DEPARTMENT OF LABOR

Occupational Safety and Health Administration

OCCUPATIONAL EXPOSURE TO LEAD

Attachments to the Preamble for the Final Standard
CHAPTER XVII—OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION, DEPARTMENT OF LABOR

PART 1910—OCCUPATIONAL SAFETY AND HEALTH STANDARDS

RULES AND REGULATIONS

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, in some cases, is removed from the body primarily through the alimentary tract and urinary system.

Lead intoxication, in its severest forms, is a cause of damage to the body or cause death. Observed clinical effects include damage to the central nervous system, including the brain, i.e., 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. The predilection 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 CaEDTA, 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, (PEIS) 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 expo-
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 biochemical 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;
2. Clinically, blood samples are relatively easy to obtain compared to brain, kidney or other tissue samples.

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, aminolevulinic acid 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 coproporphinase, 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 ALA synthetase and coproporphinase. 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)
It is the third step in the pathway of heme synthesis which converts 5-amino­methylene acid (ALA) into protophobilinogen. The enzyme mediating this reaction is 5-amino­methylene 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 1985, 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, catalyzed 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.

Measurements of Heme Synthesis Inhibition. 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 box 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 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 (113); 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 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 a 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. 284 (E).)

The inhibition of ALA-D limits the transformation of ALA into protophobilinogen. 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 enzyme's 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. Zielhuis, 1974).

Protoporphyrin. The accumulation of protoporphyrin in the erythrocytes of humans with lead intoxication has been known since 1903. (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 Yamana (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). This relationship 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)).
The concentration of ZPP in the erythrocytes starts to increase at PbB levels of about 20 to 35 \( \mu g/100 \text{ ml} \) in children, 20 to 35 \( \mu g/100 \text{ ml} \) in adult women and about 30 to 40 \( \mu g/100 \text{ ml} \) 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 children with PbB levels greater than 30 \( \mu g/100 \text{ ml} \). These results are given in Table 5. Similar exponential response, but at lower PbB levels, has been found in children (Tr. 443; Protoporphyra Boys). (Ex. 11, Ref. 147; Ch. 11, Ref. 148; op. cit., Ch. 11, Ref. 149.)

**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 this reserve capacity 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 as we know today, there is no clear evidence that the suppression of enzyme activities in the blood formation causes any departures from the normal well being unless it causes a fall in the hemoglobin level.

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The best way of assuring that this does not happen is to carry out periodic hemoglobin estimations. Changes in enzyme system, in my view, can 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-U, nor increased 2FP 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 changes 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 individual in the case of poisoning. Of course everything in life as I am sure you are familiar happens in a complex 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 added 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 accumulation and a possibility of lead toxicity miy occur much before the onset of immediate clinical symptoms. Enzyme 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 physiological 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 2FP, have been found. (Ex. 81 (C); Ex. 105 (B)) 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 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 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 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 employee". When continued exposure will eventually result in serious disease symptoms, prudent policy requires that the early stages of the disease process be avoided.
c. The Hematopoietic System. In addition to the effect of lead on the synthesis of the hemolytic radical itself, hemoglobin production may also be impaired by lead due to an inhibition in the synthesis of the protein portion of the molecule. Kassan et al. (EPA Criteria Document, Ch. 11, Ref. 174) and Wada, et al. (EPA Criteria Document, Ch. 11, Ref. 170) have shown that hemoglobin production 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 is the form of ferritin that tends to occur. The iron is a characteristic of lead poisoning and dense aggregations of ferritin in damaged mitochondria. Several investigators (Ref. 95) have demonstrated a slight shortening of the life span of erythrocytes in man's physiologic reserve caused by lead. 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(18)) used a hemoglobin level (Hb) below 12.5g/100g to define anemia; Bell (Ex. 8(c)) used a cut-off point of 10g/100g. Fischbein et al. (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 life spans, 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.
between 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. 599) studied 218 workers with lead exposure of less than 65 \( \mu g/100 \text{ ml} \). No decrease in Hb was found in workers with PbB levels of less than 50 \( \mu g/100 \text{ ml} \). Upon review of Sakurai's data, Cooper and Tabershaw (Ex. 637) observed increases in the proportion of workers with abnormally low levels and PbB levels of 70 \( \mu g/100 \text{ ml} \) in their analysis, they placed workers in groups according to PbB levels: 40, 40 to 69, 70 to 90, and 100+ \( \mu g/100 \text{ ml} \).

