr. 7462, 7476-77; Ex. 354(D). p. 3; Ex. 354(U) p. 2; Ex. 354(AA) pp. 3, 20, 23; Ex. 402, pp. 8-9; Ex. 453, p. 53; Ex. 447, p. 3.) The MRP provisions, and the explanatory information contained in this attachment, have been carefully prepared in view of all expressed employer fears concerning potential abuse of MRP. Only the most reckless and determined employee should be able to take advantage of MRP. To abuse MRP, an employee would have to deliberately ingest lead (deliberate inhalation should be prevented by employer control of air lead levels, respirator usage, and work practices). Once employers have educated all employees of the many and varied toxic properties of lead, deliberate worker ingestion of lead will likely be contemplated by only the most demented of individuals. Too much ingestion could cause encephalopathy and rapid death. (Ex. 1, p. III—5.) Repeated ingestion of lesser quantities of lead could render a worker impotent, sterile, or quite ill in a variety of ways. Continued ingestion of lead might place a worker on a kidney dialysis machine in later years. The foregoing health risks should not be taken lightly by any worker considering abusing MRP by eating lead. And, with this in mind, OSHA is confident that deliberate worker ingestion of lead should prove no more a problem in the future than it has been in the past. (Tr. 7720, 7476-77, 7825-27.)

ATTACHMENT D—FEASIBILITY

1. *Introduction.* In setting standards for toxic substances, the Secretary is required to give due regard to the question of feasibility. Section 6(b)(5) of the Act mandates that the Secretary shall set the standard which most adequately assures employees' safety and health "to the extent feasible, on the basis of the best available evidence." Additionally, in the development of occupational safety and health standards, "considerations shall be the latest available scientific data in the field, the feasibility of the standards, and experience gained under this and other health and safety laws."

Accordingly, OSHA analyzes the feasibility of proposed standards. In addition, OSHA assesses the economic impact of its standard in order to satisfy a series of Executive orders requiring economic impact analysis of major government regulations. (E.O. 11821, 11949, 12044) OSHA makes such analyses available to affected parties for comment and subsequent hearing prior to issuance of final rules, and invites the submission of other information on the economic impact and feasibility of proposed standards. In developing a standard for exposure to lead, OSHA has evaluated both the economic impact and the feasibility of the standard on the basis of the entire rulemaking record, including the information developed by its own studies of the proposal and submissions by the public. On the basis of the best available evidence, therefore, OSHA has determined, as explained in detail below, that the permanent standard is feasible and will not have an undue impact on the national economy.

To aid in its assessment of technological and economic factors, in early 1976, OSHA contracted with John Short and Associates, Inc. of Salt Lake City, Utah, to identify the industries in which employees were exposed to lead, to investigate the technological feasibility of compliance with the proposed PEL of 100 $\mu g/m^3$, and to estimate compliance costs and impacts for all affected industries. After review of the Short report, OSHA determined that the report had some serious methodological deficiencies and that additional data collection as well as more indepth analysis of the major impacted industries was necessary (See Ex. 65A). D. B. Associates, Inc. ("DBA") was hired to perform this task. DBA subcontracted the economic impact analysis to A. V. Adams Associates, Inc., and the final report from DBA ("DBA report," Ex. 26 with addenda and errata, Ex. 65B and 65C) is

the official economic impact statement (EIS) for the Department of Labor on the lead standard. The Short report was released to the public on January 4, 1977, and has been entered into the record as exhibit 22. Since it was not considered to be reliable in its assessment of costs of compliance, it was therefore designated as a preliminary EIS.

The DBA report uses various sources. It extracted most of the reliable information from the Short report and augmented it with its own research. It used a draft of the Lead Industries Association (LIA) study conducted by Charles River Associates ("CRA") ("Economic Impact of Proposed OSHA Lead Standards," 4 volumes, March, 1977, Ex. 127) for economic data in the smelting and storage battery industries and information gathered from published material and industry sources. Additionally, for the battery industry analysis, it relied to a great extent on a July 1976 study by Industrial Health Engineering Associates, Inc. (IHE) performed for the Battery Council International ("Engineering Cost and Feasibility Study — Proposed OSHA Lead Standard," Ex. 29 (29A)). For the secondary smelting analysis, it relied on a draft of an IHE study performed for LIA ("Final Report: Engineering Cost and Feasibility Study, Proposed OSHA Lead Standard, Secondary Smelters," Ex. 138D) and an unpublished study from 1975 by CRA. For the primary smelting industry, it also relied on an IHE study performed for Amax Lead Company's Missouri smelter (Ex. 3 (108)). In addition to the DBA report, OSHA contracted with the Center for Policy Alternatives (CPA) at the Massachusetts Institute of Technology to have a cost and economic analysis performed to assess the impacts of medical removal protection (MRP) (Ex. 439A, with addendum, Ex. 439B, and errata, Ex. 439C). CPA delivered its report in November, 1977, and additional hearings were held on it. (See attachment C for discussion of feasibility of MRP.) This report, along with the DBA and CRA reports, provide the basis of OSHA's economic analysis of costs and impacts associated with implementation of the lead standard. In addition to these, several cost studies (e.g., Ex. 123C; Ex. 231) submitted to the record were considered along with all relevant comments and testimony.

2. *Guidelines for determining feasibility.* The OSH Act has been characterized by the courts as technology-forcing legislation (*Society of Plastics Industries, Inc.* v. *OSHA,* 509 F.2d 1301, 1309 (2d Cir. 1975) (vinyl chloride standard); *AFL-CIO* v. *Brennan,* 530 F. 2d 109, 121 (3d Cir. 1975) (mechanical power press standard)), and in the case of lead, substantial changes in technology may be required to achieve the necessary degree of worker protection. Section 6(b)(5) of the Act cites "feasibility" as a limitation on what OSHA can require of employers under the standard, but there has been limited judicial guidance on how feasibility determinations should be made by OSHA. This section is provided to set forth OSHA's view of its obligations under the OSH Act and to establish a framework around which feasibility determinations for the lead standard have been made. It attempts to bring together the relevant case law and agency policy which articulate important issues which should be addressed in determining the feasibility of a standard.

Feasibility determinations cannot be discussed in isolation from a unit of reference. In assessing whether this standard is feasible, OSHA has asked: Feasible, with regard to whom? Must it be feasible for every firm to comply? For all stages of production? For different manufacturing techniques? For old and new firms? For small and large plants? For the industry as a whole?

The OSH Act and its legislative history do not address this problem, but guidance can be found in decisions of appellate courts which have reviewed prior OSHA standards. OSHA has recognized that different industries, groups within an industry, and individual firms have different technological problems and differing capabilities to make the technological changes needed to protect workers. (*Industrial Union Department* v. *Hodgson,* 499 F. 2d 467, 479-81 (D.C. Cir. 1974) (Asbestos standard); *American Iron & Steel Institute* v. *OSHA,* 577 F. 2d 825, 833, 836 (3d Cir. 1978) (coke oven emissions standard); *AFL-CIO,* 530 F. 2d at 120). These differences are not necessarily determinative in assessing a standard's feasibility. The court in the asbestos case indicated that the standard does not have to be feasible for every firm in order to be upheld. It did imply that a standard which would lead to the demise of an industry is not feasible. The court did not directly address the issue of whether a standard would be upheld as feasible if it caused the demise of certain subgroups within an industry (e.g., old firms, small plants, certain manufacturing techniques). However, the decision implies that the standard could be considered feasible if the demise of the subgroup did not adversely affect the competitive structure of the industry. Even if the demise of the subgroups does adversely affect the competitive structure, it is not clear from the case that this fact alone would make the standard infeasible.

Thus, it appears that the appropriate unit of reference for determining the feasibility of an OSHA standard is not necessarily individual firms or small groups of firms. On the contrary, OSHA standards may be upheld as feasible even if they lead to the demise of some firms or industrial subgroups so long as the industry as a whole is able to comply with the standard. How much of the industry must be able to comply in order for the standard to be upheld is still an open question.

Because of the strong mandate which the OSH Act imposes for the protection of workers, OSHA standards must often attempt to "force" the development of "new" technology for protective purposes. The exact parameters of this authority are difficult to chart; however, there various sources to draw upon for guidance. At a minimum, it is clear that OSHA is not limited to mandating the adoption of technology which is "fully developed." (*SPI* v. *OSHA,* 509 F. 2d at 1309.) This means that OSHA can go further than simply requiring the diffusion of an already fully commercialized technology to firms that were not using it. Rather, standards may in effect require "improvements in existing technology" or "the development of new technology." (*SPI* v. *OSHA,* 509 F. 2d at 1309.) Thus, requirements for changes of a more-than-incremental nature and changes in fairly early stages of development are apparently feasible within the meaning of the Act.

Furthermore, the courts have realized that in determining the feasibility of change, OSHA can look to the stage of development of new technologies and is permitted to make reasoned judgments, i.e., predictions, about their future utility. (*1*) Thus, OSHA has looked to both "existing capabilities and imminent advances in their art." (*2*) Similarly, a finding that a technical advance is feasible can be made on the basis of evidence that a technology is in the "experimental" stage. (*3*)

Although from these cases OSHA derives substantial leeway as to the amount of new technology it can mandate, there appears to be a constraint upon its authority to determine how the development may take place. Specifically, in the coke oven case, OSHA's requirement that R. & D. be undertaken when, after implementation of the required controls, compliance was not achieved, was invalidated. (*AISI* v. *OSHA,* 577 F. 2d at 838.) The rationale for the court's action appears to be that the requirement was too speculative and open-ended. The case does not, however, stand for the proposition that OSHA cannot de facto require R. & D. because of the stringency of a promulgated standard. On the contrary, if the need for R. & D. arises in order to comply (which, of

course, is not always the case, especially when already-developed technology can be purchased), it may of course be performed. It is not the need for R. & D. to which the 3d Circuit was objecting in the coke oven decision but rather, simply, the unavoidable "affirmative duty" to perform it.

While OSHA's authority to insist, via its standard-setting, upon significant technological change is clear, the cases do not offer much guidance as to the frame of reference for determining "newness." In the intra-industry context it is clear that mandatory diffusion of the latest techniques is appropriate. This interpretation, offered in the no-hands-in-dies case, relies upon the act's legislative history, which expresses the intent of Congress to bring lagging firms up to the standards of the more progressive members. (*4*) Going further, the vinyl chloride and coke oven cases both recognize the necessity and feasibility of requiring technology which surpasses that currently in use in even the leading firms. (*5*)

Because the degree of newness involved in any such change is likely to be different for different industries or different firms within the same industry, the cases permit standards to take such factors into account. For example, the no-hands-in-dies case recognized that "different applications" have different technological capabilities, (*ALF-CIO* v. *Brennan*, 530 F.2d at 120) and the coke oven case considered the problem of old firms versus new firms and retrofit versus new technology.

It is unrealistic to discuss the feasibility of a technological change apart from the time period within which the change is to occur. A change which may be infeasible in the short run, either because the costs are prohibitive or because the technology is not fully developed or both, may become feasible over a longer period of time.

Where the technology is fully developed and ready for commercial application at the time the standard is being developed, immediate compliance may not be feasible because of cost factors. The relationship between time and cost of compliance was recognized implicitly by the court in the *IUD* case when it upheld OSHA's two-fiber standard for asbestos. (*IUD* v. *Hodgson*, 499 F.2d at 479.) The standard authorized a 4-year delay in compliance with the requirement because OSHA was concerned with the economic impact of immediate compliance on the asbestos industries. The court upheld OSHA's decision to promulgate prospective standards when the agency considered immediate compliance to be infeasible.

Where the technology needed to protect workers is not fully developed and ready for commercial application, time is needed in order to permit its development and implementation. The greater the degree of change needed and the earlier its stage of development, the more time will be needed to make the change.

The relationship between these two elements—time and stage of development—was indirectly addressed by OSHA and the courts in regard to the no-hands-in-dies standard. OSHA revoked the standard for a number of reasons, among which was its technological infeasibility. Absent OSHA's revocation, the standard would have required immediate compliance. OSHA determined that the technology needed to comply was "not universally possible in the near future." With such a short timetable for compliance, the technology would clearly need to be in a late stage of development to be feasible. The court implicitly recognized the relationship between time and stage of development in stating that OSHA's determination of infeasibility must consider "existing technological capabilities and imminent advances the art." Neither OSHA nor the court addressed the issue of whether modifying the standard to require compliance at a much later date would be feasible.

Determining a time period for compliance and the feasibility of the industry's compliance within the time frame are particularly difficult problems when the technology needed is in an early stage of development and requires major changes in the industry's production processes. A longer time period will increase the likelihood that the needed technology will be developed. It will also permit a greater variety of technological responses, including those which reduce costs. At the same time, the longer the time horizon for compliance and the less developed the technology needed, the more difficult it will be to predict the specific compliance technologies that may arise and their attendant costs.

While determining the feasibility of short-term compliance with a fully developed technology will rely heavily on considerations of costs, more factors are relevant in the determination of feasibility for long term compliance where the technology is not fully developed. Factors which may appropriately enter into the determination include:

(1) *The general innovativeness of the industry* (more innovative industries may be more capable of developing larger changes in earlier stages of development than less innovative industries, other things being equal);

(2) *The financial and technical resources available to the industry* (industries with both substantial technical expertise and financial resources may be more capable of developing the needed technology and in a shorter time period than industries without these resources);

(3) *The degree of change needed and its stage of development*. (the greater the change needed and the earlier its stages of development, the more time will be needed for compliance);

(4) *Certainty of product market* (increasing demand and the absence of close substitutes would allow significiant price rises or other economic adjustments needed for conversion);

(5) *Size and complexity of plant or process requiring alteration* (large-scale industrial processes may rquire more innovative and expensive modification); and

(6) *The experience of recent technological change in similar industries* (the ability of smilar industries to develop successfully similar new technology is some indication of the standard's feasibility for the industry in question).

In developing the lead standard, OSHA has incorporated an implementation schedule based, in part, on its best judgment of the time periods sufficient for "improvements in existing technology" or the "development of new technology." OSHA has also taken into account the degree of technological change which will be required in order to meet the standard as well as the distribution of its impacts. These considerations, which relate closely to technology, are all relevant to determine feasibility. An additional element in this determination is cost.

The Act does not address the issue of economic considerations, and the legislative history is "at best cloudy." (*AFL-CIO* v. *Brennan*, 530 F. 2d at 122.) Case law, however, indicates that economic considerations can be taken into account in determining feasibility. In the asbestos case, the D.C. Circuit Court of Appeals noted that the "thrust" of the legislative history "seem(s) to be that practical considerations can temper protective requirements." (*IUD* v. *Hodgson*, 499 F. 2d at 477-78.) This reasoning has been followed by the third circuit in the "no-hands-in-dies" and coke oven emissions standards. Therefore, the economic cost of a standard appears to be one among many "practical" factors which OSHA may consider in its determination of feasibility—and one which does not have to be accorded greater importance than technological factors or administrability.

OSHA recognizes the need to assess economic impact, especially for standards where compliance costs can be large. In factoring economic considerations into the decisionmaking process, various approaches to the problem can be used. To date, the courts have not imposed any particular methodology on OSHA. On the contrary, the asbestos case, which offers the most thorough analysis of the OSHA decisionmaking process, has indicated that it is a process "inherently legislative"

in nature and dependent to a greater extent on "policy judgments" than on "purely factual analysis." (*IUD* v. *Hodgson*, 499 F. 2d at 475.)

OSHA has and will continue to make these difficult policy judgments with a view toward fulfilling the objectives of the Act and with the recognition that "there can be no question that the OSHA Act represents a decision to require safeguards for the health of employees even if such measures substantially increase production costs." (*IUD* v. *Hodgson*, 499 F. 2d at 477.)

Although OSHA can impose substantial costs on employers, it is not yet clear exactly what degree of costs can be imposed within the meaning of feasibility. The *IUD* case offers the most guidance. It maintains that a standard is still feasible even if it is "financially burdensome," "affects profit margins adversely," or results in the "demise" of individual employers. (*IUD* v. *Hodgson*, 499 F. 2d 478.) More specifically, the coke oven standard was held to be feasible even though it was projected to increase competition from foreign producers, decrease industry earnings per share by about 13 percent, require approximately 10 million man-hours of work, and was estimated to cost up to $1.28 billion. (*AISI* v. *OSHA*, 577 F. 2d at 836.) In the case of vinyl chloride, the standard was upheld as feasible even when industry projected that the costs of VCM would rise from 7.41¢/lb. to 12.71¢/lb. (*SPI* v. *OSHA*, 509 F. 2d at 1309.)

Although there are several examples of feasible standards, there has been only one instance in which a standard was considered infeasible. This case, the no-hands-in-dies standard, does not offer an especially good example for determining the outer bound of feasibility because of the peculiar circumstances surrounding it. (*6*) Therefore, guidance as to the limits of economic feasibility derives mostly from dicta in this and other cases. In the coke oven case, for example, the court suggested that a standard which "imperils the existence" of the industry might be infeasible. Similarly, the third circuit spoke of "massive economic dislocation" as a measure for an "unreasonable" standard. (*AFL-CIO* v. *Brennan*, 530 F. 2d at 123.)

Clearly, in a determination as complex as that concerning economic feasibility, there are many different kinds of factors which OSHA takes into account. The cases have not been silent on this issue; however, it is important to realize that what they have said has been offered always as guidance and not as a directive. Consistently since the asbestos case, the courts have recognized the wide discretion which Congress gave to OSHA to make policy judgments, and they have generally been deferential toward both the agency's factual determinations and its decision process. (*7*) Nevertheless, there are several elements which the courts have suggested that OSHA may consider:

(1) The issue of intra-industry competition. In the asbestos case, the court recognized that if only a few employers could comply quickly, a delay in the standard might be appropriate in order to avoid increased concentration in that industry. Moreover, a significant adverse effect on a subgroup within the industry may be considered in determining feasibility. In Congress' opinion, however, the intra-industry competitive problems were seen to be severe without regulation, because recalcitrant employers were able to profit from their lack of concern for health and safety. (S. Rep. at 5180, supra, n. 3) Therefore, regulation may in some cases actually improve the competitive health of the industry.

(2) The issue of inter-industry competition. The asbestos case recognized that a standard which renders an industry less able to compete with substitute products is less feasible (other things being equal) than one which does not. Moreover, in discussing the issue of industry-specific standards, the court recognized that differing standards which give one industry a competitive advantage over the other might be grounds for a challenge to the standard, if the industries were in direct competition. Similar standards or different standards for noncompeting sectors would not raise this possibility.

(3) The issue of foreign competition. The asbestos case recognized that foreign competition may be a consideration in determining the feasibility of a standard. Nevertheless, in spite of evidence from industry representatives that U.S. goods would be unable to compete with imports, the standard was upheld. A similar claim was made in the coke oven case by the steel industry. Although the court noted this claim, the standard was again found to be feasible.

It should be emphasized that even though standards may result in adverse impacts in any of the above regards, they do not necessarily become infeasible. On the contrary, the case holdings indicate otherwise, as does the court's qualification of its discussion of these factors in the asbestos case:

> These tentative examples are not meant to illustrate concrete instances of economic infeasibility but rather to suggest the complex elements that may be relevant to such a determination. (*IUD* v. *Hodgson*, 499 F. 2d at 478.)

Another economic issue which has been discussed in the case law concerns the feasibility of promulgated standards as applied to individual firms. Although the issue is one of legitimate concern, it is clearly not dispositive in assessing the feasibility of standards. On the contrary, the appropriate unit of reference in assessing feasibility in standard-setting is not the individual firm. Therefore, while the cases recognize the general intent of the act "not to put employers out of business," equal recognition is given to the reality that standards can put individual firms out of business. The *AFL-CIO* case suggests that there are businesses "so marginally efficient or productive as to be unable to follow standards otherwise universally feasible." (530 F. 2d at 123.) It even suggests that they are "industrial activities involving hazards so great and of such little social utility that the Secretary would be justified in concluding that their total prohibition is proper if there is no technologically feasible method of eliminating the operational hazard." (530 F. 2d at 121.)

This language clearly suggests that groups of firms or product lines might legitimately be forced out of business in certain circumstances. Moreover, both the *AFL-CIO* and *AISI* cases recognize that there are mechanisms to mitigate hardship for individual firms in circumstances involving a strict, but feasible standard. (530 F. 2d at 124; 577 F. 2d at 835.) These include variances, (*8*) abatement modifications, and the ability to challenge the application of a standard in citation contests.

A final issue to consider on the subject of economic feasibility derives primarily from a series of enforcement decisions involving the noise standard (e.g., *Secretary of Labor* v. *Continental Can; Turner Co.* v. *Secretary of Labor*, 561 F. 2d 821 (7th Cir. 1977).) These cases have held that a determination of whether engineering controls are a feasible compliance option involves a balancing of costs and benefits. Although these cases look to the standard-setting cases for guidance as to the meaning of the word "feasible" when it is used in a standard, the gloss they have put on the definition of feasibility has never been approved in the standard-setting context. On the contrary, *Arkansas-Best Freight Co.* v. *OSHRC*, 529 F. 2d 649, 654 (8th Cir. 1976) specifically rejected the notion of a cost-benefit approach. Therefore, *Turner* and *Continental Can* run counter to established precedent in the standard-setting cases and their rationale is not considered applicable.

3. *General principles.* OSHA has determined that compliance with the standard generally may be achieved by the application of existing methods of exposure control, although in some cases this will require imaginative and rigorous application of these methods. In a few instances, technological development may be necessary. In this section OSHA has presented the basic principles on which its conclusions regarding feasibility for each industry are based.

Dr. Melvin First, an experienced industrial hygiene engineer and professor of environmental health engineering at the Harvard School of Public Health, explained the basic principles of controlling lead exposure. (Ex. 270.)

He stated that workers could be separated from contact with lead dust or fume by erecting physical barriers between the worker and the contaminant or by the use of exhaust ventilation that creates air currents to sweep airborne dust and fumes away from the breathing zone of workers and draws them out of the workroom. His testimony documents the specific application of these two principles to the lead industries (Ex. 270, pp. 29-32):

"Every operation that can be mechanized and automated is capable of being enclosed by tight physical barriers and placed under slight negative pressure to prevent outleakage of dust or fume laden air to the workroom. Material conveying by mechanical or pneumatic means is easily adaptable to ventilated enclosure as a substitute for hand shoveling, movement by front-end loader, or by open conveyors of many types. Mechanical movement of solids by screw, belt, apron, drag flight, vibrating, or bucket conveyors is a well established technology. Methods of enclosure and exhaust ventilation are well documented and easy to apply. The principal areas of concern are the transfer points and these must be carefully exhaust ventilated to counter a tendency for these areas to become pressurized by the rapid flow of material and to leak outward through cracks or access ports in the housings. When enclosures are tightly constructed, exhaust air requirements will be modest and entrainment of lead dust by the exhaust air, negligible. Pneumatic conveying of granular materials and powders is accomplished through leaktight systems. The critical control point is where the material comes to rest, as in a storage silo. At that point, the conveying air must be discharged through a suitable air cleaning device. Industrial cloth filters are customarily used for this purpose. For liquids and molten metal, pumping through pipelines is an excellent and safe enclosed material transfer means.

In all these cases, the worker is physically separated from the lead-containing materials. Other means of isolating workers from lead-containing materials by physical barriers are to place them inside air-conditioned work booths from which they can perform their tasks by remotely controlled mechanisms. Bucket loading of conveyors from storage pits, overhead cranes, and bulk loading stations lend themselves to this method of placing workers inside a protective atmosphere enclosure. This system is widely used in the iron and steel industry.

When workers cannot be separated from lead exposure by physical barriers, they can be protected by the use of exhaust ventilation applied through hoods of suitable construction and properly located to provide the required protection factors. The principles of exhaust ventilation for the protection of industrial workers are well established and widely applied. They are based on the creation of a controlled air velocity to draw clean air past a worker, through the contaminated zone, and then to sweep the contamination out of the workroom and into an air cleaning device to capture all the entrained material before release of the ventilation air to the environment. In all cases, it is intended that the worker will be able to work in the clean air zone, upstream of the controlled airflow, and that the design velocity will be maintained at such a level that lead dust or lead fumes will be unable to travel upstream even when propelled by convective air currents generated by hot processes, by moving machinery, or by air jets generated by the manufacturing processes.

In spite of well designed and operated protective systems, some small amount of lead products is bound to escape to the workroom and to settle on all horizontal surfaces. If allowed to accumulate, settled dust becomes air suspended through virbration, traffic, and by other means, and makes a major contribution to the lead-in-air concentration. Therefore, continuous and scrupulous cleanliness is a rigid requirement for all lead industries. In my opinion, lead industries must make adequate provisions for thorough plant cleaning, on a weekly basis, to include overhead machinery, bins, ducts, rafters, and cranes as well as floors, walls, and machines. Stairs, ladders, platforms, and catwalks should be permanently installed to make all such overhead structures easily and totally accessible for vacuum cleaning. Horizontal pipe runs, in which dust settling may occur, should be equipped with end caps that can be removed easily for pipe cleaning during the weekly maintenance period and, securely replaced at the conclusion.

Other lead controls of importance include: (1) Use of enclosed hoppers for material storage in place of open storage piles, to eliminate ground contamination and wind erosion.

(2) Use of enclosed conveying from storage silos to automatic weigh hoppers for batching as a substitute for the use of front-end loaders for this purpose.

(3) Paving all surfaces subject to contamination from vehicles as an aid in prompt and thorough cleaning by washing and/or vacuum cleaning.

(4) Designing all shop ventilation systems to place all workers in a clean airflow that is upstream of all lead producing operations.

(5) Pressurizing with fresh, clean air all nonproduction areas (offices, labs, change rooms, eating places) so that lead-contaminated air cannot enter these spaces from the production shops.

(6) Using central station vacuum cleaning systems with multiple service ports and discharging the contaminated air to the main exhaust stack through efficient filter systems to prevent atmospheric contamination and reentry of dust laden exhaust to the work areas.

Dr. First's testimony, echoed by many engineers and industrial hygienists during the rulemaking (e.g. Schneider, Tr. 2057-2100; Stewart, Tr. 2577-2619), leads to the conclusion that rigorous and innovative application of basic engineering and industrial hygiene techniques will, in almost all cases, enable employers to comply with the standard. "When one correctly applies principles of engineering control, an operation or a machine is totally controlled. That is to say, when an operation or a machine is properly enclosed, it no longer discharges lead dust to the workroom atmosphere; when an operation or a machine is properly exhaust ventilated it no longer is capable of discharging lead dust or fumes into the workroom; when a process has been automated, no worker is in the vicinity to be exposed to lead emissions. Therefore, as a practical matter, machines and processes are 'controlled' or they are 'not controlled'; there are no way-stations on the road to process control. You either do it or you don't." (Ex. 270, pp. 23-24.) Schneider added:

> My contention is that with proper engineering control coupled with good maintenance and good work practices, proper design of process to minimize emissions, and education of workers and good hygiene that we can today achieve levels in the atmosphere of less than 50 micrograms per cubic meter of air. (Tr. 2065-66.)

Dr. First further testified from an engineering point of view "the time required for a conscientious employer to comply can vary from 9-12 months for the design, construction, and installation of relatively simple and conventional systems, such as exhaust ventilation hoods and associated dust systems, enclosed automatic conveyors, and central vacuum cleaning systems, to approximately 4 to 5 years for the construction of entirely new modern plant that incorporates innovative, mechanized, and automated production and material handling systems and processes." (Tr. 2309.) DBA's estimates of time frames were similar. David J. Burton of DBA stated that as a general matter the implementation of simple controls could take as little as "several months" while a very complex system could take as much as 40

months. (Tr. 1025.) Dr. First (Tr. 2310, 2328, 2382) and Knowlton Caplan of IHE (Tr. 3931-33) also noted time limitations on obtaining equipment parts, and adequate engineering assistance. These factors are incorporated into the implementation schedule provided in the standard rather than make many firms apply for a temporary variance.

OSHA believes the implementation schedule is reasonable in this regard and does not universally require 5-10 years as suggested by LIA. (Ex. 335, p. 131.) As a general matter, Dr. First's observation of other industries' experience is relevant. He noted that drastic reductions in exposure to coal dust, vinyl chloride monomer, and asbestos fibers were achieved very rapidly where the effort was made. (Ex. 270, pp. 18-19.) OSHA has no reason to believe the results in the lead industry will be otherwise.