Hemoglobin concentrations ranged from 10.9 to 16.0 \( g/100 \text{ ml} \). The mean was 14.8 \( g/100 \text{ ml} \). There was no significant correlation with age. There was a significant negative correlation with blood lead level (\( r=-0.220 \), \( p \) less than 0.01). Thus of the 56 employees (18.7 percent of those examined) with hemoglobin concentrations below 14 \( g/100 \text{ ml} \), 28 were in the 114 employees with blood levels of 50 \( \mu g/100 \text{ ml} \) or higher. In a study that evaluated the effect of lead exposure over time, Tolsa (Ex. 518) studied 33 workers shortly after they became occupationally exposed to lead. After 100 days, the average blood lead was 50 \( \mu g/100 \text{ ml} \) and the 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 \( \mu g/100 \text{ ml} \). It has been suggested that PbB levels in the cross sectional studies may have been higher at an earlier point in time and point design eliminates this criticism, while also confirming the results of the cross sectional studies.

**Special sub-groups at risk.** Given the 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. McNamara and recently showed 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 500,000,000 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 congenially 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. 483)

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 usually aware of their disorder; however, it must be the responsibility of the employer to examine prospective employees and not to expose lead individuals with sickle cell anemia. (Ex. 57, p. 31)

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 unequivocally 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 is 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. This 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 trait are at greater risk from lead exposure than those without it.

Other nongenetic factors may also put workers 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. 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. Following these factors, there have lower Hb levels than men; the normal range of values for adult males is 14-18g/100ml, while for adult females it is 12-16g/100ml. 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 pg/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 pg/100 ml. Following the proposal, there was considerable discussion concerning the occurrence of anemia below a PbB level of 80 pg/100 ml. A longitudinal study done by Tola showing lowering of the Hb levels at a PbB level of 50 pg/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 low Hb level 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. Basal levels of Hb in children may be significantly lower than in adults, and their PbB levels must be established for control purposes. The long-term health effects of changes in Hb levels in children must also be considered.

The longitudinal study done by Tola showed that PbB levels as low as 50 pg/100 ml result in a significant lowering of Hb levels in populations with blood lead levels as low as 50 pg/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/100ml 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 pg/100 ml or above would over a working lifetime lower 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 work load. 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 can occur within a population at PbB levels of 50 to 70 pg/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.

I. Effects on Mitochondrial Function. As was previously discussed, the heme radical is not only a constituent of the hemoglobin molecule, but also a component of mitochondrial respiratory proteins called cytochromes. The effect of lead on heme synthesis 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 mitochondrial effects caused by inhibited heme synthesis is limited, but the implication of such disruptions is 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.

Most of the clinical effects of lead poisoning are due to the fact that PbB levels for most employees are above 30 pg/100 ml. The longitudinal study done by Tola showed that PbB levels as low as 50 pg/100 ml result in a significant lowering of Hb levels in populations with blood lead levels as low as 50 pg/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/100ml 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 pg/100 ml or above would over a working lifetime lower 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 work load. 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.

There is evidence of heme synthesis inhibition and mitochondrial disruption in other tissues of the body. Secci et al. (Ex. 5(223)) 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 pg/100 ml.

In his testimony, Piomelli used a study on the livers of rats which had been exposed to lead concentrations of 35 to 40 pg/100 ml. Lead granules were observed in the mitochondria by electron microscopy. (Tr. 459, Ref. Walton, 1972) 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 PbB levels in rats with PbB levels of 50 pg/100 ml, which 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 the induced mechanism may be a part of the physiological mechanism by which these tissues are affected. Plomell has specified that mitochondrial disruption resulting in altered cellular respiration may be a mechanism that leads to 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 respire.

Dissociated tissues are more or less sensitive to lack of oxygen. For instance, it is very well known that the brain and nervous system are more 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 well known sensitiveness is tied onto the entire organ, but it also occurs at the level of individual cells. The effects of lead on heme synthesis and its sequelae to the nervous system are particularly sensitive to lack of oxygen. As I have mentioned before, when an individual is poisoned with cyanide, 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)

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 these 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. The 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 ZPP 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 study the occurrence 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, 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 noted a connection between PbB level and the occurrence of various symptoms. Bertic (Ex. 69), found lead colic occurring in patients whose PbB levels were in the range of 40 to 80 μg/100 ml. Sakural (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 is best illustrated in rat liver cells 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.