Compliance with the permissible exposure limit for a few industries employing a small proportion of the total workers covered by this standard will require reliance upon technological change. The following is a discussion of how OSHA views such change and the role it plays in the compliance scheme of this standard.

A basic proposition which must be emphasized is that technological change is a very complex phenomenon. There is no sure, simple method for producing it. The process of technological change does not occur as the result of any prescribed sequence of events. Nor does it always arise from research and development, even though R. & D. may be an important stimulant. Rather, innovation is the result of a complex interconnection among various factors, including basic research, new technical ideas (invention), and the recognition of market opportunities, among others. Similarly, it is important to realize that because the process of change varies greatly from industry to industry, it is generally more appropriate to consider specific context in an industry than in the aggregate. A last basic proposition is that there are many different kinds of technological change and various parameters to describe it.

One important parameter considered in this standard is the degree of change. Often major changes are referred to as radical innovations, whereas more modest changes are termed incremental. Another way of categorizing degrees of change involves consideration of whether the change takes place along either a newly or a previously important dimension. For example, the development of a new smelting process based on hydrometallurgy (a new functional dimension) is likely to entail a greater degree of change than improvements in the existing pyrometallurgical process. However, radical change is often more costly or difficult to elicit than incremental change. On the other hand, radical changes may offer the opportunity for a wide variety of ancillary improvements, thereby decreasing their long-term cost. They may also be more protective of workers. Thus, the degree of change is one necessary element to be considered in assessing the feasibility of the change sought.

Another parameter concerns the stage of development of the technology that the standard requires. Virtually all new technologies must go through some development process. This need not be a formalized effort, nor need it proceed in any prescribed sequence. As mentioned above, innovation is no longer generally viewed in terms of a sequential model, but rather one in which various demands and resources interact in a complex and interconnected way. Nevertheless, the standard distinguishes between changes in early and late stages of development. For example, a new process like hydrometallurgy, which is still in the development stage, will generally need more time (and more resources) to be implemented than one for which there is already a working prototype or pilot plant. Conversely, a technology like the Bergsoe process of secondary smelting which has already been implemented, is likely to require only a modest period of further development and a small amount of resources.

In some cases, new technologies may simply be adopted with no need for a development effort. Thus, it is common to distinguish innovation (the first new use) from diffusion (its subsequent adoption). The former case would represent the generation of new technology and the latter, transfer. Although diffusion is usually equated with simple adoption, as the new technology is diffused further and further from its source, it will require significant adaptation in order to be applied successfully in the new context. The Hawley Trav-L Vent, a mobile exhaust device developed for foundries, is currently being adapted for use in primary smelters with some success. Thus, the stage of development of technology for compliance purposes is a determinant of the time within which compliance can be achieved, the effort needed to promote widespread adoption, and the resources involved in making the change.

Another parameter considered by OSHA relates to different types of change. One distinction can be made between "add-on" technologies versus product or process redesign. The former refers to responses (often incremental in nature rather than radical) which do not alter the basic design characteristics of the technology. Such changes may be quick and effective, but they are not always the best long-term solutions. Knowlton Caplan of IHE testified that "the cost of hygiene provisions (engineering controls) in a new plant, designed with the hygiene standard in mind is typically one-third to one-half that of attempting to achieve comparable results in an existing plant not so designed." (Ex. 29(29A), p. 2.)

Another distinction can be drawn between product and process change. OSHA is primarily concerned with improving the health consequences of industrial processes. Sometimes, however, the industrial response to OSHA regulation may be to develop a substitute product. This method of control may be the most efficient solution for some firms in the pigment manufacturing industry.

A last element considered by OSHA is the historical pattern of change within an industry. As mentioned above, some technologies may be undergoing a phase of product improvement whereas others may stress process change and cost minimization. These patterns are a function of the inherent characteristics of the technology and the extent to which its development responds to market needs. As such, the future pattern of change is to some extent predictable. In many instances, the industry response to OSHA standards will bear a strong resemblance to the pattern of technological development absent such standards. Therefore, OSHA has examined the technological characteristics of the industries affected by this standard so as to assess both the technological constraints and potentialities of the industry in question.

In sum, OSHA has attempted to be sensitive to the complexities and various aspects of the process of technological change in its attempt to incorporate new technology into its compliance scheme for this standard. This has facilitated prediction of the kinds of technology likely to arise in response to the standard and the time period within which they can be expected, thus allowing OSHA to know, in general terms, what is feasible. It has also suggested different options as alternatives in designing the standard so as to achieve compliance strategies optimal in terms of protective capability and compliance cost.

In establishing the requirements of this standard and evaluating whether compliance is feasible, OSHA has identified affected industries and investigated the available technology in those industries. It has attempted to estimate the length of time necessary to implement the technology required, taking into account firms' need to plan, construct, test, and refine their

efforts, as well as the economic factors involved. The result is that OSHA has incorporated into its compliance scheme an implementation schedule based on OSHA's judgment of the time each industry, as a whole, will need to effect the technological changes necessary for compliance. Interim milestones are required for those industries where ultimate compliance will take several years and where significant protection can be accomplished in a shorter period. The time limits also take economic factors into account in that they are expected to enable firms in the industry to implement these changes without serious economic repercussions to the industry as a whole.

In the five industries where significant technological change will be required to comply with the PEL (primary and secondary smelting, pigment manufacture, nonferrous foundries and battery manufacture), cost estimates cannot be ascertained with accuracy because of the numerous compliance options possible within the extended compliance schedules. The economic considerations factored into the time limits for these industries to achieve the PEL involve an assessment of reasonable "planning horizons"; i.e., the time for firms to develop longrun solutions (from add-on technology to total recapitalization) that offer the industry maximum flexibility or for new firms to enter the industry.

The implementation schedule represents a merging of both economic and technological factors used to evaluate feasibility. Firms can choose from an array of technical solutions over a time frame sufficient for longrun economic optimization. Since all firms in each industry face the identical PEL and time constraints, the process of the internalization of the cost of compliance acts on the decisionmaking process of the firm and the industry in the same manner as any other market signal. Depending on how firms judge a number of longrun factors including product demand, amount of investment sunk in the existing physical plant and managerial expertise, and alternative rates of return available on the necessary capital, some firms may choose to exit the market and invest in alternative ventures. Of course, other firms with different longrun expectations may choose to enter the market.

The implementation schedule is incorporated into the "methods of compliance" paragraph of the standard, and the basis for the time limit for each industry is explained in industry-by-industry analysis below.

4. *Industry analyses and technological conclusions.* On the basis of all the evidence accumulated during the rule-making proceeding, OSHA has determined that:

(1) Compliance with the engineering control implementation schedule in paragraph (e)(1) of the standard, with minimal reliance on supplemental protective equipment, is feasible; and

(2) By the dates specified in the standard, compliance with the PEL, by the use of engineering controls, work practices, and respiratory protection is feasible.

These conclusions are based on the best available evidence of what each affected industry, taken as a whole, can achieve with presently available production and control technology and imminent advances in the art. These conclusions are necessarily industrywide generalizations, and since some involve projected compliance activities, rely in part on policy judgments. OSHA recognizes that compliance problems may exist at individual plants or work areas, but concludes that these problems can be better dealt with through enforcement activities where solutions can be worked out by affected parties.

The following is a detailed discussion of the technological factors in the major industries affected by the standard.

a. *Primary Smelting And Refining.* (1) *Introduction.*—The primary lead industry ranks fifth (after iron, aluminum, copper, and zinc) in tonnage of metals produced in this country. Four companies—ASARCO, St. Joe Minerals, Amax and Bunker Hill—own the seven facilities that smelt and refine primary lead. Western smelters date from the early part of this century; smelters for the Missouri lead belt were built during the 1960's. An estimated 3,055 employees in the primary smelting sector are exposed to lead. (Ex. 26, p. 5-3).

Primary smelting involves three basic steps—sintering, smelting, and refining. In sintering, a concentrate of galena ore (PbS) is mixed with fluxes and roasted to drive off sulfur dioxide. This operation produces "sinter," a mixture of lead, lead oxide, and slag, which is smelted by a blast furnace at temperatures above 2000° F. The blast furnace reduces the constituents of the charge (coke, fluxes, and recycled slag sinter) into molten lead and slag. Fifteen ton ladles on overhead bridge cranes transport the molten lead to open drossing kettles about 14 feet in diameter. These kettles rest in firebrick settings that keep the lead at the temperatures needed (700° to 1200° F.) for drossing. During drossing, the molten lead from the blast furnace is stirred, and the impurities (dross) are skimmed. The impurities in lead ores vary. Colorado ore, unlike Missouri ore, has a high copper content. The lead is further refined through a softening process that removes antimony and other metals.

Because pyrometallurgy (the extraction of metal from ores by heat) requires extreme heat at variable temperatures, control of emissions in primary smelting has been difficult. For example, material that splashes or drips during transfer of molten lead collects and freezes at the rim and pouring lip of the ladle. These thick, lumpy accretions can interfere with a tight fit between hood and vessels. Ore with significant amounts of copper produces copper matte, which corrodes iron, steel, and most steel alloys.

Thus, the corrosive property of the molten metal has prompted the use of open vessels and crude mechanical methods. The nature and scale of primary smelting have made the application of standard engineering techniques difficult. While the problems are difficult, the hearing record indicates that, with new techniques and methods, they are surmountable.

(2) *Summary.* After reviewing the record, OSHA has concluded that in all operations except perhaps maintenance work and where process upsets occur, the 100 $\mu g/m^3$ level is feasible within the 3 year time period in the implementation schedule through retrofitting and some modification of existing processes. This conclusion is not in agreement with the conclusions of DBA and lead industry representatives. (Ex. 335, pp. 122-123) After reviewing all the exhibits and testimony, OSHA is convinced that the reason for this disagreement is not so much a matter of differing professional judgment in what could be achieved, but in the interpretation of the term "feasibility." Industry representatives' and DBA's claims of infeasibility of the 100 $\mu g/m^3$ level (and even the present 200 $\mu g/m^3$ standard) are, in part, based on the view that for an exposure level to be feasible it must be attainable immediately at all work stations at all times. (Tr. 3971-72; 796, 797) This interpretation was rejected in *SPI* v. *OSHA* where the Second Circuit affirmed an exposure level for vinyl chloride which OSHA claimed would be attainable in several years for most job classifications most of the time. The Third Circuit also rejected a similar claim of the steel industry that the coke ovens emissions standards was infeasible because the Fairfield steel mill, used by OSHA as an example of the feasibility of the 150 $\mu g/m^3$ PEL, did not meet the level at all job classifications at all times. DBA and industry representatives also limited their considerations to retrofit technology only and did not generally consider technological change unless it had been proved successful and could be implemented immediately. (Tr. 5793;

Tr. 796-97; Tr. 872-73; Ex. 26, pp. 4-5, 4-8; Ex. 29 (29A)) Long-run technological solutions were not considered, even those which may be more cost-effective. This creates an a priori limitation on the gamut of possible approaches to compliance. For ecample, the IHE report for the Amax smelter did not consider changes in process as a means for controlling emissions. It observed that materials in the sinter plant are difficult to handle, but did not explore the possibility of flash agglomeration or pelletizing of the dust. It stressed the difficulties of pumping bullion without analyzing the use of ceramics, of rare elements to produce steel alloys resistant to corrosion, of leaching the corrosive components prior to smelting, or of gravitational flow through enclosed chutes to transfer bullion.

As stated above, OSHA's view, supported by judicial opinion, is that "feasibility" in a rulemaking context is not that narrow. In addition, OSHA's experience with other standards leads us to be more optimistic than industry representatives about the success and speed of technological change.

Judgment about the feasibility of a 50 $\mu g/m^3$ standard differed sharply. On the one hand, the steelworkers asserted that "with the possible exception of some maintenance tasks, there exists today the engineering knowledge necessary to control work exposure to inorganic lead compounds at or below 40 $\mu g/m^3$." (Ex. 343, p. 143.) On the other hand, Michael Varner, Corporate Manager for ASARCO's Department of Environmental Sciences, maintained that a "50 $\mu g/m^3$ standard would be an unachievable standard in any smelter or smelter to be built. * * * Looking at the scale of these operations, it is out of the question to achieve 50 $\mu g/m^3$." (Tr. ——).

OSHA has concluded that compliance with the PEL may require up to 10 years for this industry. Primary smelting is not generally regarded as innovative. Dr. First characterizes the history of technological change in this industry as conservative and having "a strong bent to make changes very slowly and in small steps." (Ex. 270, p. 17.) Other limitations on the rate of change are the size and complexity of the hot metal operations in these plants. The difficulty of controlling exposure levels is detailed in the discussion of specific operations below.

Further, the degree of technological change necessary to achieve 50 $\mu g/m^3$ may require development and implementation of innovative technology, possibly including alternatives to pyrometallurgy. OSHA believes that the 10 years provided in the implementation schedule represent maximum flexibility for compliance by an industry which may need to rebuild in part or in whole to achieve a healthful workplace.

Hydrometallurgical production methods are likely to be commercially viable within the 10-year limit, however, less comprehensive forms of process redesign and/or adaptation of developmental projects discussed in the section on specific operations may prove to be sufficient. (Tr. 1463.)

The long-term economic conditions facing the primary smelting industry are detailed in another section. However, it should be noted that the extended compliance time will permit firms in this industry to choose the most cost-effective methods for achieving the PEL and thus lower the cost burden. (Tr. 883.)

Witnesses at the hearing were optimistic about the development of new processes for primary smelting. Knowlton Caplan, president of IHE, while skeptical about the current technological feasibility of a 100 $\mu g/m^3$ standard, expressed faith in the future development of "more effective and less costly engineering systems." (Tr. 5723.)

Frank Block, research director at the Reno Metallurgy Research Center for the Bureau of Mines, described one such potential development, a hydrometallurgical method for recovering lead from galena concentrate. (Ex. 128; Tr. 3386-34-17.) This process does not involve any sintering or smelting and may require no refining. It leaches galena concentrate in a hot solution of ferric chloride to produce lead chloride, which, in turn, is electrolyzed to produce metallic lead. The new process generates no sulfur dioxide. It would be more economical than current techniques and could operate at smaller capacity. It could also be used with Missouri or Western concentrates.

To date, this research has been conducted in the laboratory on a small scale. Block expects the process to virtually eliminate exposure to lead since the operation is closed and wet, although further investigation is needed. This project is in its early stages of development, but industry seems to be very interested in its progress. It has been successfully operated on a laboratory scale, although there are some potential problems that may need to be solved. The Bureau expects to run a larger scale laboratory experiment for a year or 18 months to enable it to build a pilot plant. The pilot plant could be built and operated for 3-4 years, at which time there should be enough experience with the pilot plant to have developed a commercially viable design.

(3) *Specific Operations.* (a) *Concentrate Handling and Storage.*—Concentrates brought from the concentrate plant to the smelter are sampled to determine their composition. They are stored in large bins until mixed and pelletized. Typically, conveyor belts carry the pelletized mixture to the sintering machine. Exposures exceed 200 $\mu g/m^3$ at many plants. (Ex. 26, pp. 5-3, 5-10.) The hearing record suggests that the PEL can be achieved for this operation because it can be completely enclosed. Caplan, for example, found no problem for the Buick smelter to comply fully with the 100 $\mu g/m^3$ standard. (Ex. 3 (108).) Edwin S. Godsey, Chief Fume and Dust Recovery Engineer for ASARCO, referred to a totally enclosed ore handling system being designed for the El Paso plant. (Tr. 6513.)

Effective controls include covers, hoods, and exhaust for all belts and transfer points as well as covers and exhaust for bins. Special handling of flue dust and other fines, perhaps in air conveying systems or by wetting in a pugmill, will also be required. (Ex. 26, pp. 5-9.)

(b) *Sintering.* Because a sintering machine is a traveling grate furnace that transfers, breaks, and sizes materials in order to drive off sulfur in the form of sulfur dioxide, sintering typically produces a considerable amount of dust. Indeed, one plant reported levels as high as 14,000 $\mu g/m^3$. (Ex. 26, pp. 5-10.) While the record contains conflicting evidence, OSHA has concluded that a TWA or 100 $\mu g/m^3$ is technologically feasible in the sintering operation. Caplan stated under questioning that "with enough hard work and money and good luck" sintering could "usually" be controlled to 100 $\mu g/m^3$ or less. He stated that, except in upsets, 100 $\mu g/m^3$ can be attained for the sinter machine operator who spends most of his time in the control room and in the areas where feeding the charge into the sinter machine occurs. "Marginal" compliance can be achieved on the return sinter circuit. (Tr. 5757.)

Appropriate controls include enclosure and ventilation of belts and systems for handling material; enclosure and exhaust on the updraft section of the sintering machine and an exhausted tunnel enclosure on each side of the machine. Machines for breaking and sorting sinter will also require enclosure and exhaust. All operator stations will need HVAC (high volume) filters. (Ex. 26, pp. 5-9.) Dr. Thomas Smith, who conducted the study of secondary smelters for DBA, testified that "a great deal can be done to control emissions from sintering," although, to his knowledge, the processes under development had not yet been installed. He presumed that using the maximum amount of control would produce lead in-air levels below 100 $\mu g/m^3$. (Tr. 800.) Controlling airborne lead concentrations in sintering

to 100 $\mu g/m^3$ may thus require process modification as well as retrofitting in some operations. One possibility is secondary enclosure of the sintering machine. (Tr. 5806.) In other plants a combination of engineering controls, work practices, and occasional respiratory protection along with careful maintenance and housekeeping will prove adequate.

The record does not contain specific evidence about the techniques necessary to achieve a 50 $\mu g/m^3$ standard, but because of the small number of employees who monitor this essentially automated operation, the use of administrative controls and air conditioned control rooms or enclosures might prove adequate to meet the 50 $\mu g/m^3$ PEL on a time-weighted basis. Respirators may be occasionally necessary to supplement other controls. It is important to note that adoption of hydrometallurgical processes such as that described by Block would eliminate the sintering operation and thus the exposures associated with it.

(c) *Blast Furnace.* The blast furnace is the primary reduction unit in the smelter. High employee exposures occur during its operation, especially when the tuyeres, the passages through which air is blown or injected into the furnace, must be cleared of solidified slag or lead. Workers punch out the tuyere hole with iron bars or pnuematic hammers. (Ex. 3 (103).) At present, workers engaged in this task wear respirators.

Exposures at existing facilities range from 140 $\mu g/m^3$ to 24 $\mu g/m^3$, (Ex, 26, pp. 5-10; Tr. 6509; Tr. 6462). The hearing record suggests that achieving an 8-hour TWA of 100 $\mu g/m^3$ will be difficult, but possible, through overhauling. Such overhauling could require as much as 5 years. New plants can be designed and built to meet a 100 $\mu g/m^3$ level.

Burton testified for DBA that control of blast furnaces is a "very difficult process." He did not believe that sufficient engineering controls have been developed to control blast furnaces "at all times." (Tr. 813.) Other witnesses stated that only a 200 $\mu g/m^3$ level could be obtained in existing plants. Caplan judged that even if "all the developmental projects" at Amax's Buick Smelter, such as higher power velocity at the furnace end of the tuyeres, were ultimately successful, results would probably be on the order of 200 $\mu g/m^3$, not 100 $\mu g/m^3$. (Ex. 3 (108), p. 9.) Varner noted that after extensive addition and revision of ventilation control on lead bullion and slag pots at its three smelters, ASARCO could at times achieve a 200 $\mu g/m^3$, but not a 100 $\mu g/m^3$ level of exposure. (Tr. 6452.)

OSHA is confident, however, that conventional techniques not generally in use could further control emissions from blast furnaces. They include: (1) Adequate top side exhaust hoods; (2) adequate local exhaust systems over tapping ports; (3) successful application of the "travel vent"; (4) adequate slag granulator and launder exhaust; (5) redesign of the tuyere punching operation; (6) covers, enclosures and local exhaust ventilation at the conveyor belt transfers, loading chutes, and hoppers at the top of the blast furnace; (7) filtered HVAC provided for operator stations, offices, crane cabs and heavy equipment operator cabs; (8) dilution ventilation; (9) fresh air supply to work stations (air-supplied islands or standby pulpits); and (10) the implementation of a successful housekeeping program coupled with employee training and cooperation. However, in some existing plants, attaining an 8-hour TWA of either 100 $\mu g/m^3$ or 50 $\mu g/m^3$ may require employee rotation and perhaps occasional respiratory protection.

(d) *Drossing plant.* Drossing is a form of refining. Dross, which is a crust of semi-solid caked, lumpy material 6 inches or more thick, is removed from the top of the molten lead in the drossing kettle and transported to the dross furnace by a large scoop handled on an overhead crane without local exhaust ventilation. The dross reverbatory furnace is itself a major source of contamination. Exposure levels range from 150 $\mu g/m^3$ to 2,000 $\mu g/m^3$. (Ex. 26, p. 5-10.) OSHA expects that in these operations, which are extremely difficult to control, compliance with either a 50 or 100 level could require the use of respirators until experimental processes prove practical as anticipated in the implementation schedule.

Despite consideration of several experimental techniques, IHE had "little hope bringing lead in air concentrations to the 100 $\mu g/m^3$ level." (Tr. 5698.) Caplan envisioned the adaption of the Hawley Trav-L-Vent system, a patented technique used on a smaller scale in the brass foundry industry to control emissions during removal of dross. Caplan described the Trav-L-Vent as a "wind box that moves along a straight rectangular duct, picking up and laying down a strip of conveyor belting which forms the top side of the duct, by means of a set of rollers." (Tr. 5698.) He theorized that a double set of vents, one on the bridge of the crane and one along the rack, would allow the two-dimensional motion of an exhaust hood. A flexible duct would permit tilting of the ladle. A second similar system could be installed for skimming the dross and charging the dross furnace. Caplan cautioned that the application of the Trav-L-Vent to an operation this size would be experimental and that its adoption would require an additional bridge crane.

Other experimental techniques were also discussed. Caplan speculated about the possibility of drossing at a higher temperature in order to produce a more granular and powdery dross that could be removed by a Berzelius® machine (a vacuum drossing machine). (Tr. 5712.) Leach electrolysis would, of course, also eliminate exposures. Other controls include conventional hoods for the lead and matte top holes of the dross reverbatory furnace, side draft hoods for the lead runner, and partial enclosure and exhaust of the matte granulator.

(e) *Refinery.* Refining removes antimony and other elements and produces a product of lower hardness and strength. Many of the problems in controlling drossing also figure in refining. In particular, no proven method exists for controlling the movement of materials by ladle. In its report on the Bunker Hill smelter at Kellogg, Idaho, NIOSH observed that, "it may be difficult to control fumes emanating from the kettles because of the nature of the process." (Ex. 300.) Exposures range from 100 $\mu g/m^3$, to 900 $\mu g/m^3$ (Ex. 26, 5-10). One plant has, however, submitted data which indicated airborne lead levels of 50-100 $\mu g/m^3$. (Ex. 26, p. 5-10.) It thus appears that a level of 50 $\mu g/m^3$ could eventually be met in all refineries with conventional technology.

In some plants, dross created on refining bins is skimmed by crane and dumped into a pile to be picked up by payloader for recycling through the sinter plant. Obviously, this type of practice must be eliminated because dust from such an operation contaminates the refinery, casting area, and drossing plant. (Tr. 5695.) Some plants currently dump dross skimmed from refinery pots into ventilated bins that are unloaded underneath a ventilated enclosure. Such a procedure controls the dumping but not the skimming operations. According to IHE, air lead measures at the area have remained above 200 $\mu g/m^3$. (Ex. 196, p. 9.)

AMAX plans to install a Berzelius® machine to skim the caustic dross during refining and pneumatically convey it to a fabric filter. A quench chamber will minimize the possibility of fires in the filter. Collected dust from the filter hoppers would be discharged in a fume slurry system. Although previous experiments with the Berzelius ® machine at the drossing pots have not met with success, Caplan found many indications that it could be made to work in the refining process. (Ex. 3(108); Ex. 196, p. 10; Tr. 5696.) If so, the machine offers an approach that could also be implemented in other plants.

Caplan also suggested that the use of the same controls for refinery operations as for drossing plants. These controls include the Hawley Trav-L-Vent, ventilated enclosures for dross handling, etc. Other controls advocated during the hearing were: (1) Ventilated bins and ventilated enclosures for the dumping and handling of dross; (2) mechanized drossing machines; (3) fresh air supply islands or operator enclosures (stand by pulpits); (4) smooth floors made of concrete or steel; (5) vacuum systems for housekeeping purposes; (6) dilution ventilation; (7) piped-lead systems; (8) separate ventilated enclosures for ladle cooling; and (9) sand-seal systems to achieve a seal for kettle covers. Many of these controls are presently in the experimental stage. Although not yet successfully implemented in a primary smelter, they can be expected to lower employee exposures considerably.

(f) *Crane and heavy equipment.* Past experience shows that sufficient air-conditioning equipment can control the cabs of cranes, payloaders, front-end loaders, dump trucks, paving sweepers, road-cleaning machines, and other equipment to any level of exposure. Caplan, for example, said that although more efficient filters require more power and more frequent changing, "it is perfectly possible to get air that virtually has no contamination in terms of particulate matter." He further stated that it would be difficult but feasible to retrofit the cabs of vehicles that handle lead-bearing materials. (Tr. 5731-32.) Varner, however, testified that ASARCO had found that air-conditioners and filters continually plug up and that in some operations the thermal convective forces carry the contaminated material right past the crane cabs. (Tr. 6707.) Retrofitting cabs is complicated and may require custom-built equipment, but OSHA has determined that control of this operation to either 100 $\mu g/m^3$ or 50 $\mu g/m^3$ is feasible.

(g) *Baghouse, flue dust handling.* Flue dust is a major source of contamination. As Svend Bergsoe explained, the flue dust is, in all smelting companies, "the evil, the root of the evil * * * it is all over the smelter, it is under the writing desk, it is on the floor, in the air, it is everywhere." (Tr. 5161-62.) It is clear that without effective control of this dust, control efforts in other areas of the smelter will be severely hampered. OSHA has concluded that there are feasible methods to reduce exposure levels in this operation below the PEL, but that conventional methods, even a closed automated system will not be totally effective. Handling of flue dust by open equipment, such as front-end loaders, exposes employees in the area to high concentrations of dust. Periodic exposures between 50 $\mu g/m^3$ to 2,000 $\mu g/m^3$ have been reported. (Ex. 26, pp. 5-10.) Typically, control of flue dust is achieved by total enclosure in collector bins, screw conveyors, pub mills, pelletizers, etc. To insure the integrity of the enclosed system, adequate maintenance and housekeeping are needed. For thorough housekeeping, floors under collection devices should be concrete or steel.

Bergsoe's flash furnace, which agglomerates flue dust into chunks of lead oxide, might be applicable in a primary smelter as an alternative to conventional controls. Bergsoe stated that where paper filters are used, primary smelters can agglomerate its flue dust using his flash furnace (Tr. 5173.) This technique has thus far been used to control flue dust only in secondary smelters. (See discussion below.) Thus, it is not known for certain whether the dust in a primary smelter has the properties necessary for the low temperatures in the flash furnace. (Tr. 5162-67.)

(h) *Maintenance operations.* Many maintenance operations do not readily lend themselves to engineering controls. Portable blowers, however, might be used. Burton expected maintenance operations to be considered "on a case-by-case basis depending on the location, the type of exposure, the length of exposure, and so on." (Tr. 815.) OSHA expects that compliance will require respiratory protection.

b. *Secondary smelting and refining.* (1) *Introduction.*—Secondary smelters produce much of the lead used in the United States. The industry, however, is poorly defined. The estimated number of plants, for example, has ranged from 40 to 140. (Ex. 138D, p. 1.) Secondary smelters recycle lead from discarded batteries and other waste materials. This recycling involves two phases: Smelting of the old material to recover crude lead and, in some operations, refining of the crude lead to produce pure lead and alloys for reuse.

Secondary lead smelting plants take scrap lead material from many sources, but the majority (61 percent) comes from scrapped lead-acid batteries. Lead cable covers, linotype, and recovered fume and drosses are other major sources. Some scrap is reprocessed to remove lead from other materials. Battery plates and terminals, for example, are mechanically separated, and lead-copper cables are heated to melt off the lead. Materials containing lead oxide may be processed through a blast furnace to reduce the proportion of oxide to lead metal. Lead from the blast furnance and scrap containing lead metal may be melted in refining kettles and treated by drossing to remove copper and other impurities.