Plomell 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 signs of damage to the brain and nervous system feels weak. However, it is not any longer possible to restrict the concept of health to the individual's subjective well-being. An amusing adverse effect is this 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 multiplicity of 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 mechanisms. An appraisal 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 earliest 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 the early neuropathy, 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-
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 was no 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 Seppäläinen 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 a neurological damage caused by increased lead absorption in the subclinical range is reversible and whether there is a continuing damage of the nervous system in workers with blood lead levels below, as well as above, 70 μg/100 g. Based upon these data, Seppäläinen 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. (Ref. 4663-82.)

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 constitute 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 neurobehavioral effects which were apparent in the study conducted by Seppäläinen 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 demonstrated 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 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 understood, 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 oligodendroglia and accumulations of PAS-positive globules in the perivascular glial cells. (Tr. 106.) At other times, there will be a diffuse atrophy of the brain tissue. Symptoms have been described 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, limits oxygen diffusion and shut off the blood supply. In fact, some neuropathologists 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).) Indeed, Ch. 11, Ref. 198; Ex. 95, Ref. Cantarow, p. 121). In certain cases, encephalopathy may include impairment of muscular coordination—the cerebellum—so 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. 11-15).

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 incid
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 there have been found levels 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. 23(15); EPA Criteria Document, Ch. 11, Ref. 453) Such a variability 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 of blood lead levels in adults suffering from encephalopathy.

It is important to note that the neuro-pathological findings as reported are essentially the same for adults and children. It has been concluded by thorough study that the recognition of effects at lower levels in children 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 significance for physicians in terms of survival and severity. Patients who are diagnosed as "severe" are usually suffering from epileptic-like seizures or coma—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 meningial 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. 168) Coupled with this, 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 encephalopathy. (Tr. 146) Other clinical manifestations are also less severe, anemia and colic are not necessarily present.

In cases of mild encephalopathy the neurological damage found has 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 neuro-behavioral 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 contains mainly 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(10))

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 "minimal" level of lead intake. However, 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. 1976-78) While it is not argued that all levels of lead are sufficient to discuss the evidence relating to the significance of neurobehavioral effects at low level 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, as is true in adults, 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 hematopoletic 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-
The mean age of the workers was 32.5 years (range: 18-39 years). Healthy unexposed subjects of the same educational level and age range formed a control group (N = 50), and two groups with CS exposure (50+50), by exposure limits for lead. The two groups had PbB levels in excess of 70 μg/100 ml. Blood lead levels were available for 33 subjects and ranged from 60 to 150 μg/100 ml. These authors were able to demonstrate strong indications of CNS dysfunction. Studies of six of the nine workers, which measured intelligence, speed of performances, psychomotor functions and features of emotional reactions, were used as a measure of behavior. Compared with the exposed group, and the other "healthy" exposed workers, were used as additional reference groups. All subjects were examined with a large battery of tests designed to measure intelligence, speed of performances, psychomotor functions and features of emotional reactions.

Controlled with the exposed group (N = 50), the lead workers were significantly inferior in two tests of intelligence and in several tasks that demonstrated control of psychomotor functions. In the personality test they showed less emotional reactivity but nevertheless more loss of rational control of behavior than the control group. Indications of CNS dysfunction were quantitatively milder than in the group with CS poisoning but more severe than in the group with CS exposure but without poisoning. Qualitatively, the lead syndrome differed from the effects of CS 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 control group. The first function discriminated the groups on the basis of the severity of the CNS dysfunction, and the second function yielded an optimal separation between the lead group and the control group with CS poisoning. When each subject was classified to the group to which they were most likely to belong according to his discriminant function values, 89 percent of the lead workers were classified into their proper group, 22 percent into the group with CS poisoning, 12 percent into the "healthy" exposed group, and 7 percent into the control group. When the sum of the probable errors of the discriminant function values was used as a measure of CNS dysfunction, there was no correlation between the dysfunction and the PbB levels. Six of the subjects with a PbB below 70 μg/100 ml had strong indications of CNS dysfunction. Although the causal connection between PbB 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. 7-9)