Following the drossing, the lead may be "softened" by removing antimony that has been previously added to give the lead hardness and strength. This removal is done by air oxidation in a reverberatory furnace or by oxidative slagging with sodium dioxide or sodium nitrate fluxes. Once the lead has been refined to a desired composition, it is cast into various shapes or fabricated into wires, pipes, sheets, or solders. (Ex. 26, pp. 5-29.)

Approximately 4,400 workers in the industry are exposed to lead. (Ex. 26, pp. 2-13.) Exposure levels vary among different operations, with the highest occuring in blast furnace areas. DBA analyzed OSHA compliance data and found that prior to August 1976, 83 of 171 air lead samples exceeded 200 $\mu g/m^3$. Data after this date showed 102 of 129 air lead levels above 100 $\mu g/m^3$ and 87 of 129 above 200 $\mu g/m^3$. (Ex. 26, pp. 2-17, 2-18.)

(2) *Summary.* The rulemaking record contains uncontroverted evidence that exposures in secondary smelting operations can be controlled below the 100 $\mu g/m^3$ interim level. Based upon its study of seven representative smelters, Dr. Thomas Smith testified for DBA that compliance by secondary smelters with a standard of 100 was technologically feasible. (Tr. 798.) Because of the proven ability of American industry to engineer away work hazards when required to do so, the Steelworkers also viewed the 100 $\mu g/m^3$ standard as "technologically feasible" (Ex. 343; Tr. 2308, 2313-14.) One company, Keystone Resources, which operates four secondary smelters across the country commented that "our controls are such that we feel we could also meet the action level (50 $\mu g/m^3$) specifications." (Ex. 3(39).) Before the implementation of engineering controls, average air lead at Keystone Resources was 1,036 $\mu g/m^3$. The controls reduced the average to 126 $\mu g/m^3$. (Ex. 452, p. A-137.) The results of a recent OSHA inspection at another secondary smelter indicate that it is presently in compliance with the 100 $\mu g/m^3$ level. (Ex. 26, p. 5-38, Tr. 956.)

Attaining these levels, however, may in a few instances require extensive modifications of current processes. IHE, in a study for the Lead Industries Association, analyzed one plant in detail and concluded that conventional engineering techniques alone could not control battery breaking or scrap and slag handling to 100 $\mu g/m^3$ airborne lead. (Ex. 138D, p. 8) DBA doubted that manual battery breaking, slag and scrap handling, and some maintenance operations could be controlled without process redesign. (Ex. 26, p. 5-29.)

The rulemaking record describes new approaches that may be necessary

to comply with the PEL. Michael Varner, corporate manager for ASARCO's department of environmental sciences, and Melvin First, a professor of environmental health engineering at Harvard, discussed the possibility of innovations in drossing, such as continuous vacuum drossing. (Tr. 2387-80; Tr. 6530-31) Svend Bergsoe, president of Paul Bergsoe and Son of Glostrup, Denmark, described in detail his new technique for smelting scrap lead products. (Tr. 5142-5204.) His process eliminates one of the hardest to control processes, battery breaking, by using a new type of furnace that not only digests the entire battery, but also uses the battery cases to supply 50-80 percent of the fuel required to run the furnace. (Tr. 5194.) In addition a flash furnace agglomerates the flue dust, and the process is entirely enclosed.

At the Bergsoe plant in Glostrup, Denmark, a special machine first punctures batteries to remove the acid. The drained, unbroken batteries are then mixed with coke, iron oxide, limestone scrap, return slag, and agglomerated flue dust to form the charge for a specially designed shaft furnace. Over 95 percent of the lead and antimony in the charge is tapped as crude metal, which is then refined to produce a 99.97 percent pure lead and an antimony concentrate. An afterburner treats exit gases to complete combustion. A flash furnace, which is fed directly from the bag house filters, agglomerates dust into a solid form that is easy and safe to handle. The agglomerated product is recycled to the shaft furnace, thus increasing the efficiency of production while reducing in half the amount of flue dust generated. The Bergsoe furnace is also supplied with a new kind of filter, an ITC filter, which has no moving parts and catches dust on the outside of the bags instead of the inside. (Ex. 173; Ex. 174; Tr. 5142-5204.)

With this new approach, Bergsoe's smelting operations have "run at an ambient air standard well below 100 $\mu g/m^3$." (Ex. 173, p. 10) The new process, however, applies only to smelting. Bergsoe predicts levels below 100 $\mu g/m^3$ in refining operations even in an old plant and notes that a refinery in England has kept exposure levels below 100 $\mu g/m^3$. (Tr. 5183, 5187.)

With the possible exceptions of installing afterburner and agglomeration systems on existing furnaces (Tr. 5177, 5192), the Bergsoe process would require construction of an entirely new smelting plant, estimated to cost $2.5 million for a 20,000 ton per year production, and would take 2 years for construction (Tr. 5192). This cost includes the scrap handling facility (Tr. 5199), furnace, afterburner, baghouse, refinery and even canteen and washing facilities. (Tr. 5190.) General Battery Co. argued that Bergsoe's process was not compatible with production needs of American secondary smelters which generally need to produce 50 percent hard lead and 50 percent soft lead; however, Bergsoe confidently stated that his company could build a plant with 20,000 tons per year hard lead production and 20,000 tons per year soft lead production, each guaranteed to meet a 100 $\mu g/m^3$ standard. Bergsoe has built plants in many countries and is currently negotiating with American companies. (Tr. 5195-96.)

In addition to engineering controls, witnesses at the hearing stressed the importance of a central vacuum system for meeting low exposure levels. DBA stated that a vacuum system is essential. (Ex. 26, p. 5-34.) Caplan also found such a system to be necessary. (Ex. 138D.) First testified that only a vacuum cleaning system would be "practical or consistent" with the "low levels * * * being discussed." (Tr. 2379.) In contrast, Bergsoe found that the best solution was to keep the floor wet all the time. (Tr. 5176.)

First suggested that plant modifications could improve housekeeping. (Tr. 2376.) For effective vacuuming, Caplan recommended floor surfaces that are smooth and durable, such as steel plates in the kettle area. Currently, floors in many areas of secondary smelters are made of dirt or rough, broken concrete. Caplan recommended paving for any storage area not covered with smooth materials. (Tr. 5762.)

Additionally, Mackey testified that front-end loaders could be totally enclosed and pressurized so that the operators are not exposed to any dust or fumes in the building. (Tr. 5155.)

(3) *Specific operations.* (a) *Battery breaking.*—The source for 61 percent of the lead in secondary smelter is scrap automobile batteries. (Ex. 26, p. 5-29.) Battery tops are removed; the plates and residues piled, and the top crushed to extract the posts. The DBA study observed no hoods over saws or guillotines and no ventilation around piles. (Ex. 26, p. 5-31.) Side terminal batteries and large industrial batteries were broken manually without controls. The record indicates that with the exception of manual battery breaking, all breaking operations can be controlled below 100 $\mu g/m^3$ through conventional methods. Moreover, adoption of the Bergsoe process would eliminate altogether the problem of battery breaking.

In order to control battery breaking to 100 $\mu g/m^3$, IHE proposed exhaust ventilation for the battery saw enclosure, the dumping station, and the hydraulic guillotine knife. It also recommended a local exhaust system for manual breaking. (Ex. 138D, p. 2.) First described a design for a completely enclosed, ventilated, and remote-controlled system to separate lead from scrap batteries. (Tr. 2337.) Although First's system never became operational, its design is consistent with low exposure levels.

The alternate approach of the Bergsoe process feeds the whole battery directly into a smelting furnace (Tr. 5158-61), thus entirely eliminating batter breaking and its attendant exposures. The Bergsoe process, however, requires a particular mix of scrap battery materials (Ex. 174; Tr. 5174), preferably a large percentage of polypropylene cases (Tr. 5166). No analysis has been made of the mix of materials found in the U.S., but one secondary smelting firm claimed it was not appropriate for Bergsoe's furnace. (Ex. ——, p. —.) However, Bergsoe stated that, "the whole battery production will switch over to poly batteries in 1 or 2 or 3 years time." (Tr. 5160.) OSHA believes this is correct in view of the negotiations between U.S. smelters and Bergsoe to bring his furnaces into the United States. Absent successful mechanization of this process, administrative controls and occasional respiratory protection appears necessary for compliance with the 50 $\mu g/m^3$ standard for this operation.

(b) *Scrap handling.* The DBA study found that plants piled scrap materials in open areas, some of which were paved and periodically swept. (Ex. 26, p. 5-31.) There is little other control at present. Conventional techniques for handling scrap could significantly reduce exposures. Such methods include isolating the process in a separate building with enclosed and ventilated storage areas, installing a ventilated conveyor system, and paving work areas. (Ex. 26, p. 31; Ex. 138D, p. 3.) Nonetheless, both DBA and IHE concluded that such methods would at best achieve the 100 $\mu g/m^3$ level marginally. (Ex. 26; Ex. 138D.)

As with battery breaking, First's design or Bergsoe's process might reduce or eliminate this problem, but administrative controls may be the most efficient means of achieving the PEL.

(c) *Blast furnace.* Plants currently hood the stay of the blast furnace, charge the furnace by skip hoist, and hood the lead tap. Workers, who generally are required to wear respirators in this area, manually load the skip hoist with lead, coke, and limestone charge materials. DBA observed the highest levels of lead in this area, 500-10,000 $\mu g/m^3$. (Ex. 26, p. 5-31.) Despite such currently high levels, the record indicates that lead exposure in this operation can be kept below 100 $\mu g/m^3$, if adjacent sources of emission are eliminated (Ex. 26, p. 5-32).

For the one plant studied, IHE suggested the application of such conventional techniques as "local exhaust ventilation on slag and lead tapping areas, with makeup air sweeping past the operator; a separate conveyor for charging lead materials; a two-stage charging system; an isolated, ventilated area for relining furnace crucibles; an isolated, ventilated area for slag handling; and provisions for airline or self-contained breathing apparatus." (Ex. 138D, p. 3-4.) These controls would be suitable for other plants as well. Mackey described hoods for the ladles on a "ball-bearing, swivel arrangement" so that the operator can pull it away from the furnace once the slag is tapped. He said there are "no fumes in the building." (Tr. 5149.) Hot metal is tapped into a sump area so that there are no fumes coming into the building during the tapping stage." (Tr. 5150.)

Based on the record, OSHA believe that methods currently available when combined with the use of standby pulpits or fresh air islands (Ex. 3(108)) and administrative controls should be able to reduce exposures to 50 $\mu g/m^3$ in many existing plants. Supplemental use of respiratory controls may be necessary for some tasks, although the Bergsoe process again offers an alternative that would significantly lower the exposures associated with handling charge materials, especially exposures from recycled flue dust. (Ex. 173.)

(d) *Reverberatory furnaces.* Some plants now use reverberatory furnaces to remove antimony from lead bullion. In the plants it studied, DBA found that all charge and tapping points have hoods. Its study suggested upgrading the hood and isolating the operation. (Ex. 26, p. 5-32.)

The rulemaking record contains no other information on reverberatory furnaces. Because such furnaces emit contaminants along their entire external refractory surface, they will be difficult to control. Efficient control will probably require near total enclosure. (Ex. 26, p. 5-33.) Worker exposure at isolated reverberatory furnaces, with administrative controls, probably could be controlled to a TWA of 50 $\mu g/m^3$.

(e) *Slag handling.* Plans typically handle slag with a manual or payload operation in an open area. Slag is tapped into conical molds and, when it solidifies, is broken up to recover any matte. (Ex. 26, p. 5—32; Ex. 138D, p. 4.) IHE's limited testing found unspecified low levels of lead (Ex. 138D), which if representative, could be adequately controlled to 100 $\mu g/m^3$ by ventilation (Ex. 26). IHE, however, believed these data were an insufficient basis for any conclusion (Ex. 138D).

The record contains no specific recommendations. It is reasonable to conclude that ventilation and good materials handling practices would reduce air lead levels to 100 $\mu g/m^3$ or 50 $\mu g/m^3$.

(f) *Refinery operations.* Refining takes place when metals are melted and treated in hemispherical pots to remove impurities in the form of a dross. The difficulty of controlling lead exposures in refining operations varies with the size of the operation. Those requiring overhead cranes are especially difficult to control (Tr. 5695). Portable ventilation that does not prevent access to molten lead during drossing is required. DBA found that plants currently use hoods only during drossing. (Ex. 26, p. 5-32.)

Despite the technical problems in controlling refineries, IHE concluded that conventional technology could meet a 100 $\mu g/m^3$ limit. (Ex. 138D, p. 5.) Such technology includes upgrading ventilation and providing hoods during charging and meltdown (Ex. 26, p. 5-32). Separating the refinery from the blast furnace by a wall is also important. (Ex. 138D.) Bergsoe testified that separation of the refinery from the smelter is essential for good pollution control. (Tr. 5164.) The bullion is cooled in molds and is taken in ingot form to the refinery thus eliminating the dust and fume problem in transporting bullion. (Tr. 5150.) DBA also suggested that ventilation systems will need to be upgraded and hoods provided during charging and melting to meet the interim level. Careful hooding of drossing kettles, combined with strict housekeeping and isolation from other sources of contamination within a smelter, would be essential to meet the PEL.

(g) *Casting and fabrication.* Airborne lead can be generated when lead is cast into ingots or fabricated into plates, sheets, wires, etc. DBA found that little control is currently provided for either operation. (Ex. 26, p. 5-32; Ex. 138D. p. 5.) According to both DBA and IHE hooding these areas and using local exhaust would be feasible. (Ex. 26, p. 5-32; Ex. 138D, p. 5.) In particular, IHE recommended portable hoods suitable for mobile equipment (Ex. 138D, p. 5). OSHA has concluded that attaining a 50 $\mu g/m^3$ level should not be difficult and will require isolation, local ventilation, and careful housekeeping.

(h) *Baghouse and flue dust handling.* Baghouses capture the lead fumes and dust generated by furnace operations. Some plants use automatic systems that feed baghouse dust into the blast furnace. Other plants manually return the dust, a system that involves high exposure and creates severe housekeeping problems. (Ex. 26, p. 5-33.)

DBA and IHE agreed that automated systems can meet a 100 $\mu g/m^3$ standard (Ex. 26, Ex. 138D). Edwin Godsey, chief fume and dust recovery engineer at ASARCO, Inc., described a screw conveying system being designed for the baghouse at his company's El Paso plant (Tr. 6522). Pneumatic conveyors could also be used. (Ex. 138D.) Such modifications in the process should control these operations to the 50 $\mu g/m^3$ level. Furthermore the flash agglomeration of dust in the Bergsoe process would not only facilitate safe handling of dust but also improve utilization of the dust, thus increasing efficiency. (Ex. 174.)

(i) *Maintenance operations.* Regular maintenance is, of course, essential to compliance with any standard. (Tr. 2388, 2340.) However, workers who maintain and repair dust control systems and production equipment are inevitably exposed to high levels of dust. At this time, no engineering controls are known that provide complete protection for maintenance activities, although some can reduce exposures significantly. Witnesses assumed reliance on personal protective equipment would be necessary, and did not discuss the use of portable ventilation, which could provide some measure of protection. In addition, respirators and rotation of workers will be "indispensable for a few maintenance procedures." (Ex. 270, p. 20.) In maintenance operations, OSHA expects the use of respirators to be necessary in most cases for compliance with either a 50 or 100 $\mu g/m^3$ standard.

(j) *Other operations.* Some secondary smelters manufacture lead oxide. Controls for this process are discussed below in the section on the battery industry.

A few smelters use sintering to agglomerate dusts. The sintering machine is a source of high lead exposure. Although existing sintering facilities have some hooding and fume control, this equipment would need upgrading to meet a 100 or 50 $\mu g/m^3$ standard. (Ex. 26, p. 5-33.)

OSHA has concluded that the technology exists today to allow the secondary smelting industry to comply with the PEL. New technological developments will make the task easier and less expensive. Because of the extensive modification needed to bring secondary smelters into compliance with the PEL, the compliance schedule allows 5 years, with 3 years for the interim level of 100 $\mu g/m^3$. The 5-year period is based on the testimony of First, IHE, and DBA where estimates for time to implement engineering controls were presented. In addition, conversion to new smelting processes could take place within 5 years. Bergsoe testified that construction of a

plant using his process would take about 2 years.

c. *Battery manufacturing.* (1) *Introduction.*—The battery industry is the largest single user of lead in the United States. The industry produces both SLI (starting-lighting-ignition) batteries and industrial batteries, although the latter accounts for only 7 percent of the industry's production; 138 firms operate 200 plants, which vary tremendously in size and capacity. On one hand, the 7 largest firms operate nearly 70 plants and account for over 90 percent of the batteries sold. On the other, 95 battery plants employ fewer than 20 people. Of the 16,000 persons employed by the industry, approximately 12,800, or 77 percent are exposed to lead. (Ex. 26, p. 5-42.)

Manufacture of batteries begins with production of lead oxide, either by the Barton process, which oxidizes lead in the molten state, or more often, by the ball mill process, in which frictional heat generated by tumbling lead pigs or balls produces lead oxide. Lead oxide powder is mixed into a paste and pressed onto grids cast from lead. The pasted plates are cured, stacked by hand or machine, and connected with molten lead ("burned") into groups that form the individual cells of a battery.

All these processes, especially loading and unloading at each step, generate contamination. The racks that carry the pasted plates from one operation to another are additional sources of lead dust. Dust forms as well during reclamation of rejected grids, parts, and pasted plates, and during removal of plate groups from defective batteries.

(2) *Summary.* The record indicates that in the battery industry available methods can control employee air levels of lead below 50 $\mu g/m^3$, as an 8-hour TWA, for all major processes. Indeed, more than 40 percent of employees exposed to lead in this industry may already have TWA exposures of less than 50 $\mu g/m^3$. (Ex. 26, p. 5-45.)

The steelworkers commented that "there is no real dispute in the testimony of the technological ability of battery plants to meet the proposed 100 $\mu g/m^3$ standard." (Ex. 343, p. 152-53.) Edward Baier, Deputy Director of NIOSH, pointed to the General Motors battery plant in Muncie, Ind., as an example of the success which can be achieved in controlling lead exposure in an older plant. "The majority of departments tested • • • had average air lead exposures during 1976 below 100 micrograms per cubic meter based upon personal monitors. (Tr. 1317.) The UAW asserted that "any operation in a battery plant can be controlled once provided with adequate enclosures, exhaust ventilation, or process redesign." (Tr. 5274.) In his study of 17 plants, Thomas concluded that "the general use of respirators should not be needed in a well-designed and managed lead storage battery plant. (Ex. 101A.) Similarly, Caplan, testifying on a detailed study of 12 plants IHE did for the Battery Council International ("BCI"), concluded that "technically, if all the things that we recommend were done and well done, it is our opinion that we would be able to control to 100." (Tr. 3856.) The recommended controls, presently lacking in many plants, include:

(1) Handling oxide in bulk by pneumatic conveyors instead of barrels or screw conveying systems.

(2) Mixing paste in a vertical array with leakproof connections as well as local exhaust hooding and control of ventilation flow;

(3) Mechanizing and enclosing wet and dry reclaim facilities;

(4) Hosing down the racks and pallets after each cycle; and

(5) Cleaning the floor with a central vacuum system. (Ex. 29(29A). p. ii.)

It is OSHA's judgment that these systems proposed by IHE, when combined with good work practices and administrative controls will be effective to control exposure below the PEL, primarily because they provide total control of the process and minimize the opportunity for fugitive emissions. As Dr. First stated, "The application of good control methods almost always results in air concentrations far lower than the standard for which they were designed". (Ex. 270, p. 19.)

IHE's specifications are designed primarily for larger operations. They assume that production is continuous and that operators remain at each work operation for a full shift, assumptions that do not hold for small plants. Thus, the engineering controls designed by IHE will be effective but may not be appropriate for small plants. The record suggests that less complex controls may be feasible and effective for small plants. Good housekeeping appears especially important. Both Meier Schneider, an experienced industrial hygiene consultant, and Albert Stewart, an industrial hygienist who formerly conducted lead inspections for OSHA, testified that control costs might be held down by approaching problems on a case by case basis and by emphasizing the use of good housekeeping and techniques for handling materials along with imaginative engineering to minimize the need for ventilation. (Tr. 2057-2077.) Dr. Mirer, the UAW's industrial hygienist, noted that of 30 plants surveyed by the UAW, the one with the lowest lead exposures had only nine workers. (Tr. 1007.)

Testimony from operators of small battery plants also stressed good housekeeping and work practices. For example, Don Hull, president of Dynolite Corp., a plant that employs fewer than 20 people, testified that he gives priority to housekeeping and personal hygiene. (Tr. 1246; see also Tr. 3561.) When OSHA took a series of readings in his plant at the stations for grid casting, stacking, element assembly, battery assembly, and battery filling, only one reading at one location, element stacking, exceeded 100 $\mu g/m^3$, and it was just slightly over, 110 $\mu g/m^3$. (Tr. 1247-48.)

Some operations with high exposures are done only intermittently in small plants. Small battery plants, for example, may paste plates only once or twice a week. (Tr. 3465; Tr. 1259.) To meet the PEL as an 8-hour time weighted average, such plants may not need the same controls as a plant that pastes plates all day every day. In fact, alteration of production schedules or employee rotation may be effective. Employees in small plants do not work exclusively at one station. As Stuart Manix of Lancaster Battery Co. explained, "most people try to do a little bit of everything." (Tr. 3465.) Thus, rotation of employees to positions with higher exposures for less than 8 hours per shift may also reduce 8-hour TWA averages. That is, four employees could each work 2 hours pasting plates.

New approaches may also offer small plants an alternative to IHE's engineering controls. Two firms, APSEE, Inc., and Kermatrol, Inc., testified that they could provide the technology for compliance at sharply reduced costs. APSEE, which stands for air purification through the stimulated emission of electrons, uses negative ionization to control exposures. When suspended dust particles are negatively charged by secondary electrons sent out by the system, they are attracted to the earth and held by the charge. (Ex. 316, Tr. 1177-94.) If this secondary ionization process proves as effective for lead as it has for other dusts, it would be far less expensive than traditional engineering controls, especially for smaller plants. The device has already been used in foundries, glass manufacturing plants, and other places with lead problems. Several battery manufacturers expressed interest in the system during the hearing. (Tr. 1188-1191.)

Kenneth Kerman, president of Kermatrol, testified that his particulate filtration equipment could "guarantee attainment of a TLV of 50 micrograms, and even better, depending on the circumstances." (Tr. 5217.) Its equipment consists of a layer of proprietary material added to a HEPA (high efficiency particulate air) filter.

(Tr. 5205-5240; Ex. 176, 177.) This system permits cleaning and reuse of HEPA filters, which provide absolute filtration dust collection. Kermatrol recommends the combination of this filtration with the negative pressure enclosure of a "gloveless glove box" and return of filtered air. Kerman stated that air returned at a speed of 50 to 70 feet per minute would not annoy an operator. (Tr. 5214.) Although not all operations can be done inside a box and not all materials are appropriate for the filter, these limitations do not appear to present problems for lead dust in battery plants. Indeed, the finer the material the more efficient is the filter. (Ex. 176.) Over 200 Kermatrol units have been installed, including one in a battery plant. (Ex. 177.) The construction of new plants with highly automated, enclosed manufacturing processes should enable the battery industry to comply with the 50 $\mu g/m^3$ lead-in-air standard for every operation.

The witnesses at the hearing underscored the importance of housekeeping and maintenance to supplement engineering controls. As Meier Schneider, an industrial hygiene consultant to the Teamsters, explained, "if engineering controls are not maintained, they break down, and the air concentrations in the workplace rise." (Tr. 2060-61.) The UAW observed that "lack of maintenance ventilation and process equipment, and poor cleanup of the toxic residue in the plant have been cited as major causes of preventable lead exposure." (Tr. 5053.) A 1941 U.S. Public Health Service study of the storage battery industry noted that "any control method will lose its effectiveness if not properly maintained." (Ex. 6 (45); see also Ex. 29 (29A); Tr. 3870; Tr. 2380; Ex. 101A, and Tr. 2325-26.) Rigorous maintenance and scrupulous housekeeping will be crucial for achieving and sustaining the 50 $\mu g/m^3$ standard.

(3) *Specific operations.* (a) *Oxide manufacturing.*—At present, exposure levels in this operation, which involves 2 percent of employees, are generally above 200 $\mu g/m^3$. (Ex. 26.) For plants that manufacture ball mill oxide, available controls can reduce concentrations of lead in air below 50 $\mu g/m^3$. In the 1941 public health service study, conventional ventilation control, isolation from other work areas, and vacuum collection of spilled oxide attained concentrations of air lead ranging between 60 and 100 $\mu g/m^3$. (Ex. 6 (45).) IHE recommended not only isolating the process, but also establishing dust control at the mill trunnion, the classifier, the oversize return, wherever material is handled, any drossing, and at all storage hoppers. He also urged pneumatic conveyance of material and passing the exhausted air through a fabric filter. (Tr. 3699.)

Thomas's study, which included seven plants with fewer than 50 employees, found that "plants which manufactured lead oxide used totally enclosed systems and employee exposures were minimal." (Ex. 101A.) Testimony presented at the hearing by the UAW suggests that new equipment could be designed and manufactured to meet a 50 $\mu g/m^3$ standard. (Ex. 180, p. 16.)

(b) *Oxide receiving and handling.* Battery plants that do not manufacture their own oxide receive the oxide in drums or tank trucks. Handling this oxide usually exposes employees to levels of lead in excess of 100 $\mu g/m^3$ and often in excess of 200 $\mu g/m^3$. (Ex. 26, p. 5-47.) The record suggests that pneumatic conveying systems can maintain airborne lead concentrations as low as 50 $\mu g/m^3$, if combined with rigorous preventive maintenance, isolation from other sources of contamination, and structural modifications that permit careful housekeeping.

Most plants now use barrel or complex screw conveying systems. Controlling barrel dumping of oxide to 50 $\mu g/m^3$ would require enclosing the entire process, a modification which would involve a large volume of air and increased operator time. (Tr. 3700, Ex. 29 (29A), p. 12-13.) The UAW, however, wondered why any but small plants that move oxide infrequently would use a barrel system. (Tr. 5280, Ex. 180, p. 7.)

Screw conveying systems can leak oxide from many sources, such as transfer points from shaft to trough or between conveyors. (Ex. 29 (29A), p. 12-13.) Control by such conventional techniques as improved seals would be both expensive and unreliable. (Ex. 29 (29A), p. 12-13.) Because of the difficulty in controlling leaks from screw conveying systems, the hearing record suggests the adoption of totally enclosed systems. The UAW prefers a "totally enclosed system where oxide is moved by force of air or by an auger." (Tr. 5279, Ex. 180, p. 6.) Caplan also recommended pneumatic conveying as the "best all-round solution." (Ex. 29 (29A).) According to the UAW, larger operations should, in fact, find enclosed systems more efficient than barrel handling. (Tr. 5380.)

(c) *Paste mixing.* Lead exposure in paste mixing usually exceeds 100 $\mu g/m^3$. (Ex. 26, p. 5-42.) The plants surveyed by IHE met the 200 $\mu g/m^3$ standard only marginally, and he called control of this area one of the "most troublesome." (Ex. 29 (29A), p. 13.) Small plants that use drums to dump paste into mixers could be controlled to 100 $\mu g/m^3$, according to IHE, by installing extensive hooding and facilities for cleaning the drums. (Ex. 29 (29A), p. 13.) Testimony by battery manufacturers suggested that enclosed, automatic oxide mixers would also reduce exposures. (Tr. 2888; 3705.)

To achieve a level of 100 $\mu g/m^3$ in larger plants, IHE endorsed a new design, "a vertical array with the oxide weigh hopper at the top, mixer at an intermediate level, and the paste machine at the bottom." This arrangement would also incorporate special dust control and would involve new equipment as well as additional work space in some cases. (Ex. 29 (29A), p. 15.)

The record suggests that, under certain conditions, approaches for meeting a level of 100 $\mu g/m^3$ will also achieve a level of 50 $\mu g/m^3$. In addition to isolation, preventive maintenance, and modifications to permit assiduous housekeeping, conditions needed to attain a level of 50 $\mu g/m^3$ include denying the operator access to the paste to test consistency.

(d) *Pasting.* Employee exposures in pasting operations generally exceed 100 $\mu g/m^3$. (Ex. 26, p. 5-47.) To reduce this level, the hearing record emphasized the need for effective washdown in both small and large plants. (Ex. 29 (29A), p. 16; Ex. 101A.) IHE determined that to maintain levels of 100 $\mu g/m^3$ pasting departments in average-sized plants would require not only local ventilation at the feed, take-off end, and rack of the pasting machine, but also enclosure of the pasting area with net air flow toward the center of the pasting line from both ends. (Ex. 29 (29A), p. 16.)

Attempts to attain a level of 50 $\mu g/m^3$ might also entail isolation of pasting from other sources of contamination and modification of structures to expedite housekeeping. Data collected by Thomas indicated that pasting operations in small battery plants can be controlled to meet the 50 $\mu g/m^3$ standard. (Ex. 101A.) Thomas recommended that the pasteline feed area be built on an open grid floor over flowing water or that the floor be kept permanently wet. (Ex. 101A.)

(e) *Plate curing and handling.* Experiments conducted by IHE demonstrated that washing the racks used to transport pasted plates at the end of each cycle will control this source of air lead to 100 $\mu g/m^3$, if floors and other works surfaces are kept clean. IHE deemed vacuum cleaning of floors to be necessary once per shift. (Ex. 29 (29A), p. 35.)

In addition, measures were advanced to control dust from moving and handling of pasted plates below 100 $\mu g/m^3$. These proposals included separating pallets used to transport grids from those used to transport plates; storing and transporting plates, elements, and

oxide-containing scrap in sealed containers; and attaching exhaust systems to tubs for storing and transporting plates. (Ex. 180, p. 9; Tr. 5284.) In addition to enclosing plate curing operations, modifications in the plant's physical structure may be necessary to permit the requisite housekeeping. Indeed, complete automation and enclosure of plate handling from pasting to assembly may prove the most practical means of reducing levels of lead in air to 50 $\mu g/m^3$.

(f) *Grid and parts casting.* Current exposures in this operation usually fall below 200 $\mu g/m^3$ and often below 100 $\mu g/m^3$. (Tr. 5977.) The hearing record contains evidence that casting operations can currently meet the 50 $\mu g/m^3$ standard. The UAW testified that "a 50 $\mu g/m^3$ exposure limit is immediately accessible in casting operations." (Tr. 5286.) Levels reported by the U.S. Public Health Service 35 years ago and by Thomas more recently further support this conclusion. (Ex. 101A; Ex. 6(45).) IHE also observed air concentrations to be generally below 100 $\mu g/m^3$ in those casting areas that were areas supplied with ventilation and exhaust hoods and isolated from other sources. IHE recommended exhaust hoods for the dross bucket and the dross skimmer as well as a hood through which the dross skimmer can be moved from the melting pot to the dross bucket. (Ex. 29(29A), p. 27.) Thomas noted that many hoods were not properly maintained (Ex. 101A.) Careful regulation of melting and pouring temperatures is also important to prevent excessive fuming from molten lead. (Ex. 29(29A), Ex. 26; Ex. 101A; Tr. 5286.)

(g) *Plate breaking and finishing.* According to the hearing record, control of this process will require exhaust ventilation both for the operation itself and for the racks and pallets used to move the plates. (Ex. 101A; Ex. 6(45); Ex. 180.) Strict housekeeping, including vacuum cleaning of racks, was also urged. (Ex. 180; Ex. 6(45).) These controls are expected to attain a level of 100 $\mu g/m^3$, and should also permit attaining a level of 50 $\mu g/m^3$. (Tr. 5977.)

(h) *Plate stacking.* Employees who stack plates are exposed to levels usually above 100 $\mu g/m^3$. With inadequate controls or work practices, exposures may go much higher. Incentive pay practices that encourage speedy handling increase contamination by discouraging good work practices and strict attention to housekeeping. Existing work tables with downdraft ventilation have not captured contaminants completely. Information in the hearing record nonetheless indicates that a lead-in-air level of 100 $\mu g/m^3$ is feasible for hand stacking operations with well-designed ventilation, strict housekeeping, and careful work practices. (Ex. 26; Ex. 101A, Ex. 29(29A), p. 27; Tr. 2587.) One small manufacturer has attained levels between 50-100 $\mu g/m^3$ for this operation. (Tr. 5977.)

To attain a level of 100 $\mu g/m^3$ in hand stacking operations, IHE recommended a rack hood, scrap barrel, and downdraft tables with larger work surfaces and enough exhaust to create a capture velocity of 250 feet per minute at the top of the stack. (Ex. 29(29A), p. 27.) Thomas preferred a downdraft ventilation greater than 400 linear feet per minute and additional ventilation drawing air away from the operator. (Ex. 101A.) Attaining a level of 50 $\mu g/m^3$ in hand stacking operations may await innovations in both technology and incentive work practices. Until such changes occur, administrative controls with some reliance on respirators may be necessary.

Machine stacking operations can be modified to conform to a level of 50 $\mu g/m^3$. IHE recommends stacking machines designed with more complete enclosures and with heavy duty doors that ensure easy access for operation and maintenance as well as downdraft tables, moveable hoods, and a hooded scraps barrel. (Ex. 29(29A), p. 28; Tr. 3710-11.) Stacking machines may not be appropriate for small operations because they are not designed to handle all the odd sizes that small plants often produce. Also, stacking machines generate additional plate scrap. (Ex. 127, p. 3-43.) Based on such information in the hearing record, OSHA expects that meeting the PEL of 50 $\mu g/m^3$ will require the use of administrative controls and respirators until stacking machines that permit almost total enclosure are designed, but that it should be possible within the time given in the implementation schedule.

(i) *Burning.* Burning operations vary greatly from plant to plant, and it is difficult to predict appropriate engineering controls from the record. Some burning operations do not require local exhaust ventilation to stay below the current standard of 200 $\mu g/m^3$ (Ex. 180; Tr. 5285; Tr. 5317), but average exposures surpass 100 $\mu g/m^3$. (Ex. 26, p. 5-49.) In those plants where burning is conducted apart from plate stacking or handling, the installation of local ventilation can reduce exposure below 100 $\mu g/m^3$. (Ex. 101A.) Such data suggests that separate burning operations may be controlled to 50 $\mu g/m^3$ with isolation, preventive maintenance, control of air velocities, and modifications that allow meticulous housekeeping. (Ex. 101A, Tr. 3711-12.)

Stations that both stack and burn plates present particular difficulties. Because dropping groups of plates into a burning container or battery case creates a moment of intense air displacement, IHE characerized controlling this operation below even 200 $\mu g/m^3$ as a "continual battle" (Tr. 3711-12). IHE concluded that only an elaborate automatic valving system can control the problem without impeding the burning. (Tr. 3211.) IHE also recommended "more and better ventilation." (Tr. 3712.) The PHS study suggested that better housekeeping and more vacuum cleaning would improve stack and burn operations. (Ex. 6(45).)

Use of a COS stacker, a machine that accepts stacked plates from a stacking machine and automatically groups and burns the plates, can reduce the unit cost of control for larger plants. (Ex. 29 (29A), p. 28.) Like stacking machines, however, COS stackers may not be appropriate for small plants. (Ex. 127, Ex. 3-43.) The record demonstrates that traditional engineering techniques supplemented by strict attention to housekeeping and maintenance can control the operation of a COS stacker to 50 $\mu g/m^3$.

(j) *Assembly operations.* The record indicates that most assembly operations can attain a level of 100 $\mu g/m^3$. In supporting such a conclusion, the PHS study suggested that exposure of some assembly employees is a result of their proximity to other, more dusty operations. (Ex. 6(45)). Thus, in some assembly operations a 50 $\mu g/m^3$ level may be readily attainable when dusty operations are isolated, housekeeping meticulous, and maintenance rigorous.

(k) *Reclaiming.* The UAW observed that "salvage and reclaim operations are often the worst exposure and last controlled operation in a battery plant." (Ex. 180; Tr. 5285.) Nonetheless, the hearing record indicates that both wet and dry reclaiming systems can attain exposure levels of 100 $\mu g/m^3$ through engineering controls or process modifications. For large plants with wet reclaiming, IHE foresees an automated, hopper-loaded, wet tumbling system from which the reclaimed paste would be pumped to the mixer for reuse. (Ex. 29(29A), p. 34.) Because such recycling requires additional equipment for handling, IHE believed it would be uneconomical for small plants. For smaller plants, IHE envisioned an enclosed system in which the plates remain in scrap barrels during washing. (Ex. 29(29A), p. 34.)

Dry reclaiming produces large quantities of dross and requires careful control. (Tr. 5319) Caplan detailed one possible arrangement for attaining a level of 100 $\mu g/m^3$ in dry reclaiming operations: "The melt pot would be totally enclosed and scrap fed to it by a skip hoist. The skip hoist would be operated from outside the enclosure while the doors to the enclosure are closed. The empty barrels will be

highly contaminated, both inside and outside, and must be thoroughly washed before returning to the production areas. Consequently, there must be a barrel-washing station adjacent to the dry reclaim areas. To eliminate dusting while drossing, the drossing operation must be conducted from outside the enclosure with the drossing ladle kept within the enclosure. The dross pot enclosure, pig-casting station and barrel-washing station require adequate ventilation. The exhausted air would pass through a scrubber before being released to atmosphere. The exhaust air cannot be used for recirculation. Water consumption at the barrel-washing station would be approximately 0.2 gallons per battery." (Ex. 29(29A), p. 34.)

(1) *Other operations.* Secondary operations in battery manufacturing include forming cases, testing, warehousing, and shipping. IHE suggestes that if the preceding processes are well-controlled, exposures in secondary operations would be below 100 $\mu g/m^3$. (Ex. 29(29A), p. 37.) OSHA has concluded that a level of 50 $\mu g/m^3$ can be achieved in secondary operations if contamination from other areas does not occur and if workroom structures and surfaces are designed to permit careful housekeeping.

OSHA has concluded that conventional engineering controls and work practices are available to the battery manufacturing industry to meet the PEL. The compliance schedule of 2 years for the 100 $\mu g/m^3$ interim level and 5 years for the PEL is based on a combination of the relatively extensive equipment or process modifications larger battery plants must make to comply with the standard and the economic constraints on small manufacturers.

d. *Brass and bronze foundries.* (1) *Introduction.*—The lead content of copper based alloys, i.e., brass and bronze, may amount to as much as 20 percent by weight of the metal core. (Tr. 2786.) The lead content of copper based ingots averages 5 percent. (Ex. 26, pp. 5-73.) Over 1,620 foundries cast brass and bronze at least occasionally; in approximately 770 foundries brass and bronze are the primary raw materials. Most of these foundries are small, 75 percent employing fewer than 50 people. Although small, most of these foundries make a diverse range of products of varying price, size, and composition. (Ex. 26, pp. 5-73.) An estimated 26,000 employees are exposed.

Exposure to airborne lead results from insufficient control of fumes from the smelting or pouring of alloys. In copper-base alloy foundries, approximately 15 percent of the particulate matter in furnace stack gases from the melting of red and yellow brass is lead oxide, and up to 56 percent of the particulate matter has been shown to be lead oxide when the alloy has a high lead content. Any workers in the vicinity of the melting or pouring operation as well as employees working to operate or maintain baghouse dust collectors may be subject to inhalation of these lead containing fumes. Sources of airborne lead may also include areas where castings are cut or finished and areas where scrap is received or stored. Levels of exposure are highly variable and depend on the amount of general and local ventilation, the lead content of the alloy, the type of furnace, and the quality of housekeeping procedures. (Ex. 26, pp. 5-73, 5-75.)

(2) *Summary.* The hearing record indicates that brass and bronze foundries can achieve an exposure level of 100 $\mu g/m^3$ with 1 year. DBA concluded that feasible engineering controls are available to meet this level. (Ex. 26, p. 5-73, Tr. 800.) DBA found that most plants do not at present have enough control in effect. Significant improvements are necessary for compliance with the proposed standard. For example, half the plants currently do not use baghouses and the majority do not provide heated make-up air. Gary Mosher, representing the American Foundrymens Society, explained that "exhaust systems have been devised and designed that will close capture * * * fumes right at the ladle and the furnace." He further testified that such methods are effective in bringing exposure below 200 $\mu g/m^3$, but did not express an opinion as to whether such techniques are effective in bringing exposure below 100 $\mu g/m^3$. (Tr. 2801.)

OSHA, however, has concluded that conventional technology in the industry has been shown effective for lowering exposures from melting and pouring to 100 $\mu g/m^3$. Refinement and development of these technological changes should permit, over time, compliance with the PEL. Examples of these controls include: (1) The adoption of electrical induction furnaces with local exhaust ventilation installed during the initial furnace installation; (2) covered ladles; (3) segregated melts; (4) use of the Hawley Trav-L-Vent; and (5) increased use of dilution ventilation and directional ventilation during pouring. Compliance will, of course, also require comprehensive housekeeping, maintenance, employee training, work practices, and personal hygiene.

(3) *Specific Operations.* (a) *Molding.*—Because most foundries are small operations, construction or cleaning of molds is often done in the same area as the pouring. Molders can be exposed to airborne lead from the furnace or pouring operations. Good control of furnaces and pouring operations thus lowers exposures during molding. (Ex. 26, p. 5-75.)

(b) *Melting.* In foundry operations, solid metal is placed in an electrical induction or gas-fired furnace. The furnace melts the solid metal and raises the temperature to that proper for pouring (1,800-2000° F). As the metal is being melted, fumes containing lead are released. When the molten metal is ready for pouring, dross is skimmed off the surface of the molten metal. Skimming increases the amount of fumes released. Without proper controls, lead exposure in this area may be high. (Ex. 26. p. 5-75.)

A combination of local exhaust and general room ventilation appears necessary to reduce airborne lead to acceptable levels. These ventilation systems have been demonstrated to be feasible for controlling lead levels below the proposed permissible level. Electric furnaces are a further aid in reducing exposures to lead because they emit fewer fumes than the older gas-fired units. (Ex. 26, pp. 5-76, 5-80.)

(c) *Pouring.* Pouring can be performed at several stages and is normally done in the transfer of molten metal from the furnace to the ladle and from the ladle to the mold. Lead fumes are released during the pouring operation.

A combination of local and general ventilation is necessary to control employee exposures to below 100 μ/m^3. In foundry operations, a mobile ventilation system that attaches directly over the pouring ladle or crucible is useful for removing the bulk of fumes from pouring operations. Before the alloy is poured, dross is skimmed from the surface of the melting pot. This dross should be deposited in a barrel with a mobile ventilation system used to capture fumes. Fixed position hoods are possible in foundries using automatic pouring of standard sized molds. Fumes captured should be vented to a baghouse, and tempered makeup air should be provided.

(d) *Other operations.* Local exhaust systems that attach directly to grinding wheels or other finishing machinery are available. Grindings and scraps should be stored in closed containers. In both of these operations as well as at the baghouse, use of respirators may sometimes be necessary.

The compliance schedule for this industry is based on both technological and economic factors. Since lead levels are not too high in foundries, and since relatively simple and conventional controls are required to comply with the interim level 1 year is given to implement the necessary controls. Since further refinement of these controls will probably be necessary to attain

the PEL, 5 years is provided. This period includes economic considerations. (See the discussion in the economic section of this document.)

e. *Pigment manufacturing.* (1) *Introduction.*—Of the 114 plants that manufacture pigments in the United States, approximately 25 produce pigments containing lead. Pigment products include red lead (or, litharge), lead sulfates, lead carbonates, lead silicates, lead oxides, and lead chromates. Inorganic pigments are a prime component in surface coatings and important components in other products such as linoleum, rubber, and plastics, inks, ceramics, and paper coatings. Litharge is used principally in the manufacture of products other than paint (i.e., ceramic glazes, batteries, glasses, and vitreous enamels). (Ex. 26, p. 5-92.) The number of production employees in lead pigment manufacturing is estimated to be 2,000. DBA's survey of several plants indicated that 90 percent of the workers were exposed to levels of lead above 100 $\mu g/m^3$. (Ex. 26, p. 5-93.)

The manufacture of pigments involves a number of different processes. Only pulverizing and grinding processes for reducing the particle size are common to all members in the class. Inorganic pigment manufacture is a combination of chemical-physical processes involving both wet and dry reactions, including precipitation, filtering, washing, fusing, calcining, etc. The processes may be carried out as a batch system, as continuous production, or as a combination of the two.

Pig lead is often the basic raw material in inorganic lead pigment. Litharge and other lead forms, however, are sometimes used. Because litharge is a powder, it presents the potential for lead exposures at every transfer point. Filtering, drying, grinding, sizing, grading, blending, and bagging are all considered to be areas of potential exposure to lead. Cross contamination between operations also occurs.

(2) *Summary.* Most pigment plants are old. All but five plants visited by DBA were at least 50 years old. One plant was said to be 129 years old. (Ex. 26, p. 5-95.) Because of the age of the facilities, retrofitting may not achieve levels below 100 $\mu g/m^3$, although such methods have reduced air-lead levels to 200 $\mu g/m^3$. However, redesign of the process, including "total enclosure of certain steps and/or automation" is expected to be able to reduce levels to a 100 $\mu g/m^3$ level. (Ex. 26, p. 5-98.) The same conclusion applies to the 50 $\mu g/m^3$ PEL. As Dr. First explained, "every operation that can be mechanized and automated is capable of being enclosed by tight physical barriers and placed under slight negative pressure to prevent outleakage of dust or fume-laden air to the workroom" (Ex. 270, pp. 29-30). While such technology may require time and money to install, it is available and adaptable to the pigment industry.

The National Institute for Occupational Safety and Health has recently recommended that OSHA require employers to reduce exposures of hexavalent chromium to 1 $\mu g/m^3$. A number to dry color manufacturers produce lead chromate pigments. If a hexavalent chromium standard were adopted, it would also play an important role in controlling lead exposures.

Finally, using substitutes for lead pigments, such as organic pigments, would eliminate exposures. While substitutes may not exhibit all the properties of lead, such as resistance to corrosion and weathering, they would nonetheless be adequate in many cases. Such substitution would also reduce or eliminate exposures in all the industries that involve lead pigment-wallpaper manufacturing, glove manufacturing, pottery manufacturing, ink manufacturing, paint manufacturing, shipbuilding, and automobile manufacturing.

f. *Shipbuilding.* (1) *Introduction.*—The shipbuilding industry includes repair as well as construction of ships. The size of the shipyards and the type of vessel being repaired or built vary widely. Exposure to lead can occur during lead burning, painting, welding, and sandblasting. The number of workers exposed to lead during burning is estimated at 1,374, during sandblasting at 264, welding 16,120, and painting 4,495. However, employees often work at a variety of assignments; their work may expose them to lead only 1 or 2 days a week. (Ex. 26, pp. 5-110 and 5-111.)

Exposures during welding can originate in the base metal being welded, the coatings used on the electrodes, and the coatings on the base metal. The studies reviewed by DBA indicate that welders may be exposed to concentrations of lead well in excess of 100 $\mu g/m^3$. One 1968 study reported mean lead concentrations of 40 $\mu g/m^3$ in shipyard welding. DBA estimates that the exposures of 81 percent of welders would fall above 100 $\mu g/m^3$. Especially high exposures result from work in confined spaces and on galvanized metals coated with zinc silicates. (Ex. 26, pp. 5-113, 5-114.)

Lead burning occurs only in the construction of nuclear ships, when lead is welded to the hull in order to shield the ship's reactor. DBA estimated that 40 percent of these workers have exposures above 100 $\mu g/m^3$. (Ex. 26, p. 5-111.) Sandblasting is used to remove all coating material, including those containing lead, before painting the hull of a vessel. Although few data are available, exposure levels of sandblasters are believed to exceed the PEL. (Ex. 26, p. 5-117.) Painters, in contrast, are assumed to fall into the low exposure category. When painting is not done in a ventilated spray booth, however, most painters now wear respirators. (Ex. 26, p. 5-117.) Lead-based paint is being replaced: Some shipyards use no lead-based paint. (Ex. 22, p. 321.) The following table 1 summarizes exposures:

TABLE 1.—*Estimated Employee Exposure to Lead in Shipyards by Occupation*

[(Ex. 26, p. 5-118) (corrected)]

	Total	High exposure	Medium	Low
Welders	16,120	3,000		13,120
Painters	4,495			4,495
Sandblasters	264	264		
Leadworkers	1,374	622	622	130
Total	22,253	3,886	622	17,745

(2) *Summary.* The hearing record indicates that attaining exposures of 100 $\mu g/m^3$ is generally feasible in the shipbuilding industry. The Shipbuilders Council as well as General Dynamics and Ingall Shipbuilding, division of Litton Industries described the proposed standard as "technologically possible." (Ex. 230, p. 2; Ex. 28(30), p. 3; Ex. 3(58), p. 2.) DBA concluded that the shipbuilding industry has achieved the state-of-the-art in engineering controls. (Ex. 26, p. 2-9.) Stan Phillips, testifying for DBA, stated that "engineering controls are feasible for many lead burning and lead foundry operations in the shipbuilding industry." He further explained that there would be limited occasion, such as lead burning in confined spaces, where respirators may be needed." (Tr. 800-801.) Periodic use of respirators may also be necessary for compliance with the 50 $\mu g/m^3$ standard.

Shipbuilding is not a process that uses a production line or stations to which engineering controls can be attached. As ship construction proceeds, the work environment changes. Compliance in such an operation calls for local exhaust ventilation, using portable, flexible equipment, and absolute filters. Some confined spaces, however, may not have room for portable ventilation ducts. (Ex. 26 p. 5-119.)

Large shipyards have installed such controls. Complying with the standard may be more difficult for some small companies, especially during work on hulls painted with lead-based paints. If the proper welding practice of removing an area 3" wider than the weld is strictly followed, exposure above the PEL is less likely. (Ex. 26, p. 5-119.) Also, replacement of lead-based paint will reduce exposures in welding and repairing. (Ex. 22, p. 323.)

g. *Automobile manufacturing.* (1) *Introduction.*—The Motor Vehicle Manufacturing Association characterizes control of the lead in the working environment as "one of the most significant hygiene problems faced by the manufacturers of motor vehicles for over 40 years." (Ex. 3(2), p. 6.) The primary manufacturers of automobiles and light-duty trucks—General Motors, Ford, Chrysler, American Motors, and International Harvester—use lead primarily in the form of solder for a number of operations in the manufacture of automobiles. The amount of solder, which is approximately 95 percent lead and 5 percent tin, is estimated to range from 2 to 30 pounds per vehicle. (Ex. 26, p. 5-133.)

Solder is widely used in the body shop, for both structural applications and sheet metal work. Typically, solder is used to attach hinge pillars, center pillars, and upper back seams. Soldered body surfaces are smoothed with grinding wheels and polished. (Ex. 26, p. 5-133.) At the solder booths, airborne levels of lead may be very high. (Tr. 5249.) Lead exposures occur in these operations and in all subsequent operations until the body is painted.

Spraying of lead based primers and paint can itself be a source of exposure. In some companies, employees are exposed to lead during dipping of wire and heat treating of metals. Minor exposures may occur in engine facilities, body engineering stamping, and brake manufacturing. (Ex. 26, p. 5-133.)

(2) *Summary.* Industry in general has not yet found engineering methods alone practical for controlling airborne lead produced by the use of power tools on solder.(9) To control exposures, the industry has developed exhaust-ventilation booths in which grinders must also wear air fed helmets known as hoods. The industry has thus combined engineering controls with elaborate personal protection equipment. The Motor Vehicle Manufacturers Association asserted that "the technical state-of-the-art regarding engineering and administrative controls have (sic) been reached." (Ex. 28(36).) Refinements of the process, of course, are still possible. Two companies have reported some success with high velocity/low volume tool ventilation systems. (Ex. 26, p. 5-135.)

Spokesmen for the UAW suggested that not all feasible engineering controls have been installed. Dr. Mirer, for example, testified, "the essential engineering design feature of the grinding booth is that it is a negative pressure enclosure that seeks to contain the airborne lead, but the design specifications do not include measures to reduce the airborne lead by such measures as a downdraft or a specified capture velocity downwards." (Tr. 5252.) Frank Nix, health and safety representative of UAW Local No. 10, stated that in his plant, particles are thrown out of both ends of the booth and, because the bodies do not go through a water wash after grinding, subsequent workers on the assembly line are exposed to lead. He also expressed concern about lack of a grinding booth for repair work. (Tr. 5242-47.)

Based on the record, OSHA has concluded that until changes in design or material take place, the combination of engineering controls and airline hoods now in use appears necessary to insure that grinding complies with either a 100 or 50 $\mu g/m^3$ standard. Improvements in engineering controls, however, are expected to reduce levels significantly since this industry has historically displayed great ability to make technological change when necessary. An alternative to engineering controls would lower exposures by decreasing the amount of solder. Mirer stated that "ultimately, the only solution is engineering the solder out of the car body by redesign of the body or finding a substitute material for filling out the seam." (Tr. 5249.) Industry has already reduced solder in automobiles by substituting plastics and epoxees. One new line of cars has eliminated the use of solder in production. (Ex. 26, p. 5-133.)

Substitutes for lead-based paints would reduce exposures in spray painting. Industry, however, asserted that the qualities of lead-based paint, such as its resistance to corrosion, make substitution infeasible. (Ex. 28(6), p. 3.) In these cases, engineering controls are available. According to Mirer, "such exposures should be controlled by efficient local exhaust ventilation in downdraft or backdraft paint spray booth, without resorting to the use of respirators" (Ex. 180). The Short study suggested spacing of employees within the booth. (Ex. 22, p. 229.) For sanding of surfaces after painting, Mirer recommended ventilation and high-velocity/low volume exhaust systems or wet grinding and cleaning methods. (Ex. 180, p. 20.) Automated spray booths are available but, according to J. I. Case, a manufacturer of industrial and consumer products, such automated booths are not suitable when a variety of conditions must be met. (Ex. 28B, p. 4.) Peter Schultz, testifying for J. I. Case, also stated that manual touchups are needed after automatic spraying (Tr. 1209).

Unlike Mirer, the Short report concluded that at least in some cases exposures may exceed 100 $\mu g/m^3$ even in a adequately designed, operated, and maintained booth. (Ex. 22, pp. 228-29.) OSHA has concluded that attaining the PEL, with engineering and work practice controls is feasible but that a limited number of specific operations may require reliance on supplemental respiratory protection.

h. *Electronics.* Exposure to lead in this industry occurs primarily during soldering. Based on the data submitted by two large companies in the industry, Zenith and Motorola, OSHA has determined that compliance with the standard is feasible. Zenith reported extremely low levels, 2.1 to 8.0 $\mu g/m^3$ respirable lead-in-air for eight of its solderers. (Ex. 3(75).) Motorola also reported very low levels, 10-12 $\mu g/m^3$. (Ex. 3(66).) These levels are a result of local exhaust ventilation already in place, and thus it appears feasible to maintain air lead concentrations for the 500,000 exposed employees well below 50 $\mu g/m^3$. Additional ventilation may be necessary at stations where solder is cut and trimmed. Motorola reports levels at these sites of 67 $\mu g/m^3$ and 73 $\mu g/m^3$, slightly above 50 $\mu g/m^3$. (Ex. 3(66).)

i. *Solder Manufacturing* (1.) *Introduction.*—There are 100 companies operating 120 plants that manufacture solder. Thirty of the larger companies manufacture 90 percent of the solder. Solder is sold in the form of ingots, rods, bars, anodes, solid wire, cored wire, foil, sheet and paste. Among its many uses, solder is essential for the manufacture of electronic devices. No substitutes for solder are known. (Ex. 65B, p. 40-42.)

Refined lead is used to make lead-tin and other solders. The ratio of lead to tin, bismuth, antimony and other metals varies. In the making of solder, metals are melted down at low temperature and blended in extablished ratios. Handling of lead is minimal, but employees do handle new lead ingots before they are melted. In the melting and blending of lead, the tem-

perature is kept at 600-850° F, where few fumes are formed, because lead oxide, which ruins the solder product is formed at temperature over 1,000° F. The dross formed, however, does present a possible hazard. Finished solder is cast into blocks, ingots, rods or bars, sheets and foil, and extruded into solder wire and sheets. (Ex. 22, p. 294.)