The study serves as an early indication that behavioral effects may be occurring at reduced blood lead levels. Crockford and Mirman (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 performed to determine if the performance of the lead exposed workers were inferior to that of the control group. The population was the same as that reported by Lee and coworkers (see section on peripheral nervous system). When a discriminant analysis was performed between the four groups two of the three discriminant functions were relevant with respect to the differences between the exposed group and the control group. The population was the same as that reported by Lee and coworkers (see section on peripheral nervous system). The exposed workers appear to show a preponderance 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 Fischbel 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 follow up 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 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 toxicity, aggres-
son, 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 µg/100 ml and 90 µg/100 ml. (Ex. 225, p. 65)

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 functions assessed through the use of the multiple task performance battery, the test of visual acuity and the digit span test." (Ex. 52, p. 10) Repko acknowledged that while certain measures of performance obtained from the lead-exposed subjects showed 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 accident or noise exposure may affect the 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 as 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 analysis 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, as the data are not statistically significant 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 procedures. PbB levels in 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 of Inorganic lead absorption, a clinical electromyogram, a clinical neurological examination and demographic data were obtained from 65 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 levels which had been consistently below 80 µg/100 ml and were asymptomatic upon examination.

Repkos 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 6 m/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 (CVSP) 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 are 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.

Rekopos concluded: "It is clear from the present study data that PbB 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, 45 percent were statistically significant. These data are further enhanced by the results from the tone-decay test which demonstrates 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 lead 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 lead-exposed workers showed a statistically significant increase in reaction time test requires control by the ulnar nerve. The ulnar nerve is the 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 neurological dysfunctions noted in this study and in other epidemiologic studies are contributed to by lead exposure, even at PbB levels substantially lower than PbB 70 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: (Tas the primary motor nerve responsible for lateral movement of the fifth finger). (Ex. 422 (A), p. 63) This
Peripheral neuropathy manifested by weakness of extensor muscles of wrists and/or fingers was discovered in 19 workers. Six of the workers with extensor muscle weakness had less than 10 years of lead exposure whereas thirteen had less than 10 years exposure. The blood lead level exceeded 60 μg/100 ml in 14 cases and in 7, the level was greater than 120 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 to be typically less than 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 69.1 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. While this value of 60.1 m/sec for the control group had 55.3/sec as the ZNCV less than 55 m/sec. The prevalence of reduced nerve conduction velocities was 25 percent in the control population. While this value of 25 percent appeared 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 the prevalence of symptoms correlated with work to be discussed later for two reasons: first the PbB of the control group was 25 percent (42 percent) compared to 12 percent for controls, second the PbB of the control population was also subject to lead exposure. The prevalence was significant for two reasons: first the PbB of the control group was 25 percent (42 percent) compared to 12 percent for controls, second the PbB of the control population was also subject to lead exposure. Nevertheless, the authors reported that tiredness, fatigue, nervousness, sleeplessness or somnolence, and anxiety existed in a large percent of the previously described total population who had PbB of less than 80 μg/100 g had 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. In a follow-up evaluation Lillis et al. described their findings in a subgroup of the previously described total population who had PbB of less than 80 μg/100 g 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 treatment. 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 15 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 ZPP was 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.
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, 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, upper joint or joint pain were found with increased prevalence. This may be related, in some degree, to the rate of buildup of the lead 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 consistently found 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 gives more sensitive estimate of biologically active lead than simple blood lead examinations, as they show a good correlation with clinical abnormalities.

Dr. Lillis 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 concluded that symptoms related to lead toxicity were less prevalent in the Vernon group than in Indianapolis reflecting the better control of lead in the former case. CNS symptoms were found in 64 percent of this group as compared to 31 percent in the previous group; only 20 percent of the workers 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 chelation therapy and only 6 (5.5 percent) had been given repeated courses of treatment. The practice was 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 of 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 80 μg/100 ml or higher and in 20 percent the levels had exceeded 80 μg/100 ml.

Muscle and joint pain and/or soreness were the most 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 52 percent had lead levels of 80 μg/100 ml or higher 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 levels. (Cont.)
level or lead intoxication from his test scores alone.

This study is based on a group of workers whose blood lead and performance levels indicate that a portion of this population meets the clinical definition of lead intoxication. Erythrocyte protoporphyrin 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 able person might gravitate towards dirtier, less desirable jobs. In both Drs. Vailelus and Lillis' 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>
<thead>
<tr>
<th>Variable</th>
<th>Exposed group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(N=49) Mean SD</td>
<td>(N=24) Mean SD</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Similarities</td>
<td>17.7 ± 3.3</td>
<td>19.4 ± 2.3</td>
</tr>
<tr>
<td>Picture completion (PC)</td>
<td>5.2 ± 3.2</td>
<td>15.3 ± 5.9</td>
</tr>
<tr>
<td>Block design (BD)</td>
<td>34.7 ± 5.0</td>
<td>33.5 ± 7.7</td>
</tr>
<tr>
<td>Digit span (DPS)</td>
<td>9.6 ± 2.5</td>
<td>10.6 ± 2.5</td>
</tr>
<tr>
<td>Logical memory (LogM)</td>
<td>10.8 ± 3.4</td>
<td>12.1 ± 4.2</td>
</tr>
<tr>
<td>Visual reproduction (Vis R)</td>
<td>10.9 ± 2.5</td>
<td>12.1 ± 2.7</td>
</tr>
<tr>
<td>Bourdon Wiersma, errors (BW err)</td>
<td>22.4 ± 25.7</td>
<td>18.5 ± 12.3</td>
</tr>
<tr>
<td>Benton, time (Ben time)</td>
<td>14.0 ± 5.5</td>
<td>15.3 ± 6.0</td>
</tr>
<tr>
<td>Benton, errors (Ben err)</td>
<td>1.26 ± 2.12</td>
<td>1.91 ± 2.15</td>
</tr>
<tr>
<td>Santa Ana, preferred hand (SA right)</td>
<td>45.6 ± 6.1</td>
<td>46.3 ± 7.1</td>
</tr>
<tr>
<td>Santa Ana, left hand (SA left)</td>
<td>42.1 ± 5.5</td>
<td>41.5 ± 6.8</td>
</tr>
<tr>
<td>Santa Ana, coordination (SA Coord)</td>
<td>30.1 ± 5.7</td>
<td>27.4 ± 6.2</td>
</tr>
<tr>
<td>Simple reaction time, preferred hand</td>
<td>1,482 ± 202</td>
<td>1,446 ± 313</td>
</tr>
<tr>
<td>Simple reaction time, left hand</td>
<td>1,385 ± 241</td>
<td>1,350 ± 251</td>
</tr>
<tr>
<td>Choice reaction time (CERT)</td>
<td>1,711 ± 268</td>
<td>1,739 ± 226</td>
</tr>
</tbody>
</table>

* 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 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 levels of PbB 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. 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.

The extensive research carried out at secondary smelters and reported by Lillis, Fischbein et al. was uncontested during the public hearings. Given the soundness of methodology and number of parameters evaluated, these authors speculate that 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 study documents some of the central nervous system disorders in lead exposed workers whose PbB levels were below 80 μg/100 ml. Dr. Lillis testified on this point of view during the public hearings:

Mr. Kubenecker. 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. Lillis. 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, and 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 the protoporphrin 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, 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

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such extensor paralysis does consistently appear in the muscles controlling the hands and feet, it has become synonymous with lead poisoning. (Ex. 294, Ref. Cantarow, pp. 77-78). 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." Depressed 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, which may result in irreversible atrophy of the extensor muscles. (Ex. 95, Ref. Cantarow, pp. 126.)

If lead exposure continues, and the lead poisoning is not treated, exposure reduced, weakness may eventually extend throughout the arm or leg. Prior to the development of paralysis, workers have been known to complain of hyperesthesia over the affected areas, which is often accompanied 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 possible the 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 (cytoids and/or axons) or combinations of both.

Axonal atrophy, and segmental demyelination, and changes in the Schwann 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 PbB levels 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 to occur. (Ex. 270 (76).) 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), 127). 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 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 not only partially irreversible, if at all, with chelation therapy and/or removal from further exposure. This is 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. 270 (76).)

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.

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OSHA RULES AND REGULATIONS

RC.95, Ref. Cantarow, pp. 65; Ex. 24, (19); Tr. 105; Ex. 95, pp. 89.)