Approximately 3,500 people in this industry are potentially exposed to lead. (Ex. 22, p. 295.) Exposure levels in some areas exceed the PEL. Recent OSHA inspections at two solder plants reported levels above 200 $\mu g/m^3$ in spooling operations, furnace areas, and kettle areas. (Ex. 65B, p. 42.) One company reported that even with excellent ventilation, lead levels in the casting area reached 200 $\mu g/m^3$. (Ex. 22, p. 294.)

Powder blowing of molten solder is acknowledged to be the most hazardous operation in solder manufacturing. Powder is blown from a molten reservoir by feeding a steady stream of molten solder through an air nozzle. By the time the metallic droplets solidify, they settle into the bottom of the equipment where they are sized down to 400 mesh and finer. (Ex. 65B, pp. 40-41.) Other areas where exposure occurs are the spooling and wire drawing operations, where air-lead levels range from 100 to 300 $\mu g/m^3$. (Ex. 22, p. 294.)

The extrusion of solder into wire and other shapes is usually done by hydraulically pressing the solder slug through a dye into its final shape or further reducing its diameter by drawing it through dyes normally submerged in a lubricated liquid. This cooling liquid, which is also called a drawing solution, contains fine particles of solder that may cling to the wire as it leaves its drawing machine at high speeds. These particles may then become airborne during spooling and handling. (Ex. 65B, p. 41.)

(2.) *Summary.* Most employers protect employees from lead exposure by ventilation. Hoods, exhaust fans, vents, air ducts, and baghouses are usually present. (Ex. 22, p. 294.) Most of the eight companies contacted by Short did not anticipate problems in complying with a level of 100 $\mu g/m^3$. (Ex. 22, pp. 297-298.)

Additional ventilation may be necessary in some areas for compliance with the PEL. In a few areas that are difficult to ventilate, such as spooling, other protective measures may be necessary. Slowing the spool rate is one possible method for controlling lead levels in the spool and wire drawing area, although this method would decrease the production rate. Other administrative controls may also be possible. (Ex. 22, pp. 295.) OSHA has determined that compliance with the standard as a whole is feasible for the industry within the year allowed in the compliance schedule.

j. *Gray Iron Foundries.* (1.) *Introduction.*—An estimated 80 percent of all durable goods contain gray iron casting. The automobile industry, the largest single consumer of gray iron castings, uses about 25 percent of all cast iron products. (Ex. 65B, p. 24.) The raw materials for casting gray iron are iron scraps, a relatively small amount of pig iron, and small quantities of coke, sand, clay, iron alloys, and nonferrous metals. More than half the scrap used as raw material is obsolete scrap or waste materials. DBA suggests that most of the lead, which is an undesirable constituent, enters the process through particular types of obsolete scrap, such as bearings or other parts of automobile engines, pipe fittings, lead-based paints, soldered seams in steel cans, and some types of railroad bearings. (Ex. 65B, p. 25.) The ratio of scrap to pig iron has increased in recent years because of the growing use of electric furnaces, which can be fed exclusively on scrap. (Ex. 65B, p. 24.)

In March, 1977, 1201 foundries reported that they cast gray iron as their primary metal. About half of these foundries employ fewer than 80 people; about 20 more than 1,000. (Ex. 65B, p. 24.) Most gray iron foundries, especially the small ones, pick over their scap before melting, although in large automated foundries heavy machinery may handle the scrap. Also, employees break any lead or other resaleable metals out of the iron scrap, a procedure that significantly reduces the amount of lead entering the furnace.

Because lead is extremely insoluble in iron and will not combine with it during melting, any lead that does enter the furnace may combine with air to form lead oxide. DBA reported that analyses of flue dusts from three iron foundries showed lead contents of 6.3 percent, 6.6 percent, and 17 percent. Other foundries reported at most a trace of lead in their flue dusts. (Ex. 65B, p. 28.) If large slugs of lead scrap are loaded into the furnace, the dense lead may immediately fall to the bottom and become superheated, although in the absence of air, no lead oxides would form. Such deposits are recognized as a problem and avoided. (Ex. 65B p. 24.)

Lead exposures do not occur in all gray iron foundries. DBA concluded that the likelihood of exposures depends on the amount of lead in the raw materials, the care with which scrap is picked over before melting, the type of furnace in use, the location of the furnace inside, or possibly outside, the foundry, and the type of pollution control equipment in use. Furthermore, lead exposures are most likely to occur in the 40-200 plants that cast products such as counterweights, manhole covers and frames, door jams, machinery bases, bath tub cores, and other products in which weight is important. These products are likely to be composed of a lower grade of scrap, which might contain lead.

Also, the 536 foundries that use electric induction furnaces are more likely to produce lead exposures than those using cupola furnaces. Cupola furnaces are often located outside. Gases from a cupola furnace rise directly into the control equipment. Only employees close to charging, tapping, and pouring stations could be exposed. In contrast, coreless electric induction furnaces require close capture hooding to avoid the continuous escape of fumes. However, no significant levels of exposure have been documented at any existing plants. (Ex. 65B, pp. 25, 27), suggesting that lead exposure in gray iron foundries presents minimal control problems.

(2) *Summary.* For those iron foundries where lead is discovered to be a problem, DBA recommended the following procedures: (1) Purchasing where possible a grade of scrap certified not to contain lead; (2) examining and separating scrap more completely; (3) installing control equipment, such as close-capture hoods at electric furnaces. (Ex. 65B, p. 27.) Given the absence of current problems with exposure to lead, OSHA has concluded that compliance with the 50 $\mu g/m^3$ standard is feasible in the gray iron foundry industry within 1 year.

(k) *Ink Manufacturing.* (1) *Introduction.*—Approximately 100 of the 479 plants that manufacture inks use lead pigments; 1,000 to 1,300 employees are potentially exposed to lead. In addition, 50 "captive" ink producers (companies that produce ink for their own use) handle pigments containing lead. Many of these producers, however, are included in other sectors, as, for example, wall covering.

In ink manufacturing, the principal lead pigments are lead chromate, molybdate orange, and phloxine red. DBA estimated the 1976 consumption of these pigments in the captive and noncaptive sectors to be 20 million pounds of lead chromate, 8 million pounds of molybdate orange, and over 2 million pounds of phloxine red. (Ex. 65B, p. 37.)

Exposure is largely confined to operations involving dispersion of pigments in solvents or oils. Once the pigment is formulated into inks, the potential for exposure to lead decreases. Based on figures submitted by the National Association of Printing Ink Manufacturers, Inc. (NAPIM), DBA developed the

following characterization of exposures: (Ex. 65, p. 38).

Number of exposed employees	TWA exposure to lead-in air $\mu g/m^3$
320 to 416	200
480 to 624	100-199
200 to 260	50-99

(2) *Summary.* Local exhaust ventilation and increased housekeeping will probably be necessary in most plants where dry pigments are still handled. In addition, disposal of pigment containers (bags, etc.) will require close attention. OSHA does not anticipate that there will be exposure to lead in excess of the the PEL associated with the handling of pigment concentrates, pastes, inks, etc., which are not in dry form; reducing the use of dry pigments will also reduce exposure. Some of the major ink manufacturers, for example, are formulating base inks at central locations and shipping the base inks to satellite formulation plants. (Ex. 65, p. 38.)

1. *Paint Manufacturing* (1) *Introduction.*—Lead chromates are essential pigments in yellow, orange, and red coatings for exterior industrial, and maintenance use. In 1973, 23 million pounds of lead chromate were used in paint used for yellow traffic markings alone. Metallic scraps of lead, which are added to oil based paints to speed drying and create a durable film, are another important use of lead compounds. In 1973, the paint manufacturing industry used over 3 million pounds of lead dryers. (Ex. 65B, p. 35.) It is estimated that 9,000 employees are exposed to lead. (Ex. 22, p. 222.)

Paint ingredients are mixed in vessels of various types, with capacities ranging from 10 to several thousand gallons. Exposures occur primarily during the preweighing of additives and the transferring of lead pigments into the mixer. Exposures during paint mixing, however, are limited to a brief period during the operating shift. In these two operations, approximately 5,400 employees could be exposed to lead bearing pigments. Exposure levels reported to DBA ranged from 0 to 1,000 $\mu g/m^3$. (Ex. 65B, p. 35.)

(2) *Summary.* DBA attributed the wide disparity in reported exposure levels to the design and condition of the exhaust ventilation systems. (Ex. 65B, p. 35.) Thus, compliance with the standard appears feasible with well designed and maintained systems. In smaller plants, administrative controls may also be important. While the methods of production in small plants are similar to large plants, the frequency of use is not. Some operations may wait 6 to 9 months between batches of lead bearing chemicals. DBA reported that lead paint mixing occurs approximately 15 to 30 minutes, once or twice per month for one small operation. (Ex. 65B, p. 35.) With use of engineering controls, work practices, administrative controls, and good housekeeping, OSHA has concluded that compliance with the standard is technologically feasible in paint manufacturing within the 1 year permitted for compliance.

m. *Wallpaper Manufacturing.* (1) *Introduction.*—In the manufacture of wall covering, exposure to inorganic lead occurs primarily during the handling and use of dry pigments. Some manufacturers still receive pigments in powder form and disperse them into oils and solvents. The weighing and measuring of the pigments, adding the pigment, and disposal of pigment bags can produce significant exposure to lead. Once the pigments are contained in pastes or inks, the problem of lead exposure is reduced. (Ex. 65B, p. 33.)

The Wallcovering Manufacturing Association (WMA), which has 18 members (75-80 percent of the industry), reported to DBA that dry pigments are handled and dispersed in approximately 20 plants and that approximately 400 persons are exposed. In addition, as many as 1,000 persons may handle pigment concentrates or ink containing lead. Such products, however, do not pose a major exposure problem. DBA was unable to obtain data on air monitoring. (Ex. 65B, p. 33.)

(2) *Summary.* Firms that use dry pigments supply exhaust ventilation and require employees to wear respirators. DBA reported that local exhaust ventilation and increased housekeeping will probably be necessary in most plants where dry pigments are still handled. In addition, disposal of pigment containers (bags, etc.) will require close attention. DBA did not anticipate that engineering controls will be necessary to control exposures to lead associated with handling pigment concentrates (paste), inks, etc. Several firms reported that they no longer handle dry pigments. (Ex. 65B, p. 33.) Thus, OSHA believes that through engineering controls or a reduction of the operation, use of dry pigment in the wallcovering industry can be controlled to 50 $\mu g/m^3$.

n. *Wire Patenting.* (1) *Introduction.*—Wire patenting is the quenching of ferrous wire in order to achieve certain desired properties, primarily high tensile strength. The wire is fed through a pot of molten lead. Lead flakes from the coiling operation or from the handling and storage of processed coils accumulate on surfaces. (Ex. 65B, p. 72.)

Approximately 100 plants patent wires. Two large companies with seven plants reported that 25 to 40 percent of their employees are exposed to lead. Based on these percentages, DBA estimated that 2,000 employees in the industry are exposed to lead. Little information is available on exposure. One source reported that lead-in-air concentrations average between 100 and 200 $\mu g/m^3$, although exposures may sometimes exceed the present standard. (Ex. 65B, p.22.)

(2) *Summary.* Because the process temperature of the quenching pot is just above the melting point of lead, the fumes emit only low levels of lead. In addition, the molten lead is covered with a floating layer of coke, charcoal, or a similar material to reduce fugitive emissions. Exhaust hoods are placed over these pots to capture lead fumes.

To meet the 50 $\mu g/m^3$ limit, very efficient ventilation systems along with necessary housekeeping programs, will be required to control lead fumes and dust. From the limited information available, DBA concluded that many plants will need some additional ventilation and housekeeping efforts. Two companies reported current compliance with the 100 $\mu g/m^3$ level. (Ex. 65B, p. 22). Thus, the 100 $\mu g/m^3$ level clearly appears feasible.

The Stelmor process, which uses air as the quenching medium, eliminates the molten lead process. Approximately 25 steel works are now using the Stelmor process, and it appears to be replacing lead wire patenting. (Ex. 65B, p. 22.) Through either this new technology or through aggessive implementation of engineering controls, the 50 $\mu g/m^3$ standard is considered to be feasible.

o. *Can Manufacture.* (1) *Introduction.*—Approximately 80 percent of steel cans have soldered side seams, and some larger cans also have soldered bottom seams. The solder used is 50-90 percent lead. Special machines, operating in a production line, perform the operation and solder several hundred cans an hour. The cans are first preheated. Next, either the seam is dipped in a solder bath at approximately 650° F or a roller lays solder in the seam. A rapidly rotating cloth-covered spindle then wipes off the excess. If the cans are dipped, a flux is layered on the top of the solder both to facilitate bonding and control lead emissions. In some operations, the cans are allowed to cool after the solder is applied, then reheated for wiping. Excess solder wiped off the cans is recycled into the bath.

Each production line is attended by an operator who monitors the solder level of the bath, recycles the excess solder from the can wiper, and monitors the operation of the remainder of the can line. The total number of workers exposed to lead in can manufacturing is estimated at 1,200-2,000.

Little exposure data are available, but two of the companies surveyed by DBA had data showing personal exposure levels to be generally between 0.002-0.4 $\mu g/m^3$ TWA, where ventilation controls were operating satisfactorily and there were no process upsets. (Ex. 65B, p. 19.)

(2) *Summary.* All four of the individual companies contacted by DBA and the Can Manufacturer's Institute indicated that, throughout the industry, solder baths are generally hooded and that most solder wipers are hooded or in the process of being hooded. The temperature of the solder bath is closely controlled with frequent monitoring to insure good quality control on the soldered can seams. Solder bath lead emissions are most likely to occur when the bath is being charged, but such emissions are usually captured by the ventilation systems.

Reheating the soldered cans with an open flame to prepare them for wiping may also be a source of lead emissions. The magnitude of this problem is uncertain. One company noted that lead dust emissions from the can wiper can present a problem through direct worker exposure and to housekeeping. At least one company uses area vacuuming to control lead dust. Maintenance activities on the soldering and can wiping areas of the can line can also be sources of worker exposure. None of the companies use respirators to control exposures.

Given the low levels of exposure, OSHA has concluded that it is clearly feasible to meet the 50 $\mu g/m^3$ standard. Present engineering controls have proven themselves both adequate and feasible and should not have to be supplemented by respiratory protection.

p. *Printing.* (1) *Introduction.*—The printing industry, which includes newspapers, periodicals, magazines, books, and commerical printing, is the third largest industry in the United States. The primary use of lead in the industry occurs in hot metal typesetting. Lead constitutes 60-80 percent of the type metal and gives the metal its low melting point. Hot metal typesetting includes linotype, monotype, and stereotype processes. All involve the same basic steps: Type is cast from the molten lead alloy (500-560° F) and remelted after printing. (Ex. 22, p. 193.)

Melting pots, where the highest exposures occur, are hooded and located in a separate room which is well ventilated. Moreover, no worker spends 8 hours in the melting area. TWA exposures appear low. Of the 175,000 workers potentially exposed at the time of the Short study, most were exposed to levels below 50 $\mu g/m^3$. Short estimated that 20 percent, or 35,000 employees were exposed to levels of lead between 50 $\mu g/m^3$ and 100 $\mu g/m^3$. (Ex. 22, p. 196.)

The introduction of cold type (photosetting) and offset processes are rapidly reducing the number of employees exposed to lead. Most newspapers have converted to cold type processes. The American Newspaper Publishers Association/Research Institute (ANPA) reports that only 134 newspapers now print with letter-press stereotypes. The population of daily newspaper employees exposed to lead has shrunk to approximately 10,850. (Ex. 65B, p. 20.)

ANPA also indicated that recent break-throughs have speeded conversion and that by 1980 very few papers would still be printed using hot metal processes. These break-throughs may be applicable to a lesser extent in other segments of the printing industry. (Ex. 65B, p. 20.) A small percentage of the industry, however, will continue to use lead type, which because it leaves a clear impression on the paper, will be chosen for small orders, finer printing and specialty type faces. (Ex. 22, p. 195.)

(2) *Summary.* One method of compliance, converting from hot type processes, is already taking place in large segments of the industry. Exposure data collected by ANPA indicate that employee exposures to lead in-air have substantially decreased in recent years and that minimal modification of existing engineering controls would be necessary to bring most establishments into total compliance with the proposed standard. Complying with the 50 $\mu g/m^3$ standard is thus feasible for the printing industry.

q. *Pottery and Related Products.* (1) *Introduction.*—The pottery industry consists of a number of large companies and hundreds of small operations. The exact number is not known. A typical large plant employs 150-300 people. Small operations may have as few as a single employee. (Ex. 22, p. 211.)

Employee exposures occur as a result of the handling, application and use of lead-based glazes. The glaze is made up of finely divided powder called lead frit, which is nonsoluble lead silicate, lead boro or bi-silicate. The frit is mixed with water and sprayed on the base structure (plates, cups, etc.) The spraying is typically done in an enclosed area. The piece is then placed on a "setter" which is introduced to a kiln for firing. (Ex. 22, p. 211.)

The exposed population includes production workers engaged in the following industries: Earthenware food utensils, vitreous china food utensils, vitreous plumbing fixtures, and porcelain electrical. The total population of potentially exposed employees is estimated to be between 500 and 5,700. (Ex. 65B, p. 47.) Exposures greater than 100 $\mu g/m^3$ could occur in handling and mixing of frit as well as spraying operations. (Ex. 22, p. 211.)

(2) *Summary.* Hooded spray areas have been installed at many plants. Compliance with the PEL will require increased housekeeping and maintenance. In addition, local exhaust ventilation will be required at frit handling stations, at mixing operations, and at spraying operations. (Ex. 22, pp. 221-212.) OSHA has concluded that with improved controls, housekeeping, and maintenance, compliance with the standard is feasible.

r. *Other Industries.* The preceding industries were examined in DBA's followup study for high priority industries. (Ex. 65B.) Most of the other industries in which employees are exposed to lead were assessed for technological feasibility in the Short report (Ex. 22.) Because these industries generally have very low lead exposure, any compliance activities will require very simple engineering controls. Short's conclusions regarding these industries' ability to comply with the 100 $\mu g/m^3$ level are equally applicable to the 50 $\mu g/m^3$ PEL.

5. *Industry Analyses and Economic Conclusions.*—(a) *Introduction.* The economic impact assessment for this standard has been made by OSHA after careful evaluation of all relevant evidence in the rulemaking record. OSHA's conclusion on this aspect of the rulemaking is that compliance by employers with the standard, under the conditions and implementation schedules contained in it, is feasible. Compliance will not cause "massive economic dislocation" to the affected industries and will not place undue inflationary pressure on the national economy. This section begins with a general discussion of problems associated with data collection and cost estimation techniques used in the principal economic studies in the record. Cost and impact analyses of DBA and CRA are compared, and differences are reconciled to the extent possible using other record evidence. Conclusions on economic impact are then presented on an industry-by-industry basis, followed by a discussion of aggregate impacts on the U.S. economy.

In making this assessment, OSHA has considered it appropriate to isolate those costs which are attributable solely to compliance with the requirements of the new, permanent standard for lead. Where employers are currently engaging in activities which the new standard mandates and are voluntarily incurring the costs associated with them (for example, biological monitoring is common throughout the lead industries) no additional costs to em-

ployers are counted. Employers also have many obligations under general and specific existing OSHA standards (e.g., permissible exposure limit for lead of 200 $\mu g/m^3$ (29 CFR s1910.1000), protective clothing (29 CFR s1910.132), and hygiene facilities (29 CFR s1910.141)), and some have additional obligations, primarily airborne monitoring and medical surveillance, under existing abatement agreements. These have been omitted to the extent possible. There are often related costs attributable to other OSHA standards (arsenic, sulfur dioxide) or to standards under other Federal or State laws, primarily air and water pollution control. Here also, this economic assessment attempts to include only those costs fairly attributable to the incremental difference between existing obligations and new obligations the standard will impose.

Isolating these costs and impacts to avoid double counting does not mean that OSHA has ignored the economic costs to affected industries from other sources. Costs from other sources, including other OSHA standards, have been considered in the same manner as any other known costs facing an industry. As such, they become part of the economics of the industry from which the likely impact of the lead standard is measured.

The DBA report and the CRA report provide much of the information used in OSHA's economic analysis. The DBA report covered a total of 17 industries and examined five industries in depth—primary smelting, secondary smelting, battery manufacture, brass and bronze foundries, and lead pigment manufacturing. These were believed to be the ones which would be most substantially affected by the proposed standard. These industries include all producers of lead and users that account for 60 percent of lead consumption in the United States (Ex. 26, p. 1-1). The criteria used to select these industries were the number of employees exposed to lead, the levels of exposure, initial estimates of compliance costs, initial estimates of value-added as a result of compliance costs, and availability of information. shipbuilding and automobile manufacturing were examined, but without full economic impact analysis. Ten additional industries were examined in less detail.

Cost of compliance were first developed for each of the five major industries, and the impacts associated with those costs were analyzed with respect to the impact on consumers, business, and government; the effect on market structure and competition; the effect on supplies of important materials, products, and services; and the effect on employment and productivity.

Cost estimates were expressed in terms of capital expenditures, annually recurring costs, total annualized costs, and additional labor and energy requirements. Capital expenditures in most cases represented the cost of engineering controls to comply with the PEL, and annually recurring costs were an aggregate of costs associated with environmental monitoring, medical surveillance, training, recordkeeping, operation and maintenance of capital equipment, housekeeping, and other requirements of the standard where recurring or continuing obligations exist. Annual costs associated with capital assets, such as depreciation and interest, were figured to arrive at a total annualized cost for each industry. (*10*)

Evaluation of the data collection process, the study methodology, and certain assumptions and generalizations employed in the DBA report has led OSHA to conclude that the actual costs and impacts attributable to the proposed standard will probably be far lower than the report estimates. A factor in this conclusion is the significant degree of "double counting" in the cost estimates used by DBA. This double counting took several forms. The one most frequently mentioned in the hearing and in the studies and comments submitted for the record is the potential overlap of expenditures for compliance with Federal or State air and water quality standards and OSHA regulations. For example, the Environmental Protection Agency has published an economic impact statement in conjunction with its impending National Ambient Air Quality Standard for lead and its cost estimates are based on dust and fume collection systems within the plant. While an effort was made by DBA to identify and eliminate costs associated with compliance with other regulations (Tr. 719, 880), this was not always completely successful (Tr. 783). Since much of the cost data were submitted by industry sources as aggregates without detailed specification, verification of the exclusion of air and water pollution control costs was not possible. David J. Burton of DBA testified that he could not vouch for every single cost estimate received from companies and that only in those cost estimates he himself developed or verified were there attempts to eliminate overlaps. (Tr. 880) But as Burton admitted, "there obviously has to be some overlap in installing OSHA controls with air pollution controls as well because particular standards, particularly the emission standards and the proposed new lead emission standards, are going to be more stringent, and, of course, most plants are not just allowed to remove the contaminated air now from the plant and put it outside as it was in years past." (Tr. 880-881). It is also likely that some of the engineering control costs submitted by primary smelters for the lead EIS are counted twice because they are for equipment which is also instrumental in controlling arsenic, sulfur dioxide, and other air contaminants (Tr. 6412-14) and would have to be expended notwithstanding the lead standard.

Another form of double counting included in the cost estimates provided in the DBA report involves the inability to distinguish engineering control costs attributable to compliance with the proposed standard from those connected with compliance with the existing standard of 200 $\mu g/m^3$ The costs of compliance with the proposed standard can theoretically be calculated for any of three increments: From an assumed base of no controls, from the existing level of control (some firms are not now in full compliance with the existing 200 $\mu g/m^3$ standard (Tr. 794, 782-83, 6407)), or from an assumed base of full compliance with the current standard. Burton stated that:

> Ideally, the costs of complying with the new requirements of the standard should be isolated from the requirements already promulgated, but not presently being complied with. For the industries studied, all of the cost estimates associated with compliance, except engineering costs, are estimates of the incremental costs of compliance. Unfortunately, it is not possible to accurately establish the incremental costs associated with engineering controls. The main reasons for this shortcoming are as follows:
>
> (1) The State-of-the-Art of the effectiveness of engineering controls is not developed to the degree necessary to make such judgments. It is thought that the same basic approaches may be used to control exposures to either 200 or 100 $\mu g/m^3$.
>
> (2) Engineering controls are often built to standard specifications which are not designed to provide a specific control efficiency, but rather the optimum control possible for the particular system.
>
> (3) Exposure levels characterizations of the workplace are not sufficiently complete to provide data necessary (a) to establish complete contributory emissions, or (b) to establish accurately the levels of reduction necessary to meet a 200 $\mu g/m^3$ level and a 100 $\mu g/m^3$ level.
>
> (4) The conditions of exposure, and hence the needs for control, vary widely from operation to operation, plant to plant, day to day, hour to hour, and even from employee to employee at the same operation. (Ex. 26, p. 4-10-4-11.)

OSHA's view is that Burton's initial statement is correct; that is, for those employers currently not in compliance, the costs attributable to compliance with existing legal requirements should not be counted as part of the costs attributable to compliance with proposed, future obligations. (See also letter of Dr. Corn, then the Assistant Secretary, to George Becker, expressing the same position, Ex. 64; Tr. 4627-

28.) If a new standard were not promulgated at all, the former costs would be incurred nonetheless. For this reason, OSHA considers capital costs for engineering controls in the DBA report to be considerably overestimated insofar as DBA was unable to differentiate the various incremental costs.

A third form of double counting that may occur when costing controls is inclusion of the costs of process equipment with control equipment. It is particularly difficult to separate these costs when control strategies involve the modification or replacement of existing production equipment, but if the new process configuration increases productivity, to that extent its cost should not be charged to control.

In addition to the double counting of data, the reliability of the data itself is often questionable. This results not from any shortcomings of the contractor in collecting the data, but from deficiencies inherent in the process itself. Given the time and resource limitations in a study of this kind, most of the information must of necessity derive from the industries being regulated with minimal opportunity for the contractor to independently verify what controls would be required and what costs and impacts would be incurred. It is obvious that industry sources would tend to be "generous" when asked to supply cost estimates for studies whose economic conclusions would affect the ultimate severity of a regulation that could adversely affect profits. In the analysis of the primary smelting industry, engineering control cost estimates were provided by the companies to the contractor for six of seven facilities. Only one of these cost estimates was verified by DBA. (Ex. 26, p. 5-12). In the secondary smelting industry, all data from the seven plants studied were company estimates (Ex. 65C, addendum to table 5.15); in the battery segment, the primary data come from the CRA study (which depended in the IHE study) and from 12 plants that transmitted information to DBA (Ex. 26, p. 5-43).

Verification of company estimates was also impossible in many case because estimates, such as those from the 12 battery plants submitted to DBA, were given anonymously (Ex. 26, p. 5-50), or detailed information needed to verify the overrall estimate was not supplied. This was primarily because of the companies' desire to keep the information confidential for competitive reasons or because companies believed disclosure would result in compliance activities (Tr. 748; Ex. 65A, p. 4; Ex. 26, pp. 5-99, 5-109).

Further overestimation of cost results from CRA's and DBA's generalization of firms' tax treatment of OSHA-related expenditures. If accelerated depreciation can be used, (*11*) tax savings will occur which will, in part, offset the cost of controls. Another tax benefit that was not considered, one which will lower costs by creating tax savings, is the investment tax credit. (Tr. 1026-27; 4629)

For the five major industries studied, the DBA report contained three categories of estimated costs—low, best, and high—in order to account for errors and to bound the magnitude of costs. In the primary smelter industry where estimates for each plant were obtained, the "best" estimate for the industry is the aggregate of estimates for each plant (Tr. 826-828). The "low" estimate was based on the lowest per employee and per ton of production cost found in the industry multipled by the total number of employees and tons of production; likewise, the "high" estimate was based in the highest per employee and per ton of production cost. In the other four industries, the same methods were used to obtain high and low estimates, but since estimates were not obtained for each plant, the best estimate was calculated by using the geometric mean of the costs per employee and per ton for each plant in the sample. (See, e.g., Ex. 26, p. 5-40).

The high and low estimates are not considered to be especially meaningful in estimating actual costs, particularly in the primary smelting industry where the best estimate is the total of individual estimates for each plant in the industry. (Tr. 829) In the pigment industry, the disparity between high and low is so great as to render the figures meaningless as a measure of the cost of meeting the proposed standard. (*12*) Because of double counting, DBA's "best" estimate is considered by OSHA to be the highest actual cost that compliance with the proposed standard would yield.

The CRA report, submitted by counsel for LIA, assesses the economic impact of the proposed standard on the primary smelting, secondary smelting, and storage battery industries. When different methods of calculation and presentation are accounted for, the DBA and CRA cost estimates are essentially the same. (*13*) This is due to the fact that the cost data comprising their estimates originated from the same sources. CRA obtained company estimates from each of the seven plants in the primary smelter segment as did DBA (DBA independently estimated one plant's costs). CRA calculated costs in the secondary smelter industry by extrapolating from eight plant estimates, seven of which were identical to the seven DBA had used (Compare Ex. 127, Exec. Summ., p. 25, table 5 with Ex. 26, p. 5-30, table 5.15). And in the storage battery segment, the cost data are based on the same IHE study of 12 plants used by DBA. (Ex. 127, Exec. Summ., pp. 31-32).

It is apparent from the fact that each industry's cost totals are similar in both the DBA and CRA reports that CRA engaged in the same forms of "double counting" as DBA in its data collection. In fact, CRA stated that "costs of compliance are the incremental costs of improving air-lead concentrations from current levels to 100 $\mu g/m^3$ * * *" (Ex. 127, p. 2-38) Mr. Wise of CRA explained that incremental cost determinations were irrelevant because "we were not involved in doing a cost/benefit type of analysis comparing marginal cost to marginal benefit. Our focus was on what the impact on industry structure would be in trying to attempt to comply with the proposed standard." (Tr. 3343-44) Additionally, in summarizing market impacts on primary producers, CRA's conclusions are based not only on the $13.2 million resulting from the standard, but on "other regulatory expenditures presently anticipated in the industry." (Ex. 127, p. 2-35).

The cost calculations in the two studies vary for several reasons even though the same basic cost data were used. In the secondary smelting industry, CRA extrapolated from its sample to the whole industry by using cost per unit of capacity while DBA used cost per unit of production and per employee. In the battery industry, an assumption that companies would mechanize certain manual operations was made by CRA not used by DBA. This resulted in a 12 percent greater estimate in the DBA report for total industry capital costs based on the same original data. (Ex. 127, Exec. Summ., p. 32.) In the primary smelting sector, DBA's figures are higher due to the addition of an inflation factor of about 7-8 percent. In one case, the inflation factor was added twice, which accounts for the 18-percent difference in capital costs for Amax Lead Co. (*14*)

With respect to annual costs, CRA's total annualized costs (Ex. 127, Exec. Summ., p. 2, table 1) substantially exceed DBA's costs (Ex. 65B, p. 12 (errata for table 1-1, Ex. 26, p. 1-5)) in the secondary smelter and battery segments because they are expressed as before-tax costs. (*15*) For example, if figures for the secondary smelting industry are examined and allowance for the different method of determining total industry costs as mentioned above are made, the annual recurring costs are virtually the same—$16.7 million for CRA versus $15.8 million for DBA. It is only when CRA annualizes the capital costs and adds them to the annual recurring costs to obtain total annualized costs that the CRA and DBA totals differ so markedly. The

reason is that when DBA presented total annualized costs it adjusted for the corporate tax rate of 48 percent. It is the inclusion of the tax saving attributable to deductions for annual expenses that accounts for the difference in industry estimates. Working backwards, if CRA's total annualized cost of $26.2 million in the secondary smelter industry were decreased by the tax savings, the total would be $13.6 million, substantially in line with DBA's estimate of $14.8 million.

There are however, three inexplicable deviations in the cost estimate used by the two studies. One occurs in the annual recurring cost estimates for the primary smelting industry. DBA's estimate for the seven plants is $12.5 million, and this figure does not include the annual charge to capital (interest and depreciation) that CRA's $13.2 million estimate does. If the annual charge to capital is subtracted from the total annualized costs, CRA's estimates for the same expenses are $5.836 million for the long term and $8.259 million for the short term, both significantly less than DBA's. Since, in almost all cases, the estimates came from the companies there appears to be no reason for the gross disparity between estimates. (*16*) Comparing the cost breakdowns in each report (Ex. 26, tables 5.8-5.14 with Ex. 127, tables 2-15 and 2-16), it is obvious that the information supplied or verified to each researcher was significantly different.

The other deviations occur in the capital cost estimate for the Bunker Hill and St. Joe smelters. For the Bunker Hill smelter, DBA's estimate of $18.4 million for engineering controls exceeds CRA's estimate by $9.2 million. Dr. Burrows of CRA, when questioned about the difference, explained that Bunker Hill supplied him with two engineering reports, one done by an outside consulting firm, the other done internally. CRA used the internal report with the lower estimate, which they felt was more appropriate because its methodology was similar to a study done for the Amax smelter by IHE (Tr. 3371). DBA obviously used the estimate from the external study. Burrows testified that Bunker Hill was not certain which estimate was correct. Although in their written comment to the record (Ex. 3(71), p. 4.) Bunker Hill claimed that an independent consultant estimated engineering control costs to be $17 million (DBA's $18.4 million figure apparently was an adjustment due to inflation), OSHA agrees with CRA's judgment that the $9.2 million estimate is more appropriate.

For the St. Joe smelter, DBA did its own estimate ($6.9 million in 1976 dollars) while CRA accepted the company's estimate of $10.6 million. The accuracy of each cannot be verified since CRA submitted no breakdown of costs. This is further complicated by a comment submitted by St. Joe which said that a respectable engineering firm estimated the cost for engineering controls of the Herculaneum smelter to be $15 million. (Ex. 28(10), p. 4.) The Steelworkers, however, claimed that St. Joe informed its stockholders that the $15 million was for both EPA and OSHA standards. (Ex. 343, p. 174.)

Using the cost estimates they derived, DBA and CRA assessed the economic impacts on each industry studied. The following discussion will proceed industry-by-industry, setting forth the conclusions of each report and OSHA's conclusions. It should be noted that the economic impact analyses are based on a 100 $\mu g/m^3$ level for which OSHA has determined the cost estimates to be substantially overstated and often based on insufficient or unverifiable financial information. (*17*) OSHA believes however that the best available evidence has been pursued and collected.

OSHA did not undertake a formal analysis of cost of compliance with the 50 $\mu g/m^3$ PEL as it did for the proposed 100 $\mu g/m^3$ level. As a result of the rulemaking proceeding, OSHA determined that the proposed level did not provide the adequate worker protection mandated by the act and that a 50 $\mu g/m^3$ PEL would be required. OSHA has concluded that the record contained adequate cost information for most industries. In addition, review of the record revealed that compliance with levels below 100 $\mu g/m^3$ might, in several industries, require extensive technological development for which long periods of implementation time would be required thus precluding meaningful quantification of cost. However, the record was sufficient to predict that compliance within the times given would not result in undue economic hardship on those industries. This qualitative impact analysis is based on the record evidence concerning the financial and technical resources available to the various industries, the certainty of product and factor (production inputs) markets, and the availability of more cost-effective alternative methods of compliance.

b. *Primary lead smelting and refining.* (1) *Costs of compliance.*—The following table compares the cost estimates for the primary lead sector made by DBA and CRA to meet the 100 $\mu g/m^3$ interim level. (Table 2.)

Table 2

	CAPITAL COSTS ($MM)		RECURRING ANNUAL COSTS ($MM)		ANNUAL CHARGE TO CAPITAL		PRE-TAX TOTAL ANNUALIZED COST		AFTER-TAX TOTAL ANNUALIZED COSTS	
	DBA	CRA	DBA	CRA	DBA	CRA	DBA	CRA	DBA	CRA
Bunker Hill	18.400	9.236	2.649	1.233	N.C.	1.439	N.C.	2.672	N.C.	1.389
St. Joe	7.500	10.627	2.211	1.414	N.C.	1.656	N.C.	3.071	N.C.	1.597
Amax	9.540	8.144	1.814	1.365	N.C.	1.269	N.C.	2.634	N.C.	1.370
ASARCO (Total)	20.830	19.247	5.833	1.823	N.C.	2.999	N.C.	4.822	N.C.	2.508
(ASARCO/Omaha)	(4.500)	(4.153)	(1.209)	(0.425)	(N.C.)	(0.647)	(N.C.)	(1.072)	(N.C.)	(0.517)
(ASARCO/East Helena)	(5.900)	(5.499)	(1.444)	(0.491)	(N.C.)	(0.857)	(N.C.)	(1.348)	(N.C.)	(0.701)
(ASARCO/El Paso)	(5.250)	(5.483)	(2.060)	(0.498)	(N.C.)	(0.752)	(N.C.)	(1.250)	(N.C.)	(0.650)
(ASARCO/Glover)	(5.180)	(4.767)	(1.120)	(0.373)	(N.C.)	(0.743)	(N.C.)	(1.116)	(N.C.)	(0.580)
TOTAL	56.270	47.254	12.507	5.836	12.373	7.363	24.88	13.199	12.94	6.864

Source: Ex. 26, Tables 1.1, 5.8-5.14; Ex. 127, Tables 2-15 through 2-19
N.C. = Not calculable from data provided

Given the earlier discussion about the unreliability of cost estimates, OSHA has determined that the upper limit for capital expenditure to meet the 100 $\mu g/m^3$ interim level is in the vicinity of CRA's estimate of $47.2 million (in 1976 dollars) with the better estimate, taking double counting into account, as much as one-third lower, or $32 million.

There is really no accurate way to determine the extent of double counting. It appeared that as much as 30 percent of the cost attributed to compliance at ASARCO's El Paso and East Helena smelters was, in fact, for compliance with air quality (EPA type) regulations (Tr. 4635-38, 4653-54, 4041-43), and one-third of the total $6.9 million estimate for engineering controls at St. Joe's was for baghouse expansion and renovation, a project that is essentially directed toward control of external air pollution and that will provide increased product recovery. (Ex. 65C; Tr. 2071.) Double counting of costs also occurred in DBA's use of ASARCO's estimates for six plants (Ex. 3 (106), App. 7; Tr. 6507) because ASARCO did not eliminate costs to reduce levels to 200 $\mu g/m^3$. Finally, there were instances of double counting of control costs with production costs. Kenneth Nelson, an ASARCO vice president, testified that costs will be offset by increased production at the El Paso smelter (Tr. 4654). Given the above, reducing the total capital cost estimate for the 100 $\mu g/m^3$ interim level by one-third for all forms of double counting is reasonable.

Total annualized cost estimates are likewise overestimated since a major component is the annual charge to capital. Since the capital costs are estimated to be lower, so then should the total annualized costs. Likewise, a decreased estimate in capital costs should yield a decreased estimate in annual recurring costs, primarily in the area of operating and maintenance of engineering controls. Operating and maintenance cost are substantial—as much as 10 percent of the capital costs and 70 percent of annual recurring costs (e.g., Ex. 127, table 2-16, where total annualized costs minus annual charge to capital equal $5.836 million, and annual operating costs equal $4.050 million or about 70 percent).

Since OSHA's revised estimate for capital costs is between $32 million and $47 million, the operating costs, reduced proportionally, should be between $2.713 million and $4.050 million. Adding the revised estimated annual cost of capital (between $4.986 million and $7.363 million)(*18*) and other annually recurring costs ($4.228),(*19*) the revised estimate for pretax, total annualized cost at the 100 $\mu g/m^3$ level for the primary sector is estimated by OSHA to be between $11.927 million and $15.641 million.

After-tax cost, figured on the corporate rate of 48 percent, should then be between $6.202 million and $8.133 million. The use of accelerated depreciation on capital property and applicable tax credits would further reduce the industry's annual costs.

The result is that the total annualized cost to the industry for the 100 $\mu g/m^3$ level, based on its total 1975 production,(*20*) would be approximately 0.4 cent to 0.6 cent per pound. On a per employee basis,(*21*) it is $2,030 to $2,662.

Compliance costs for the PEL for this sector cannot be estimated because of the multiple, potential compliance strategies available to firms within the 10-year implementation schedule. The economic implications of a 10-year planning horizon are however, assessed in the impact section below.

b. *Factors affecting the impact of compliance cost on primary smelters and refineries.* The impact on the primary production sector of the lead industry will depend: (1) On the structural characteristics of the industry and its market; (2) on the ability of firms to shift costs forward into price, or backward onto the factors of production; (3) on the distribution of cost among firms in the industry; and (4) on the financial strength of the affected firms.

(1) The primary production sector consists of four firms which operate seven plants, four of them belonging to ASARCO. St. Joe, Amax, and Bunker Hill do both smelting and refining at their plants. Of the four ASARCO plants, only Glover (Missouri) has smelting and refining capacity. East Helena (Montana) and El Paso (Texas) are smelters only; Omaha (Nebraska) is a refinery. St. Joe and Amax are located in the Missouri new lead belt, Bunker Hill operates in Idaho with non-Missouri lead, and ASARCO is in both segments of the industry. The non-Missouri and foreign ores have a lower lead content and contain higher percentages of zinc, copper, silver, and other minerals. They can be processed only in plants designed to handle concentrates of this sort and therefore they go to El Paso, East Helena, or Bunker Hill for smelting and do not go to Glover, St. Joe, or Amax. St. Joe operates the largest smelter-refinery and supplies almost all its ores from its own mines. (Ex. 127, p. 2-27.) The others rely partly on custom smelting performed for other suppliers, although Amax's Buick mine produces more than the smelter/refinery capacity (Ex. 127, pp. 2-27 through 2-28). Bunker Hill owns several mines which supply about 20 percent of its capacity; the remaining capacity is custom smelted from western ores.

ASARCO is the most diversified firm in the industry and is both horizontally and vertically integrated. It owns several mines which, along with the zinc, copper, and secondary lead smelters it owns, provide 20 percent of the material entering its two western smelters. These in turn feed the Omaha refinery. The other 80 percent is custom smelted, while Glover's production is almost entirely on a custom basis from Missouri mines (Ex. 127, p. 2-29).

The market in which the primary lead firms sell their product is supplied from two other important sources as well.(*22*) Secondary smelters supply 45 percent of the market (Ex. 26, p. 6-9; Ex. 127, p. —), and imports account for 5-9 percent (Ex. 26, p. 6-7, 6-9). The primary sector demonstrates an oligopolistic supply pattern (Ex. 26, p. 6-7), characterized by price leadership (Ex. 26, p. 6-10) and restricted entry of new firms (Ex. 26, pp. 6-7, 6-10; Ex. 127). The total market is much more competitive, however, due to the presence of the competitive secondary and foreign suppliers whose products are almost completely undifferentiated from the primary producers.(*23*) The market structure is such that it sharply limits the market power of the primary producers in the long run. Foreign supplies, especially, may increase substantially in the long run if domestic prices exceed the world price, as measured by the London Metal Exchange (LME) price, by some margin currently estimated to be 2-3 cents, the estimated cost of transportation and tariffs. (Ex. 26, p. 6-7; Ex. 127, Exec. Summ., p. 17.)

The supply of ores and ore concentrates is also diverse. Some domestic mines are owned by smelters, some are under long term contracts, and others are independent. Supplies also come from a number of foreign sources and some domestic ore goes to foreign buyers. Western lead mines have been declining in recent years and many of those remaining are marginal. Missouri mines are significantly more profitable. (Ex. 127, pp. 2-1 through 2-19.)

(2) The ability of the firms to shift costs either forward or backward depends on the elasticities of supply and demand in the pertinent markets. The demand for lead is derived from the demand for the products in which it is used. The most important of these is storage batteries, which accounts for approximately 48 percent of lead consumption in the United States. The demand for batteries is relatively inelastic, being largely a function of the number of automobiles in use. Since there are presently no substitutes for lead in the production of batteries, the

demand for lead in this use tends also to be relatively inelastic.

The next most important use for lead is in gasoline, but this market is declining as unleaded gasoline is inceasingly used. The long run significance of this use for lead is therefore steadily declining.

There is a very large variety of other uses for lead, none of which accounts for more than approximately 6 percent of total consumption. Analysis of the elasticity of the total demand for lead on the basis of a study of the markets for lead products is thus not feasible. CRA and DBA relied therefore, on statistical studies of historical data to estimate the elasticity of demand for lead.

"There seems to be little question but that in the very short run the elasticity of demand for lead is quite close to zero." This is a conclusion shared by DBA and CRA (Ex. 26, p. 6-20). As to periods greater than a year, there is conflicting evidence on the price elasticity of lead demand. CRA cited its studies over a 10-year period that indicated that this demand was relatively inelastic (Ex. 127, p. 2-50). It then reported a study by J. M. Heineke that estimated the demand for lead to be extremely elastic in the long run. The CRA study apparently accepted Heineke's estimate as being correct and so, in turn, did DBA, although the reasoning behind this acceptance is not clear. The CRA report states that previous CRA estimates were based on annual data, while Heineke's work used monthly data. No explanation was offered of why monthly data would provide more accurate and substantially different estimates of long run elasticity.

Under questioning, Dr. Burrows of CRA repudiated Heineke's work (Tr. 3378). He did point out that previous CRA studies had been aimed at determining short run elasticity for lead demand and that those studies had not taken into account the effects of price increases on market growth and new uses. Nevertheless, he seemed to say that, in his opinion, the demand for lead was relatively inelastic.

OSHA has independently assessed the validity of Heineke's work because the determination of the long-term elasticity of demand is crucial to the economic analysis of the primary lead industry. OSHA has concluded that Heineke's study is not valid and that CRA's initial observation that long-term demand for lead in the United States is relatively price inelastic is correct.

Heineke's study is a mathematical multivariate analysis relating the quantity of the lead ingots consumed to the average monthly price on the New York Metal Market, national income as measured by the Index of Industrial Production—Manufacturing, and a "disturbance term" which is a "stationary stochastic term with zero expectations." The data used cover the period 1948-1965. Since the disturbance term is essentially random, his formulation purports to explain consumption of lead ingots in the United States on the basis of changes in national income and domestic price. Time lags are explained by the contracting procedures common to the industry (causing several month's lag) and technological substitution away from lead (explaining long run elasticity). He assumes, therefore, that changes in domestic consumption not explained by changes in the level of industrial activity are explained largely by price changes. Such an assumption would be essentially correct if the market demand was essentially the result of business and consumer decisions in a reasonably competitive and free market. However, it ignores several other influences at work in the market during the period (1948-1965):

a. The Federal Government maintained a national security stockpile of lead, purchasing or selling lead in the market at various times on the basis of policy decisions not determined by considerations of the economics of materials substitution in industrial production relative to the price of lead.

b. There are two market prices of significance to U.S. consumption, the New York Metal Market price (used by Heineke) and the LME price, which is the price of imported lead.

c. Quotas were in force in some of the years covered, limiting imports and contributing to shortage of lead in some years, thus limiting consumption.

d. Consumption changed in some years in anticipation of price changes rather than being lagged after price changes.

e. The price of substitute materials, notably plastics, declined independently of the price or supply of lead, causing some users to substitute other materials for lead.

These other market forces are not randomly distributed and do not average out. They could account for a significant portion of the price elasticity of demand as measured by Heineke. Furthermore, they are not present in the same way in the present market and there is no reason to expect that they will affect the future as they did in the period 1948-1965. CRA's earlier studies of the lead market cited by DBA (Ex. 26, p. 6-5, n.1), appear to be much more complete and of greater validity. They indicate that total U.S. demand for lead is relatively inelastic. OSHA has concluded that the demand for lead is inelastic with respect to any reasonable range of prices in the short run and relatively price inelastic in the long run.

The CRA study explains the rather steady realtionship between the LME and U.S. price in terms of a very high long run elasticity of supply. They argue that this is largely attributable to the ability of foreign producers to increase their supplies in the U.S. market whenever the U.S. price rises considerably more than 2-3 cents above the LME price. In the long run, it was asserted, this would prevent domestic producers from raising prices to recoup the costs of compliance with the lead standard. Several factors could operate to minimize the possibility of foreign imports, especially in response to a small increase in the differential.

World demand has been increasing steadily. In the 10-year period between 1965 and 1974 world consumption of refined lead increased by a third from 3,182,200 to 4,350,300 metric tons annually. (Ex. 127, Table 1-1.) In part this reflects the growth of the automobile as a primary means of transportation in the developed countries. Also significant is the increasing demand for lead for other industrial uses as demonstrated by the rapid growth of the market in Africa (66 percent) and Asia (25 percent) since 1968. (Ex. 127, p. 1-2), CRA asserts that lead has been produced on a constant or declining cost basis and assumes such will be the case in the future. In order to make this argument, they constructed a graphic representation of U.S. and LME lead prices over time. (Ex. 127, Exec. Summ., p. 18.) They deflated all prices by the Wholesale Price Index for all commodities (1967=100). After this adjustment for inflation, the long-term movement of these prices appears to tend toward a rather consistent price range. CRA does not offer an econometric analysis of this price behavior. They conclude that it is explained by long run production at constant costs. (Ex. 127 Exec. Summ., p. 17.)

Like the Heineke study, this analysis fails to account for the extra-market forces which shift supply (as well as demand) curves over time. CRA assumes that discovery of new deposits is functionally related to changes in the price of lead. It would appear that such discoveries have played a major role in lowering cost of production, but there is no evidence that the new mines were brought on-line in response to rising prices. Absent the shifts in supply which result from new discoveries, there is every reason to believe that the normal case in which production is subject to increasing costs does prevail in the primary lead industry. In discussing the problems of the non-Missouri mines, CRA states,

"Rising costs and lower yields are responsible for much of this decline. Many of the mines which date back to early years of this century have been forced to seek ore at much deeper levels with corresponding increases in costs." (Ex. 127, p. 1-29.) It is reasonable to assume that this condition of increasing costs applies to all mines in the long run and that only the discovery of new sources will lower production costs. Such discoveries, like the new Missouri lead belt, are unpredictable.

CRA argues that the constant cost case is likely to continue in the future because newer mines are likely to open in countries with lower labor costs. (Ex. 127, p. 1-20.) In fact, a principal reason some decline in production costs is associated with new discoveries is the fact that they may be mined by more capital intensive measures. The new Missouri lead belt produces 80 percent of U.S. lead ore with 30 percent of U.S. lead miners. (Ex. 127, p. 2-13.)

OSHA has concluded that lead is produced under conditions of increasing costs. The increase in foreign production in response to changes in world demand will raise the international costs of production. The impact of the cost increase will depend on the magnitude and direction of extra-market forces.

If smelter costs abroad are raised because other governments follow United States environmental and occupational health regulations, then world prices will rise even more sharply and the competitive position of the U.S. lead industry will improve. Increased costs due to the upgrading of foreign health and environmental standards are likely. Dr. Michael Williams, a British occupational physician, stated that "the United States has pioneered the use of sensible industrial standards, and (has) great influence on the practice in other countries." (Ex. 234(6), p. 93.) CRA does not foresee the same "types" of costs being incurred by foreign producers but agrees that to the extent foreign producers of refined lead incur costs from upgraded standards the price of their product would similarly have to be increased. (Tr. 3287.)

The U.S. may elect to protect the domestic market from competition with foreign lead that is produced with an unfair advantage due to lack of concern for public and occupational health. Under this condition the domestic industry would be able to pass all such costs through in the form of price increases without cutting output.

There is also a possibility of a cartel artificially raising world prices thereby allowing U.S. prices to rise to maintain the 2-3 cent differential. Several industry observers maintain that this is presently occuring, but CRA, acknowledging that it might have been a factor in the 1975 period of high prices, discounts the success of a cartel in permanently supporting prices above competitive levels. (Ex. 127, pp. 2-66 to 2-69.)

When costs are passed back to the mines in the form of reduced prices offered for lead ores and concentrates, the question of the elasticity of these supplies becomes important. The limits on such a backward shift were described by CRA as two-fold. First, some mines were of such marginal profitability that even small backward shifts would doom those operations to closure. CRA assessed profitability of these mines using metal prices far below today's market. (*24*) (Ex. 127, p. 2C-3.) Since prices for lead have risen dramatically since the CRA study, none of these mines could now be considered marginal.

It should also be noted that increasing smelter charges to meet environmental costs have precedent within the nonferrous industry. The ASARCO custom copper smelter at Tacoma, Washington, uses such a method to cover some compliance costs associated with air quality standards. (*25*)

The second obstacle noted by CRA was that foreign smelters might be able to bid the ores away from domestic firms. For such a case to hold true (even if transportation cost remain within the 1-2 cents per pound range), it must be assumed that relative costs (excluding incremental OSHA costs) remain constant between foreign and domestic smelters—an unlikely assumption given the potential increased costs for foreign suppliers discussed earlier. Further, CRA assumes that the differential in bids sufficient to produce a shift to foreign smelters is equal to the transportation costs. They attribute no price to the additional risk involved in dependence on foreign smelters. It would seem that U.S. mines would pay some premium to maintain the greater economic stability inherent in a domestic smelting industry.

(3) For the reasons mentioned earlier, only limited data are available on the financial condition of individual firms and plants. For primary smelters, the data relevant to an assessment of the impact of compliance costs were available only for St. Joe and Bunker Hill. General company data are presented in the record for Amax and ASARCO, but not for the lead smelting and refining operations within the company totals.

From the data available, it appears that St. Joe Minerals Co. is the financially strongest of the four primary smelting companies. Its average annual rate of return (earnings before taxes) on total assets over the past 10 years was calculated by DBA to be at least 1½ times that of the second strongest, Gulf Resources and Chemical. ASARCO and Amax were third and fourth, respectively, in this category. (Ex. 26, p. 6-13.) St. Joe's Mineral Corp. also has the lowest debt to equity ratio of the four companies, a measure of the company's ability to finance capital expenditures. In 1975, St. Joe's was 0.540 (Ex. 26, p. 6-12), while ASARCO's was 0.740, Amax's 0.818, and Gulf Resource's 1.342. (Ex. 127, table 2-14.) It should be noted however that Gulf Resources' ratio has steadily decreased by one-half since 1971 while ASARCO's and St. Joe's have more than doubled in the same period. A trend for Amax was not determinable.

CRA presented pretax income data for the Bunker Hill Co. and estimated that 50 percent was attributable to the lead operation. The company's income has varied substantially from year to year since 1970, but the average (in 1976 dollars) is $10.664 million for total profits and about $5.332 million for lead operations. (Ex. 127, table 2-24.) In 1976, Bunker Hill's profits were $6.1 million, 12 percent less than the previous year. (Ex. 343, p. 173.) Income data was also presented by CRA for St. Joe's Herculaneum smelter, but only for 1973 and 1974. (Ex. 127, table 2-25.) Net pretax income in 1973 was $5.357 million and $7.170 million in 1974.

(2) *Impact on the industry.* The characteristics of the primary lead smelting and refining firms and their markets, discussed above, provide the basis for the impact analyses shown in the record. DBA's analysis assumed essentially that the costs of compliance with the 100 $\mu g/m^3$ standard must be borne by the primary smelter companies and estimated the impact these costs would have on each firm's financial position. CRA assumed some partial shifting of costs, primarily backward to the mining companies supplying the primary lead smelters. The primary emphasis of the CRA study was on the changes that might occur in the market and industry structure. OSHA's conclusions are as follows:

(1) *The primary smelting companies will probably be able to raise the price of refined lead as much as 1 cent per pound in order to pass compliance costs to consumers of its product. This increase will be sufficient to cover the incremental costs of meeting the 100 $\mu g/m^3$ interim level.* DBA and CRA concluded that it would not be possible for firms to increase the price of lead. CRA attributes this to the high elasticity of foreign supply (Ex. 127, pp. 2-51 to 2-56), and DBA concludes that high elasticity of the demand for lead will have the same effect (Ex. 26,

p. 6-25). CRA's and DBA's conclusion is somewhat doubtful for several reasons. First, given OSHA's revision of estimated costs to the industry, the necessary price increase would be smaller than predicted by CRA and DBA. Second, the demand for lead in the long run, as well as in the short run, will most likely be price inelastic, and finally, the foreign supply of refined lead will probably be relatively inelastic in the short run, the significant period in which domestic producers could recapture a substantial portion of compliance costs. As to the long run, several factors can and may operate to make the foreign response to changes in U.S. price indeterminate. Given the revised, total annualized cost estimates for the primary sector of between $11.927 million (best estimate) and $15.641 million (high estimate), it appears that the price of lead need only be increased by 0.8 cent to 1.1 cents per pound to cover the cost of achieving the interim level. (*26*) This is based on industry production figures of 1975. (*27*)

The demand for lead will probably be substantially price inelastic in the long run. CRA's studies over the past 10 years, Dr. Burrows repudiation of Heineke's work, and OSHA's evaluation of Heineke's conclusions support this. Therefore, demand factors should not play a significant role in the industry's pricing decisions. With respect to supply, the factors affecting the long-run behavior of firms are numerous.

The increasing cost of producing lead (absent new discoveries) may impact on foreign producers sufficiently in the short run to reduce the incentive to shift production to the U.S. market. Foreign governments may follow the U.S. lead and compel similar environmental and occupational health constraints on their industry. Trade barriers or trade agreements limiting foreign imports may be adopted. (*28*)

These factors affecting supply are highly speculative and no firm conclusions can be drawn other than that foreign supply is probably price inelastic in the short run, thereby allowing a short-run price increase, and possibly inelastic in the long run if one or more of several possible factors materialize.

At least one major producer, Amax, is confident that the industry will be able to pass costs forward. They stated that the costs of the standard "would certainly add to the price of our final product which in turn will have to be passed on to the consumer." (Ex. 3(67), p. 5.)

(2) *Compliance costs can, in part, be shifted backward to suppliers of ore.* CRA concluded that costs could be shifted, in part, backward onto suppliers through a reduction in the price paid for ores and concentrates (Ex. 127, Exec. Summ., pp. 8-10). This would be an accounting transaction to St. Joe, which is supplied by mines owned by the same company. DBA did not evaluate backward shifting of costs. The extent to which this could be accomplished minimizes the cost impact on the primary producers. OSHA has concluded that the limits on the backward shifting of costs are not as severe as indicated in the CRA analysis. The increasing price of lead has improved the marginal conditions attributed to several mines by CRA. Further, the incentive to ship abroad depends on foreign costs maintaining their present relationship to U.S. costs excluding OSHA impacts, a questionable assumption. Finally, OSHA believes that the differential can rise somewhat above the cost of transporting the ore to foreign smelters because of the obvious advantages of adequate U.S. smelting and refining capacity to the domestic mines.

(3) *The industry has the ability to pass costs forward or backward sufficient not only to recover the cost of the 100 $\mu g/m^3$ interim level, but to assure that any likely cost associated with the PEL will not jeopardize long-run profitability.* In the assessment of market power, OSHA disagrees with the conclusion in the CRA report. The difference is most apparent in the analyses of the non-Missouri operations of ASARCO. (Ex. 127, pp. 2-79 through 2-84.) CRA calculates the annual compliance cost of the proposed standard to these operations at $3.7 million or approximately 1 cent per pound of refined lead. They are aware that ASARCO had announced its intention to spend $55.2 million at El Paso and $32.2 million at East Helena to control air quality problems associated with lead productions. These capital costs, when annualized, produce an additional 6.2 cents per pound expense to the company, almost one-third of the market price of lead used in the analysis. The CRA cost passback analysis limits ASARCO's recovery from the mines to a maximum of 2 cents per pound. Their elasticity analyses preclude any long-run price increase. They conclude that the incremental OSHA costs seriously jeopardize continuing operation of the ASARCO western smelters and refinery since the air quality controls would seem to cost ASARCO 4 cents per pound of profit. They attribute ASARCO's willingness to continue in business to the externalities of custom smelters which extract "metals such as silver, cadmium, bismuth and selenium as well as the slag processing which improves the flexibility of the ASARCO system." (Ex. 127, p. 2-84.) CRA makes no attempt to document this claim. It is obvious that ASARCO was willing to risk an enormous sum of money. Either they anticipated an ability to recover that long-run expense in terms of price increases or cost passbacks or some combination of both.

OSHA concludes that the segment of the primary industry claimed to be in the most financial trouble, the western custom smelters, have sufficient market power to survive enormous increases in costs. The money scheduled to be spent on air quality problems may alleviate some occupational lead problems as well. More important, it is the most impressive possible statement of the perception of the long-run viability of the industry by the largest producer. (*29*)

The 10-year period set forth in the methods of compliance section is based primarily on technological factors. This time should be sufficient for any firm to completely rebuild an existing smelter (Ex. 3(103), p. 5) or to construct new capacity.

This extended compliance period also assures economic viability of the PEL. Production efficiencies may arise from new processes, such as hydrometallurgy, sufficient to offset EPA and OSHA cost. Retrofit technology may be refined that will effect control greater than now envisioned for existing equipment and thus lower long-run costs of compliance. DBA stated that "we can expect to see new, innovative and cost-effective compliance methods being introduced as a result of enforcement of the standard." (Ex. 26, p. 2-16.)

The 10-year compliance time constitutes a planning horizon sufficient to allow all firms maximum flexibility in capital planning. OSHA believes the long-run outlook for the industry is favorable and there exists some combination of engineering controls and work practices, including administrative controls, which will permit all four firms to remain in the market. Because the economic and environmental conditions of the western smelters vary widely from those in Missouri and among themselves, OSHA has established a time frame designed to maximize the technological and economic options for the industry. This compliance period is sufficient to allow each firm the opportunity to assess the likely state of the market and to raise the capital necessary for conversions required by air and water quality standards, other OSHA standards, and the 50 $\mu g/m^3$ lead standard. OSHA has concluded that this flexibility is necessary for achieving the most cost effective solution for the industry consistent with necessary worker protection.

(4) *If primary smelting firms were forced to absorb all the costs of compliance in the short run, they would nevertheless remain profitable and com-*

petitive. To the extent that increased costs can not be passed back to suppliers or forward to consumers, the primary lead producers must absorb them internally, i.e., pay for them out of profits. From the record evidence as a whole, it appears that each of the affected firms can shift or absorb compliance costs of the interim level and remain profitable and competitive. Of all the primary producers, only Bunker Hill's profitability is in question and the cost impact should be such that OSHA costs alone would not threaten the company's economic viability.

One method for assessing this is to attempt to predict the impact on a firm vis-a-vis certain numerical indices of the firms' financial condition. The problems with this approach are in choosing the most important and relevant indices and in obtaining information that is not ordinarily available.

DBA evaluated the impact on each firm by estimating the impact on the rate of annual return on total assets and on the price of a share of common stock. (Ex. 26, pp. 6-11 through 6-16.) CRA compared profit and debt to equity ratios (Ex. 127, p. 2-33 through 2-35; 2-70 through 2-86) while other parties suggested looking at net profits (Ex. 343, pp. 170-174.) Each of these indices has validity when used to compare the relative strength of firms, but if looked at absolutely can be misleading.

DBA's conclusions are misleading because its calculations are based upon cost estimates that are significantly overstated. For example, the cost estimates it used for the Bunker Hill smelter show the impact on Gulf Resources to be a reduction in the rate of return on total assets from 13.34 percent to 6.28 percent. (Ex. 26, p. 6-13.) This, however, is based on compliance costs at least double those which OSHA has determined to be reasonable. Similarly, the percentage decrements for the other firms, St. Joe (1.56 percent), ASARCO (1 percent), and Amax (0.3 percent) would be even smaller if adjustments were made using the revised cost estimates. The same is true in the percentage decrements predicted for the firm's common shares. The result is that DBA's conclusion that Bunker Hill would have to shoulder an inordinate compliance burden compared to the other firms is weakened. Gulf Resources' return on assets will decrease more than the other firms', but it will still have a rate higher than ASARCO and Amax.

DBA was not able to obtain disaggregated financial information on the Bunker Hill Co. or on their or other companies' lead operations, so its analysis was of necessity based on the parent company's financial condition. Its conclusions, then, give a measure of relative financial impact on each of the parent firms in the primary sector, but are not useful in determining the effect on the industry in terms of competitive structure. Other than stating that the firms' profitability would be reduced, DBA was not able to determine whether any primary producer would curtail its lead operation. (Ex. 26, p. 6-26.)

The Steelworkers asserted that each of the four firms could pay for all the capital improvements estimated by CRA out of 1976 profits alone. (Ex. 343, p. 172.) Their calculations showed that compliance costs as a percentage of 1976 profits were as follows:

Company	Capital costs (percent)	Annual costs (percent)
ASARCO	45.6	11.3
Amax	5.4	1.7
St. Joe	15.4	4.5
Gulf Resources	54.3	15.9

From the firms submitting data on their lead operations alone, St. Joe's return on sales was claimed by the Steelworkers to be 34.1 percent, indicating exceptional profitability. Bunker Hill's decline in profitability in 1976 to $6.1 million was attributed to a decrease in lead production 20 percent below capacity to meet state environmental standards and a "continuing softness in the zinc and by-products markets." (Ex. 343, p. 173.) The Steelworkers noted that the company expected its air pollution problems to have been abated by mid 1977, enabling them to return to near capacity production.

CRA evaluated each firm's profitability and their ability to shift costs back to suppliers of ore. They concluded that Bunker Hill, with the heaviest costs of compliance and little chance to shift cost back to suppliers, might prove uneconomical for Gulf Resources to continue to operate. This analysis is somewhat misleading because it fails to isolate effects attributable only to the cost of the proposed OSHA lead standard and bases its conclusions on the combined OSHA/EPA-type costs equivalent to 1.54 cents per pound of refined lead produced.*(30)*

Initially, production at Bunker Hill is expected to increase (Ex. 343, p. 173), thereby lowering the cost per pound, but more important, the cost attributable to the OSHA standard is less than 1 cent per pound (0.95 cent by CRA's calculations). This is only 0.23 cent in excess of the 0.72 cent per pound that CRA estimates Bunker Hill can pass back to the mines under the best conditions. (Ex. 127, p. 2-73.) Under the worst conditions, the differences would be 0.8 cent. (Ex. 127, p. 2-74.) This means that OSHA compliance costs at 90 percent of operating capacity (126,000 short tons) will be between $579,000 and $2.016 million annually.

Looking then at profitability, CRA concluded that if Bunker Hill was forced to absorb between $2.3 to $3.9 million, the consequences would be "severe." However, as pointed out above, Bunker Hill's 1975 profit was $6.2 million. Its average profit between 1970 and 1975 was $10.664 million overall and about $5.332 million from lead operations. Absorbing costs of $0.579 to $2.016 million will cut into profits, but those costs are only 5 percent to 19 percent of the firm's average profits. This mitigates CRA's conclusion; in fact, CRA states that if Bunker Hill had only to absorb a cost of $1.54 million (the EPA-type costs) Bunker Hill's profitability would not be jeopardized. (Ex. 127, p. 2-76.)

CRA found that St. Joe could not shift its compliance costs and would have to absorb $3.07 million of added annual costs (pre-tax), although it could continue to operate profitably. Similarly, Amac was expected to continue to operate, although absorbing approximately $1.25 million of annual cost (pre-tax), since it would be able to shift only a part of its compliance costs.

The impact on ASARCO, according to CRA's analysis, would be mixed. Its non-Missouri plants would be able to shift only a part of their compliance costs. However, if Bunker Hill were to close, ASARCO would be the only remaining processor of non-Missouri ores and this would enable it to shift a larger share of its compliance costs. It would still have to absorb substantial costs, however, at these plants. The Glover plant, on the other hand, would have the lowest compliance cost of all primary smelters and would be able to shift all its costs. This plant could even increase its profit if the Bunker Hill plant closed and Glover expanded its production.

(5) If compliance costs reduced the profitability of Bunker Hill to a point where Gulf Resources decided to close its lead operations, the competitive structure of the primary sector would be largely unaffected. DBA stated it this way (Ex. 26, p. 6-26):

> If one or more producers of primary refined lead should be forced to shut down lead refining operations, concentration in primary refined lead production could increase substantially. Such an event would no doubt facilitate cooperative behavior among the surviving primary lead producers. However, this probably would not affect significantly the nature of competition in refined lead.
>
> The degree of concentration in primary refined lead production is already potentially high enough to achieve a joint monopolistic result as a consequence of the mutually recognized interdependence of the four large producers. This could occur without

the necessity of resorting to overtly collusive conduct.

That this result is not presently attained is due to forces being exerted from outside the primary lead segment of the market, viz., from secondary lead, refined lead imports, and the threat of entry. These forces would still be operating no matter what the degree of concentration in primary refined lead. Thus the competitive situation probably would not be significantly affected even if the imposition of the proposed occupational lead exposure standard leads to a reduction of the number of firms engaged in primary lead production.

(c) *Secondary lead producers.* (1) *Costs of Compliance.*—DBA's and CRA's estimate for compliance costs (in millions of dollars) for the 100 μg/m³ level in this industry are roughly similar as indicated below:

	DBA	CRA
Total capital costs	51.1	60.6
Recurring annual costs	15.8	16.8
Annual charge to capital	12.7	9.4
Total pre-tax annualized costs	28.5	26.2
Total after-tax annualized costs	14.8	13.6

As discussed earlier, the differences between the two reports' capital cost estimates, since they are based on almost the same sample, are due to the method of extrapolating costs from the sample to the entire industry. DBA's method, based on cost per unit of production, rather than unit of capacity as CRA did, and per employee gives a more realistic estimate because actual production costs are more relevant to the assessment of economic impact than costs based upon capacity which are potential production costs. The difference in total annualized costs is due to the difference in the annual charge to capital.

Actual costs attributable to the proposed standard will be somewhat lower both because of the double counting inherent in the data collection process and the favorable tax benefits available to the industry. The nature of double counting in cost estimates for this industry is the failure to separate costs of compliance with the present standard from costs of compliance with the new standard. Most secondary smelters are not now in compliance with the present standard (e.g., Ex. 26, p. 5-36) and would incur substantial costs to achieve compliance. While this incremental cost was not assessed, a rough estimate can be made by looking at a recent cost estimate for a secondary smelter to comply with the 200 μg/m³ standard. In 1976, IHE estimated the cost for a secondary smelter producing 10,000 tons per year to comply with the 200 μg/m³ standard.(*31*) Capital costs were given as $324,200 and annual operating and maintenance costs were $21,650. This estimate is equivalent to $32.40 per ton of production and almost 50 percent of the costs per ton estimated by CRA and DBA for seven typical plants to comply with the 100 μg/m³ standard. These costs to reach 200 μg/m³ may or may not be typical of the secondary smelting industry. However, with the IHE three plant study in the battery industry (Ex. 138C) and similar double counting in the primary smelter industry, OSHA has concluded that DBA's estimate should be reduced by at least one-third. The resulting best estimates if all costs are reduced proportionally in the secondary smelting industry will be (in millions of dollars):

	Best estimate	High estimate
Total capital costs	34.1	51.1
Recurring annual costs	10.4	15.8
Annual charge to capital	8.4	12.7
Total pre-tax annualized costs	18.9	28.5
Total after-tax annualized costs	9.8	14.8

The total annualized cost is thus equivalent to 1.3 cents per pound of production in 1975.(*33*)

The cost of attaining the PEL of 50 μg/m³ cannot be precisely ascertained because the industry faces several options for longrun compliance. However, an upper limit (the cost of completely rebuilding the industry with the latest available technology) is determinate. To completely rebuild with the Bergsoe process would cost approximately $90.6 million excluding land costs.(*34*) Bergsoe estimated that the system produces sufficient profit for complete capital recovery in a 2-3 year period. (Tr 5192.) Control costs are more than offset by production efficiencies discussed in detail below. While such costs cannot be precisely separated from production costs, it is clear that they should be well below the costs needed to retrofit most existing equipment to the PEL.

(2) *Impact on secondary smelters.* Secondary lead producers are quite highly competitive. There are many firms, some of which are subsidiaries of primary producers (e.g. ASARCO) and some related to battery producers (e.g. General Battery). Although concentration has been increasing (Ex. 26, pp. 6-6, 6-7), production within the industry is still not highly concentrated, primarily as a result of low entry barriers. Sources of scrap can be easily acquired and initial capital requirements are low. (Ex. 127, p. 1-29.) As a result, secondary producers have little control over prices, even in the short run, essentially following the market. (Ex. 26, p. 6-10.) They will be able to shift compliance costs forward onto product prices only if primary producers raise prices, and it appears that under certain conditions primary producers may be able to do so. OSHA has determined that the DBA impact assessment is faulty in two respects. First, DBA did not consider the possibility that primary smelters might be able to pass through 1¢ per pound of the compliance costs and secondary smelters would benefit accordingly. More importantly, DBA did not analyze the ability of secondary firms to pass cost back to scrap dealers. CRA anticipates that the average compliance cost will be passed back and thus only firms whose costs exceed the average would have to absorb any compliance cost even absent a price rise. CRA concludes that some high cost, marginal firms may cease operations. CRA did not predict serious adverse impact to the industry. (Ex. 127, Exec. Summ., p. 77.)

DBA estimated the impact on two firms, ASARCO and NL Industries, for which it had specific financial data. The analysis indicated that ASARCO's rate of return on total assets would decline by 1.53 percent as a result of absorbing compliance costs at its secondary plants. NL Industries would experience a decline of 8.02 percent in its rate of return from 12.7 to 11.7 percent (Ex. 26, p. 6-16.) Financial information was not available from other firms in the industry to enable OSHA to assess the profitability of firms and the cost impact on their continued existence. DBA reported that "all of the participating companies indicated that they would not retrofit some existing equipment but would close some operations because they could not cover the costs, and/or would increase production at some of their least affected facilities." (Ex. 26, p. 5-38.) DBA also expected no major changes in the structure of the industry.

Largely for technical reasons discussed earlier, OSHA has concluded that retrofit engineering controls may require up to three years for installation and, accordingly, allows that much time for the industry to achieve the 100 μg/m³ interim level. For the less efficient producers, particularly those energy savings from the Bergsoe process. Those smelters generate over 50 percent of their energy from the burning of the battery cases. Coupled with energy savings from current battery breaking and case disposal, this process is much more energy efficient than current techniques, even without the additional ventilation energy that retrofit would require.

d. *Battery manufacturing.* (1) *Costs of compliance.*—Cost estimates for compliance in this industry with the interim level of 100 μg/m³ were presented by DBA and CRA. Capital cost estimates, attributable entirely to engineering controls, were $345 and $307.7 million respectively. (Ex. 65B, p. 12; Ex. 127, Exec. Summ., p. 2.)

Total annualized after-tax costs were $42.2 and $37.5 million.

The sole source of the capital cost estimates in both cases was a study prepared by IHE for the Battery Council International. (Ex. 29 (29A).) The engineering controls selected by IHE for each process and production level in battery plants were accepted without modification by CRA. DBA, in addition to obtaining cost estimates from 12 unidentified plants, incorporated the CRA data in its cost analysis for OSHA, thereby implicitly incorporating the control specifications of IHE. Almost all of the battery manufacturers that provided testimony at the hearings also relied on the IHE study in defining company control costs.

OSHA has evaluated the IHE study and has determined that the study's cost estimates show a substantial amount of double counting and that its use by CRA, DBA, and individual manufacturers resulted in grossly exaggerated industry costs.

IHE surveyed 12 battery plants that it claims were a representative sample of the industry. The plants surveyed ranged in size from 40 to 519 employees and produced from 330 to 12,000 batteries per day. (Ex. 29 (29A), pp. 3-4). For these 12 plants, a very detailed analysis was provided of the processes and equipment in use and the engineering and work practice controls judged by IHE to be most cost-effective to control them to or below the proposed permissible exposure limit of 100 $\mu g/m^3$. Industry experts, industry consultants, and OSHA's contractor each agree with the IHE conclusion that the specified controls are technologically feasible and may reasonably be expected to reduce lead-in-air levels below the 100 $\mu g/m^3$ level.

From this, costs were estimated for each operation, with variable costs within the same operation to allow for different production capacities and manufacturing methods. These costs were used by CRA to obtain compliance estimates for its 83 plant samples and by individual manufacturers who presented cost information.

The cost estimates used by IHE do not differentiate costs for compliance with the present standard, do not account for equipment or ventilatory capacity in place, improperly allocate the costs of production equipment to control costs, and improperly include costs for external air and water pollution control.

For battery plants not in compliance with the present 200 $\mu g/m^3$ standard, a substantial amount of the cost should be attributable to meeting the present standard. An indication of the magnitude of this form of double counting is found in a three plant study done by IHE for counsel of LIA. (Ex. 138C) This study, which estimates the cost of compliance for three of the 12 plants in the BCI sample, shows that the costs for achieving the incremental change from noncompliance to compliance with the current standard can be as high as 30 percent of the total cost of compliance with the 100 $\mu g/m^3$ level. For manufacturers not presently in compliance with the 200 $\mu g/m^3$ standard, costs may be overestimated by about 30 percent. (Tr. 3340.)

IHE's estimates also implicitly assumed that operations in the plant where air levels exceeded 100 $\mu g/m^3$ (even if they were minimally above) would be totally uncontrolled and that entire, new ventilations systems would be required. In other words, if 10,000 cfm were required to ventilate an oxide mill, the cost of ventilation would be figured on the basis of 10,000 cfm, even if the plant presently had 6,000 cfm on the mill. OSHA recognizes that increasing the ventilation is not simply a matter of "adding on" cfm's, but in many cases existing dust control systems can be salvaged, adopted, enlarged, or used in various ways. Despite denials (Tr. 3902), IHE completely discounted this possibility (Ex. 349, pp. 5-11) whereas an "engineer designing a control system for a plant would attempt to minimize the cost of the project by maximizing reuse of existing equipment." (Ex. 349, p. 6) The testimony of a small battery manufacturer, Laher Battery Co., illustrates this principle. 16,000 additional cfm's were added to its plant for $30,000; using IHE's figures ($8/cfm), the same capacity would have cost $128.000.

IHE's approach also did not rely on accurate air sampling data when costing all those operations that were over 100 $\mu g/m^3$. Operations where employee airborne lead levels were slightly in excess of 100 $\mu g/m^3$. were treated the same as levels 10 or more times 100 $\mu g/m^3$. (Tr. 3901.) It is clear, and IHE itself suggests, that work practices and housekeeping can reduce air lead levels about 20 percent (Tr. 3934). It is inappropriate to use entire new systems, as IHE recommends, when simple and inexpensive solutions are available. (35)

IHE's recommended changes also include new production equipment. For example, in large plants, the cost for oxide mixing and pasting machines is estimated at $1.848 million. Of IHE's 12 sample plants, four fit into this category. The average capital cost of compliance for them is $3.593 million. Thus, IHE's cost for production, not control equipment, in just one of 14 operations, is over 50 percent of the total cost. In addition, no value was allowed for production equipment replaced by the new equipment. Costs for external air and water pollution control systems were also improperly added into IHE's cost calculations. Cost for a water treatment system is estimated as high as 250,000 ($35,000/year operating cost), while dust control systems are estimated at $8.00 per cfm which includes the entire fabric filter collection system to avoid external air pollution.

Two other factors affect cost estimates for this industry—the use of the IHE report to estimate costs for the approximately 100 small battery manufacturers combined with a revision OSHA has made from the proposed standard which would permit work practices, including administrative controls, to be used on an equal priority with engineering controls.

Although IHE asserts that its sample was selected to be representative of the industry, not one of the 12 plants studied was from the group with less than 20 employees, of which there are 95 firms in the industry, and only two were from the next larger size class. It may be that the processes, equipment and production pattern of all small firms are sufficiently similar so that the two smallest plants in the IHE sample are fully representative of this segment of the industry, but testimony from small battery manufacturers casts doubt on this conclusion. In small plants, each production process is not continuous and operators do not remain at each work station for a whole shift. In addition, the minimum production rates for which size differences are recognized are relatively large, e.g., 1140 batteries per day for mixing and pasting. It is not clear that different specifications could not be devised for operations under 500-600 batteries/day.

New, and substantially less expensive, engineering control techniques may also reduce costs, especially in small plants. As mentioned earlier, the APSEE system could prove effective and Kermatrol, Inc., has guaranteed its system will be effective to reduce levels to below 50 $\mu g/m^3$. (Tr. 5217; 5208, 5220-21.)

IHE's cost estimates overestimate true costs because they focused completely on engineering controls. Dr. First testified that industry's failure to recognize the important interrelationship between good work practices and good control engineering "accounts for the astonishingly high cost estimates * * * Far lower costs for equipment additions and modifications will arise if appropriate attention is given to training employees in effective work practices and supervision." (Tr. 2313-2314.) The final standard has elevated work practices from a less preferred to an equally preferred method with engineering controls and hence has given the employer the opportunity to significantly minimize

costs by permitting the employer to place primary reliance on a low-cost, noncapital method where appropriate.

This principle is also true if the employer can effectively utilize administrative controls to reduce employees' TWA exposure. To the extent that they can, reliance on capital intensive improvements will be minimized. This will be especially helpful to the small battery manufacturer.

For these reasons, OSHA has concluded that the incremental cost of compliance will be at least one-third lower for the industry as a whole. OSHA's estimate for compliance with the interim level of 100 $\mu g/m^3$ is thus between $205.1 million and $230 million for capital costs and $25.0 million and $28.1 million for after-tax total annualized costs. These figures are probably also inflated because they include costs for all firms including the small business segment of the industry for which the expanded compliance alternatives permitted by the standard will allow for low-cost solutions involving much less capital investment.

Cost estimates for compliance with the PEL over a 5-year period are difficult to make. Essentially the same technological changes will be necessary to reach the PEL as to reach the interim level, and costs may not be significantly more. In fact, proper implementation of changes necessary to reach the interim level will likely comply with the PEL. But operating with antiquated equipment which would be expensive and difficult to retrofit, the Bergsoe process may be a more cost-effective longrun solution. Given the operating efficiencies Bergsoe claims for his existing smelters, the entire industry may eventually convert for competitive reasons. Bergsoe estimates a 2-year period will be necessary to construct a 20,000 ton smelting and refining facility. OSHA has determined that 5 years is an appropriate compliance time for meeting the PEL.

No significant change in prices is projected for secondary lead products, except to the extent primary producers can raise their price allowing secondary producers to follow. Labor requirements were estimated by DBA to increase by three resulting in a decrease in average productivity of 2.9 percent (Ex. 26, p. 6-33). It should be noted that the Bergsoe process is much more labor efficient than current smelting and refining techniques. Bergsoe testified that only three production workers are required in the smelting and another three for the refinery per shift. (Tr. 5201.) Thus, conversion to that process would result in a huge increase in productivity.

Increased energy usage was estimated by DBA to range between 16,520 and 156,000 MWH/year with the best estimate being an increase of 45,120 MWH/year. (Ex. 26, p. 5-40.) This would have no significant impact on energy supplies or demand. This estimate does not include calculation of the potential if further refinement of similar controls is necessary, OSHA expects knowledge to be obtained during the 2 year period which should limit any additional costs. On the other hand, if problems are found in the initial period, greater costs could result.

(2) *Economic Impact.* The cost estimates provided by IHE to DBA and CRA are the basis of their impact analyses. Since OSHA considers them greatly exaggerated, alternative cost estimates have been calculated from the record and have been used to establish a more rational assessment of projected impact.

OSHA's conclusion is that the battery industry will be largely unaffected in terms of production, capacity, and competition and that the price of batteries will increase by less than CRA's estimate of $1.75 per battery at retail as a result of a pass-through of increased production costs and associated mark-ups. The demand for batteries is derived from the demand for automobiles. Since there are no close substitutes and foreign competition is not significant, the long-run demand is relatively price inelastic. (Ex. 127, p. 3-12 through 3-14; Ex. 26, p. 6-37.) This allows price-setters to pass through to consumers all increased costs of production.

The battery industry is essentially an oligopolistic industry with a fringe of small independent producers who compete in regional or specialty markets (Ex. 26, p. 6-37). It is comprised of 138 companies who operate a total of 200 plants, but the five largest companies, who operate 55 plants having 78 percent of the total industry capacity, dominate the market. (Ex. 26, pp. 6-33, 6-37.) The seven largest companies operate 70 plants and sell 90 percent of all the batteries sold. (Ex. 26, p. 5-42 (*36*).) It is also an industry that has been in the process of consolidation for many years. In the past 20 years the number of firms in the industry has steadily decreased from over 300 in 1954 (Ex. 127, p. 3-4) to just 138 in 1972 (Ex. 26, p. 6-33).

The questionable assumptions underlying the IHE report lead to the conclusions drawn by DBA and CRA that approximately 100 small battery manufacturers would exit the industry as a result of the proposed standard. (Ex. 127, p. 3-53; Ex. 26, p. 6-24.) OSHA does not believe that the approximately 100 small plants will have to assume the magnitude of cost used by DBA and CRA because of the overestimation of costs by IHE, because the lead quantity in small plants is lower (Ex. 349, pp. 16-18), and because of several available low cost compliance alternatives, discussed earlier, which are uniquely suited to small plants. In addition, some small manufacturers might take advantage of economies of scale by increasing production, e.g., expanding a one-shift operation to a two-or-three-shift operation.

Some of these small firms will probably exit the market irrespective of the OSHA standard. There has been a trend in recent years of very small firms (95 firms have less than 20 employees and a total of 2 percent of the market) leaving the industry because of unprofitability. These firms have discovered shrinking markets for their products, and an inability to compete with larger companies because size is related to production efficiency. Most of the new plants in the industry have been quite large. (Ex. 127, pp. 3-6.) These factors are expected to continue to put severe stress on the small battery manufacturer without respect to additional costs due to OSHA regulations, and the consolidation trend is expected to continue.

OSHA has concluded that even if the questionable DBA and CRA prediction that approximately 100 small manufacturers would exit the market were true, the standard is nonetheless feasible for the battery industry.

Closure of 100 small businesses would have a minimal impact on the competitive structure of the industry. Thirty firms operating 100 plants will remain, and the capacity of the seven largest firms, now 90 percent of industry capacity, will increase a few percent. Competition from the smaller firms has little or no effect on the price of batteries, which is set by the major producers, except in those "interstices of the market which the major producers do not choose to capture." (Ex. 349, p. 19; Ex. 26, p. 6-42; Ex. 127, pp. 3-7 through 3-9.) The small producers may set prices in small local markets where they supply retailers directly and take, in price, the equivalent of distributor markups or where special services (picking up old batteries, fast delivery, etc.) to the retailer allow price increases. (Ex. 127, p. 3-8.)

Battery prices will increase as a result of the pass through of compliance cost. The industry price setters, the five major producers, will have compliance costs of about $0.74 per battery, with an industry average of $1.11. (Ex. 127, p. 3-35.) CRA has estimated that a cost pass-through of $0.74 will result in a retail price increase, due to markups in the distribution chain, of about $1.75 per battery. (Ex. 127, Exec. Summ., p. 37.) This will allow small producers who enter the distribution chain at advanced stages

to pass through costs of about $1.04 per battery (Ex. 127, Exec. Summ., p. 37), except where they are not in competition with the major firms.

Closing of 100 plants employing 10 persons each would mean the loss of approximately 1,000 jobs. Compliance activities require additional man-hours, however, and it is estimated that the net gain in employment, if production remains at the prestandard level, would be approximately 2,000 employees. Productivity, therefore, would decrease by just over 9 percent. The impact on wages would be small (Ex. 26, p. 6-43 and 6-44).

(3) *Compliance Schedule.* OSHA's evaluation of the technology available to the battery industry indicates that compliance with the PEL may be achieved by the same types of technological changes required to achieve the interim level of 100 $\mu g/m^3$, although further refinement, additions, and modifications may also be necessary. The compliance schedule requiring engineering controls and work practices to be used to reach 100 $\mu g/m^3$ in 2 years and the PEL in 5 years is based on the time it should take to implement the relatively conventional control methods required. Large manufacturers should have little problem meeting the costs involved, especially since they will be able to pass on all of the increased costs of production to consumers. For smaller manufacturers, OSHA has concluded that simple and inexpensive approaches can be effective in many situations, thereby drastically decreasing their inordinately excessive estimates of compliance cost. Where capital acquisition problems are encountered in meeting the implementation schedule, the flexibility in the compliance scheme for the standard should, under certain conditions, enable employers to spread compliance costs over 5 years.

e. *Brass and bronze foundries.* (1) *Costs of compliance.* Based on a survey of five foundries and two ingot producers, DBA provided estimates of capital and annual costs for compliance with the proposed standard. Costs per plant varied widely. When extrapolated to the industry as a whole on the basis of costs per ton of output and costs per employee exposed, the best estimated for capital cost was $161 million, with $41.2 million for annual recurring costs. Total annualized costs after taxes was estimated to be $42.2 million. (Ex. 65B, p. 12.) This is equivalent to approximately $1,600 per employee or 25-40 percent less than the comparable cost in the primary smelting industry.

(2) *Economic impact.* DBA projected the following economic impacts as a result of compliance with the proposed lead standard. DBA expects an increase in the average industry price of 8.7 percent, or $0.16 per pound of casting. (Ex. 26, p. 6-60.) This is approximately double the price increase necessary to cover compliance cost and incorporates an assumption regarding the ability of the industry to maintain historic profit rates. Some shifts will occur in the price differences among product types and these will favor larger foundries that have lower-than-average compliance costs. (Ex. 26, pp. 6-60 to 6-62.)

DBA's assume the long run price elasticity of demand to be fairly high due to the availability of substitute products. If this is true, total industry output will fall, some firms with high compliance costs will leave the industry, and competition will be minimally reduced. (Ex. 26, p. 6-62.)

Compliance activities will require a significant increase in employment. DBA's best estimate is 2.7 million man-hours per year, equal to 1,954 persons. This may be partially offset by employment decreases due to lower industry output. (Ex. 26, p. 6-64.) DBA's best estimate of the average labor productivity decline in 9.9 percent. (Ex. 26, p. 6-67.) This assumes no increase in output from an industry operating well under full capacity.

It should be noted that an industry trade association, the American Foundrymen's Society, which represents over 1,800 foundries, testified but did not claim that the proposed standard would cause economic hardship for the industry. (Tr. 2785-2824.)

(3) *Compliance dates.* DBA concluded that the nonferrous foundry industry is capable of attaining 100 $\mu g/m^3$ through relatively simple engineering controls. This conclusion was not disputed, and OSHA has determined that 1 year should be sufficient to implement these controls. A more extensive and refined use of these same controls should be able to achieve compliance with the PEL. Since a sizable segment of the industry does not presently employ satisfactory control methods, OSHA has estimated that 5 years will be required to allow sufficient refinement of control techniques. Given the extended compliance time, the industry will have an opportunity to recover from recent depressed conditions. Individual firms will have a longer time horizon over which to stretch compliance costs. Under these conditions, the implementation of the PEL should not cause undue economic disruption for the industry.

f. *Inorganic Pigments.*

(1) *Costs of Compliance.* DBA extrapolated industry costs from a very limited data base. Capital costs range from $4,451,000 to $109,540,000, depending upon the method of estimation. Similarly, annual cost estimates ranged from $347,000 to $14,800,000 (Ex. 26, p. 5-102). The high estimates are based on an extrapolation of the costs of new facilities, equipment, and processes (Ex. 26, p. 5-104). As such, they represent the upward bound of the cost of compliance with the PEL, as it is an equivalent of rebuilding the entire industry with health goals in mind. As the Short report (Ex. 22) contains the best available information for this industry in the record, despite its shortcomings, OSHA has used it to obtain an estimate of costs to comply with the interim level. According to Short, the upper level of cost will be $21.1 million and $6.4 million for capital and annual recurring costs, respectively. This is consistent with the geometric mean of the costs from the three pigment plants giving data to DBA. Those figures are $17.6 million in capital costs and $2.9 million in annual recurring costs. Thus, OSHA's best estimate for the range of costs for the 100 $\mu g/m^3$ level is $17.6-$21.1 million in capital costs and $2.9-$5.0 million in after-tax, total annualized costs.

(2) *Economic Impact.* DBA concluded that the prices of lead pigments would probably rise by 16.6 to 21.6 percent and that the output of the small, very competitive firms would fall by similar percentages if they tried to maintain their profit margins. Large firms would be largely unaffected. DBA concluded their analysis with this statement: "Given the regional orientation of plants, the concentration of economic market power, the existence of bilateral monopoly relationships, and the presence of a competitive fringe of buyers and sellers, our best judgment leads us to conclude that most, if not all, of the compliance costs will be passed on to the users of lead pigments and that the degree of competition in the industry will decline slightly as merginal firms are forced to leave the industry." (Ex. 26, p. 6-77.) OSHA's cost revisions in this industry mitigate these conclusions. Using the revised estimates, price increases necessary to maintain profit margins will only be 1.7 percent to 3.7 percent.

The industry faces several choices in attempting to comply with the PEL for lead and other potential OSHA standards particularly for hexavalent chromium. DBA assumed that all firms would attempt to comply with the 100 $\mu g/m^3$ level by retrofitting engineering controls. That report did point out the other options of product substitution (for lead chromates) and process redesign. The high estimate ($109 million) may be taken as a proxy for the cost of the latter. OSHA has no estimate of the likelihood of successful substitution of organic ingredients for lead in pigment manufacture.

Because the industry is old (4 of the 5 plants visited by DBA were in excess of 50 years old), retrofitting engineering controls may not be the most cost-effective solution. Even retrofitting will involve enclosure and automation of some processes. OSHA has therefore concluded that the industry should have 5 years to comply with the PEL by use of engineering controls and work practices. This time is deemed adequate for the selection of the most effective compliance strategy by the individual firms and implementation of that strategy. It should be noted that even using the $109 million estimate, DBA predicted the exit of only the most marginal producers. OSHA has further determined that it is generally feasible for firms to comply with the 100 $\mu g/m^3$ milestone in 3 years.

g. *Can Manufacture.* The DBA report estimates costs for the can manufacturing industry as a whole as $157,600 annually for the approximately 1,200-2,000 exposed workers. (Ex. 65B, p. 20.(*37*) A one-time cost of $30,000 for an initial determination is also given. No capital costs are expected since engineering controls are presently in place and when operating properly are successful in keeping employee exposure very low. If initial determinations yield airborne lead levels below the action level, and there is reason to believe they will,(*38*) the industry should have minimal annual costs. These costs would be attributable only to housekeeping and training, and by DBA's estimates should be about $260,000 per year or $130/worker per year.

The can manufacturing industry is a $5 billion industry (Ex. 3(81)), and no adverse economic impacts have been suggested.

h. *Printing.* DBA's followup on its original report showed that several segments of the industry which use lead, including the largest, newspaper printing, are rapidly moving away from lead alloy hot metal printing processes. (Ex. 65B, p. 21) By 1980 very few newspapers are expected to have employees exposed to lead, and with recent technological developments facilitating conversion to cold printing processes, other segments are expected to follow suit. The Short report estimated that in 5 to 10 years all but 5-7½ percent of the industry will have converted to the cold process, meaning that only 8,750-13,125 employees will be exposed to lead. (Ex. 22, pp. 194, 201)

Engineering controls are generally in place and because the temperatures involved in melting operations are low, exposure levels are "well below" 50 $\mu g/m^3$ as an 8-hour TWA throughout the industry (Ex. 22, p. 194). According to the Printing Industry of Illinois, air levels in sawing operations would never even approach 50 $\mu g/m^3$ (Ex. 3(25)) and would probably be in the range of 0.2 to 1.7 $\mu g/m^3$ (Ex. 3(60)). Thus, there appear to be no capital costs involved with compliance, and with adjustments for the decrease in exposed employees, the first year cost for 109,000 employees would be approximately $20,000,000 or $183 per employee. (Ex. 65B, p. 21) As the number of exposed employees decreases to the eventual range of 8,750-13,125, the total annual costs for the industry will decrease proportionally.

There are many small firms in this industry and the ones that continue to use lead are expected to be the small firms. (Ex. 22, p. 195) However, given the minimal per employee compliance costs, no adverse economic effects are anticipated.

i. *Paint Manufacturing.* The Short report estimated capital coss for 1,000 companies of varying sizes to be between $9 million and $26.8 million. Annual costs were estimated at between $6.8 million and $13.1 million. DBA confirmed the upper bound on the basis of cost data from the Sherwin-Williams Co., the only paint manufacturing firm supplying cost data directly to the record. However, DBA's conclusion is misleading because the cost data included cost of compliance with the chromate standard (Ex. 65B, p. 36) and attributed costs primarily to medical exams, air monitoring, and recordkeeping. (Ex. 3(97), p. 3) Hence, OSHA has concluded that the Short reports cost estimates are highly inflated and that the industry should not experience economic difficulty in complying with the standard.

j. *Ink Manufacturing.* Estimated costs for compliance in this industry are derived by extrapolation from the costs of an "average plant" in the industry as determined by a trade association of ink manufacturers, the National Association of Printing Ink Manufacturers, Inc. (NAPIM). NAPIM submitted the information to DBA without disaggregating the figures or explaining the study methodology.

The estimated costs are $4.6 million for capital costs and $1.25 million for annual recurring costs. (Ex. 65B, p. 39) If these figures are accurate, the annual recurring costs per exposed employee are between $962 and $1,250. Neither Short nor DBA reported predictions of economic hardship from the industry. OSHA concludes that compliance with the standard will not cause economic difficulty for the industry.

k. *Shipbuilding.* DBA estimated the costs for this industry on a per employee basis for the 22,253 workers exposed to lead. Capital expenditures totaled $126,537 or $5.69 per employee and annual expenses totaled $22.256 million or $1,000.12 per employee.(*39*) Additional labor requirements were estimated to be 1,369.5 man-years, and energy consumption 606,000 kWh.

The impact of these costs are difficult to evaluate in view of the unavailability of basic economic data. The Shipbuilders Council of America, in a brief comment, stated their opinion that the benefits would not justify the costs of the proposed standard, but made no claim of economic hardship and supplied no data for evaluating cost impacts.

An important factor to consider is the availability of a construction-differential subsidy from the Maritime Administration of the Department of Commerce to offset foreign competition. The ceiling on this subsidy is statutorily set at 35 percent, but according to The Shipbuilders Council of America, "ability to maintain a 35-percent level is now in jeopardy." (Ex. 26, p. 5-110)

Another factor is that a good portion of shipbuilding and repairing contracts are with the U.S. Government. Cost increases would be reflected in those contracts. (Ex. 3(58))

l. *Other Industries.* The DBA report performed full economic impact analyses for five industries. Costs of compliance for shipbuilding and automobile manufacturing were developed in a substantial manner. Less detailed cost estimates were developed for the can manufacturing, printing, paint manufacturing, and ink manufacturing industries. Some of the latter are merely adjustments to or confirmation of costs estimates presented in the preliminary Short study.

The Short methodology for determining and extrapolating costs to all affected industries was based on determining the number of employees in five different exposure categories for each industry (e.g., the high category had 40 percent of the employees between 50 and 100 $\mu g/m^3$ and 60 percent over 100 $\mu g/m^3$, while the low category had 70 percent below 50 $\mu g/m^3$, 20 percent between 50 and 100 $\mu g/m^3$, and 10 percent greater than 100 $\mu g/m^3$), assigning capital and annual costs on a per employee basis for each category, and multiplying the per employee cost by the number of exposed employees in each category. (Ex. 22, pp. 115-126) The per employee costs were orginally criticized during review of a draft of the report because they were based on too few data points, and further data was gathered in selected industries by DBA.

DBA's report presents empirical cost data for 10 industries, three in the high and very high exposure categories, three in the medium category,

two in the low category, and two in the very low category. When the average from each category is compared to the Short costs for each category, the relative validity of the Short estimates are enhanced.(*40*)

Since the per employee estimates in the Short report offer the best available information to extrapolate costs to the 27 industries DBA did not study, OSHA has used it to project costs to those industries. OSHA believes these costs, although developed for achieving a 100 $\mu g/m^3$ PEL, are generally appropriate costs for achieving either a 100 or 50 $\mu g/m^3$ PEL. Capital costs are for engineering controls, and in these industries exposure levels are generally low, necessitating simple and inexpensive enclosure or ventilation systems. Fugitive emissions are expected to be negligible once control devices are installed, so controls which are effective to achieve 100 $\mu g/m^3$ should result in exposures well below the PEL. Annual costs are also expected to be similar. In addition, Short provided "best" and "high" estimates for these industries. OSHA has conservatively chosen the "high" estimate for these calculations.

The 27 industries for which cost estimates have been developed in this way employ 630,335 potentially exposed workers. Capital costs for compliance total $118.4 million, and annual costs total $84.5 million. On a per employee basis, this is equivalent to $188 and $134, respectively. None of these industries presented evidence or made claims that economic hardship would result from the proposed standard.

In addition, there are many other industries in which lead exposure may occur; e.g., pipe galvanizing, brick and tile manufacturing, tanning, and bookbinding. In most cases, exposure is negligible and/or infrequent. For most of these industries, information is not available to assess the possible impacts of the standard. It is believed however that the standard will minimally affect these industries. (Ex. 22)

For all the industries covered in this section, the record does not provide explicit estimates of time required for compliance, but because exposure levels are generally low (often below the PEL) and because operations are often small scale, work practices and administrative controls could be used with some success. Only the simplest of conventional engineering controls should be required, and one year should provide adequate time to have them in place and operating.

13. *Aggregate Impacts.* DBA estimated the aggregate price and employment effects of the proposed standard. As would be expected given the size of the national economy, the anticipated changes in price and employment indices were minimal. Nationally, unemployment would be expected to decline by .08 percent (a net gain of 5,200 jobs) (Ex. 26, p. 6-81) and consumer prices would be expected to rise by .02 percent as a result of the expenditures flowing from the DBA best—estimates of compliance costs for the 100 $\mu g/m^3$ proposal. (Ex. 26, p. 6-85.) The OSHA revisions of the DBA cost estimates would reduce these impacts even further.

Since the cost of attaining the PEL was not estimated for all industries, quantification of its aggregate impact is not possible. It is reasonable to treat the price and employment effects in the DBA study as first approximations of the magnitude of the impact of the 50 $\mu g/m^3$ standard. It is clear that even if the price effects approximation incorporates understatements of the real cost of meeting the PEL, there could be no discernable inflationary impact on the U.S. economy attributable to the standard.

Likewise, the national employment and productivity effects would be inconsequential. The only significant national impact suggested in the DBA study was the possibility of labor shortages among professionals in specialized health fields. OSHA believes that such shortages would be temporary in nature and concludes that no permanent disruption of labor markets will result from the standard.

FOOTNOTES

(*1*) In *SPI* v. *OSHA*, the court quoted with approval the language from OSHA's preamble that "it is not possible to predict the degree of improvement to be obtained from engineering changes until such changes are actually implemented." 509 F.2d at 1309. In *Atlantic and Gulf Stevedores, Inc* v. *OSHRC*, the Third Circuit likewise recognized that the feasibility or infeasibility of a standard may not become apparent until the employer in good faith attempts to comply (534 F.2d 541 (1976)).

(*2*) *AFL-CIO* v. *Brennan*, 530 F.2d at 122. The language cited referred to the Secretary's determination on the issue of feasibility, which the court upheld, and said that such a determination "necessarily implie(d)" consideration of both existing capabilities and imminent advances.

(*3*) *AISI* v. *OSHA*, 577 F.2d at 835. Specifically, evidence had been introduced that retrofit devices readily adaptable to old batteries were in the experimental stage, and the court appeared to find this evidence persuasive. It is not clear whether the determination of the feasibility of the standard was based on this evidence concerning old batteries or whether the holding was based on the fact that there was substantial evidence that new batteries could comply and, therefore, the standard was feasible for the coke oven industry.

(*4*) S. Rep. No. 91-1282, 91st. Cong. 2d Sess., U.S. Congressional and Administrative News (1970) p. 5180. This section specifically recognizes that without regulation, firms which provide safe and healthful workplaces suffer a competitive disadvantage until the recalcitrant employers change their practices.

(*5*) In *SPI* v. *OSHA*, 509 F.2d at 1309 the court recognized that reactor cleaning practices in many firms (a substantial source of exposure) could be improved to conform to the most advanced practice. Even this, however, would not insure compliance and therefore new technology was necessary in both the leading and secondary firms. In *AISI* v. *OSHA*, 577 F.2d at 832-33, great reliance was placed by the court on the fact that two batteries had been able to make great strides toward meeting the standard, although even they had not achieved compliance.

(*6*) The no-hands-in-dies standard was a consensus standard, apparently intended from its inception as a desirable guideline rather than as a requirement which all firms could meet. It was adopted by OSHA without a full consideration of its feasibility. The primary grounds for OSHA's later finding of infeasibility was that the technology was not "universally possible in the near future." Therefore, the costs were judged to be "prohibitive."

(*7*) "Regardless of the manner in which the task of judicial review is articulated, policy choices of this type are not subject to verification or refutation by reference to the record as are some factual questions." Consequently, the Court's approach must necessarily be different no matter how the standards of review are labeled (*IUD* v. *Hodgson*, 499 F.2d at 475).

(*8*) Temporary variances, it is clear from the legislative history, cannot be granted in order to mitigate economic hardship. (Conf. Rep. Pub. L. 91-596, U.S. Code Cong. and Admin. News, 91st Cong. 2d Sess. p. 5231). However, temporary variances which enable firms to delay compliance for the time needed to obtain abatement supplies or permanent variance which employers may obtain if they demonstrate alternative equally protective means of compliance with a standard may have, as a side effect, the mitigation of economic hardship for certain firms.

(*9*) DBA reported that one company has had good success with traditional forms of engineering controls without use of respirators. This company, however, does not use lap soldering, the kind of soldering which is found on most automobiles and which requires considerable solder to be ground (Ex. 26, p. 5-135).

(*10*) These terms are used throughout to express costs of compliance. Terms used in other reports or studies have been adjusted to avoid ambiguity.

(*11*) An important legislative development that was not included in the report but that might provide additional economic relief for companies faced with capital expenditures due to OSHA regulation is the introduction of two tax bills, H.R. 10692 and H.R. 11078, in the House of Representatives. These bills would provide for 1 and 2 year amortization of plant or other property required by regulations under the OSHA Act.

(*12*) Excluding the one plant whose cost estimates are based on new facilities, equipment, and processes, a best estimate might be calculated from the three plants submitting data. Using the geometric mean and extrapolating to the entire industry, the best estimate for capital costs would be $17.6 million and for annual costs $2.9 million. This of course assumes the per employee cost of these three plants is representative for the 25 in the industry.

(13) There are a few exceptions in the primary smelting industry. See industry summaries below.

(14) Capital costs for Amax were estimated in a report by IHE (Ex. 3 (108), p. 4) as $8.144 million, the number used by CRA. IHE added approximately 10 percent for "escalation"; DBA added an additional 8 percent to arrive at its estimate of $9.540 million.

(15) Dr. Burrows of CRA stated that the after-tax costs are the "real" costs to a firm, although they are not the basis for making decisions of whether to stay in business (Tr. 3368-69).

(16) CRA's $13.2 million estimate is for long-term costs. For the short-term (3 years), CRA estimated costs to be $15.6 million per year. This, however, does not explain the disparity.

(17) Certain financial information (cost of capital, tax structure) is necessary to make valid predictions of any company's economic behavior (Tr. 4644, 4646). In most cases, companies do not voluntarily make information public, and for those publicly owned companies that have some public disclosure obligation, the requirements are minimal and don't yield the type of information needed for these types of economic analyses. For example, large companies were under no obligation to publish information on a plant by plant or industry by industry basis (Tr. 4630). Many of the parties to this rulemaking are divisions or subsidiaries of larger companies (ESB is a division of International Nickel of Canada, General Battery is a division of Northwest Industries, Delco-Remy is a division of General Motors, Bunker Hill is a subsidiary of Gulf Resources & Chemical Co.), and, as such, no breakdowns of their financial data can be extracted from the parent firms' annual reports and other SEC filings.

(18) This was figured in the same manner as CRA. (See Ex. 127, Table 2-17, n. 1.)

(19) There was no way to reconcile the diverse estimates for annual costs attributable to medical surveillance, air monitoring, hygiene, housekeeping, and administrative costs in the two reports. The average of CRA's long run costs and DBA's single estimate is used. It is recognized that the short-run costs will be higher, but that is offset by the higher DBA estimate which does not take long-run costs into account. (See Ex. 127, Table 2-16; Ex. 26, Tables 5.8-5.14.) DBA's total for seven plants is $6.669 million; CRA's total is 1.786.

(20) 723,879 short tons (Ex. 127, Table 2-11).

(21) 3,055 exposed employees (Ex. 26, Table 5.1).

(22) In addition to the two sources discussed below, the U.S. Government maintains a stockpile from which it sells periodically (Ex. 127, Exec. Summ., p. 16).

(23) Secondary producers can and do produce refined or "soft" lead, but because much of the reclaimed scrap lead contains alloys such as antimony and because antimonial or "hard" lead is required for the convential battery, many do not further refine the lead to produce "soft" lead.) Ex. 26, p. 6-5) However, the recent trend toward maintenance-free batteries, which do not use hard lead, has caused a downward shift in demand for hard lead, and antimony prices have dropped. (Sizemore, "Lead Recyclers Face Market Shift," American Metal Market, Batteries Section, April 26, 1977, pp. 12, 22).

(24) The price of lead used in the CRA analysis was 21 cents per pound compared with current price of 37 cents to 39 cents per pound. (Wall Street Journal (October 20, 1978, p. 38).

(25) OSHA Arsenic Record.

(26) While the 0.8 cent to 1.1 cents represents full cost recovery, the industry would have to absorb between 0.4 cent and 0.6 cent in after-tax costs if passthrough were not possible.

(27) Primary production of refined lead in the U.S. has increased since 1971 from 678,655 short tons to 767,323 short tons in 1974. In 1975, production was 723,879 short tons, a decline CRA attributes to a slack demand that year (Ex. 127, p. 2-24, Table 2-11).

(28) Australia, a significant exporter of refined lead to the United States, was virtually excluded from the U.S. market in 1974 due to "dumping" charges since withdrawn (Ex. 127, p. 1-30).

(29) Since ASARCO announced these commitments, the price of lead has approximately doubled.

(30) CRA's combination of all State and Federal water and air pollution control costs with OSHA costs obscures the attempt by OSHA to analyze only the impact of OSHA-related costs. OSHA recognizes that firms have all types of additional, non-OSHA costs of production which affect their financial viability, the aggregate of which may render them unprofitable. However, to attribute a firm's ultimate decision to cease operations to one component which, by itself, would not cause the same result is misleading.

(31) The IHE study is appended as Exhibit A to a Stipulation and Settlement to Obtain An Order, dated January 5, 1977, in *Usery* v. *Gopher Smelting & Refining.*

(32) Derived from Ex. 127, Exec. Summ., Table 5. Adjustments were made to determine costs on the basis of per unit of production, not capacity.

(33) 724,340 short tons (Ex. 26, p. 5-40).

(34) This calculation is based on 725,000 tons capacity rebuild at $2.5 million per 20,000 tons ($125 per ton) (Tr. 5192).

(35) In addition, IHE's estimates are for new equipment when, in reality, small firms often purchase used equipment to save money (Tr. 3300; 3890).

(36) The seven largest producers are ESB Co. (a division of International Nickel of Canada) General Battery Co. (a division of Northwest Industries); Delco Remy (a division of General Motors); Gould, Inc.; Globe Union; Chloride, Inc., and Prestolite.

(37) There is apparently an error in multiplication in the DBA report. The estimate should total $496,000.

(38) There is little exposure data in this industry, but two companies reported exposures among lead-exposed employees to be between .002 $\mu g/m^3$ (Ex. 65B, p. 19) and .4 $\mu g/m^3$.

(39) Capital costs are expectionally low in this industry because engineering controls to control exposure consist primarily of portable local exhaust ventilation units costing about $650.

(40) The Short report's per employee costs are as follows (Ex. 22, pp. 125-126):

Category	Capital costs	Annual cost
Very high	$10,600/$7,875	$3,210/$2,785
High	7,100/5,250	2,650/2,295
Medium	3,500/2,625	1,565/1,375
Low	1,200/875	815/725
Very low	250/0	380/380

NOTE.—Costs for each category are presented respectively as costs for large or indeterminate scale operations and small operations. Large scale operations tend to be costlier than small scale operations. (Ex. 22, p. 16.)

DBA's average cost figures for each category are as follows. Operation size was not given.

Category	Capital costs	Annual costs
Very high	$17,042	$3,116
High		
Medium	3,763	1,247
Low	128	696
Very low	99	132

NOTE.—Average for very high and high is derived from primary and secondary smelters and battery manufacturers; medium from brass and bronze foundries, paint manufacturing, and ink manufacturing; low from automobile manufacturing and shipbuilding; very low from printing and can manufacturing.

This document was prepared under the direction of Eula Bingham, Assistant Secretary of Labor for Occupational Safety and Health, 200 Constitution Avenue NW., Washington, D.C. 20210.

Signed at Washington, D.C., this 8th day of November, 1978.

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[FR Doc. 78-31912; Filed 11-13-78; 8:45 am]