Related to the pathological alterations are a progressive loss of strength in the extensor muscles to the point of total paralysis. Because...
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. Of the 19 men examined 13 had blood levels above 60 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 stimulating the median and ulnar peripheral 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 difference in 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 the exposed group which was comprised of 26 workers (18 men and 8 women) from a storage battery factory who had a mean exposure time of 4.6 (SD 4.7, median 3.7 years, range 13 months to 17 years). The concentration of PbB 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 median nerve as compared to an age matched control group. The mean conduction velocity of the slower fibers of the median nerve was 30.0±8.0 whereas the control population was 46.5±3.7 (p is less than 0.001). Based on this result, the authors conclude that measurement of the slower fibers (CVSF) of the median 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, the conclusion that 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 20 workers (18 males and 2 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 28 workers (18 men and 8 women) from a storage battery factory who had a mean exposure time of 4.6 (SD 4.7, median 3.7 years, range 13 months to 17 years). The concentration of PbB 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.

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

<table>
<thead>
<tr>
<th></th>
<th>Exposed</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
</tr>
<tr>
<td>MCV* of median nerve</td>
<td>26</td>
<td>55.4</td>
</tr>
<tr>
<td>SCV† of median nerve</td>
<td>25</td>
<td>59.5</td>
</tr>
<tr>
<td>MCV* of ulnar nerve</td>
<td>26</td>
<td>55.0</td>
</tr>
<tr>
<td>CVSF‡ of ulnar nerve</td>
<td>26</td>
<td>42.0</td>
</tr>
<tr>
<td>SCV of ulnar nerve</td>
<td>25</td>
<td>58.2</td>
</tr>
<tr>
<td>MCV* of deep peroneal nerve</td>
<td>25</td>
<td>50.6</td>
</tr>
<tr>
<td>MCV* of posterior tibial nerve</td>
<td>26</td>
<td>43.3</td>
</tr>
</tbody>
</table>

*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 poisoning; EMG was abnormal in 24 subjects, and denervation activity (fibrillations) was found in 15 cases. Among lead exposed workers with PbB 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 previously 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 home 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 serious effect than the quite reversible alterations in nervous system should be accepted, and found in

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, Re-evaluation 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 PbB 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.)
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 thus 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 is a defined point in the relationship between dose and effect in the individual, namely the point at which an effect occurs. The control group and the lead-exposed subjects were tested at different times. Lead Industries Association. In her book, she found no evidence that exposure had not been higher in the past than during the period of frequent monitoring. (Ex. 5, p. 12.)

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 PbB readings 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 60-69 µg/100 ml.

The nerves with reduced NCV's included the median, ulnar, and CVSP of the median nerve, and the CVSP 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. 23.)

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 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 subject of extensive debate. However, in OSHA's view there is no evidence that exposure had not been higher in the past than during the period of frequent monitoring. (Ex. 5, p. 12.)

In addition, there is no evidence in subsequent studies 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 his 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. 12.)
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 much affected by differences in temperature and proposed the use of a corrected method for determining temperatures.

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 justified 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. They found that 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 velocities 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 measures and nerve conduction velocities. The data as analyzed by multiple regression analysis did indicate that length of exposure was significantly correlated (p < 0.05) with upper nerve conduction velocities. 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 increased 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 was increased. While 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—55.3 ± 4.3 M/sec, Payan, 59 ± 5.5 M/sec. The percent mean conduction velocity difference in Lee's controls vs. lead workers in ulnar, median and peroneal nerves was 4.5 percent in the radial nerve conduction velocity was reduced by 13% in workers and controls. Again, in the peroneal nerve, the ratio of the mean of the standard deviation of external 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 the foot reduces, plus assumed difference in temperature here would invalidate the significance of the finding. (Ex. 234 (21), Ref. Steiner, p. 9.)

These issues were not addressed during the hearings. OSHA believes that the findings of 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 (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 8-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 median nerve of each child and the results indicated a statistically significant negative correlation between personal nerve conduction velocity and blood lead level.

\[ r = -0.38, \ t = 2.12, \ p < 0.02 \]

In a letter to the editor of the Journal of Pediatrics, September 1977, Gartside and Panke critiqued the findings of Landigran 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 correlation 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 Landigran 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 cleric females exhibited superior performance on this particular test.

(5) No pattern of hostility, depression, or anxiety is evident in either male or female smoker 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 ($p = .06$). No pattern was evident regarding an association between RC mean and any independent variable other than age.

Nerve conduction velocities are consistently lower in smoker employees than controls but the decreases are small, and achieved statistical significance only for the peroneal nerve in female smoker workers and male zinc smoker 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 smoker workers than for male lead smoker 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 smoker workers. However, "dose-effect" relations are not evident, since male lead 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 smoker workers and control groups. However, if published norms are used as a basis for comparison, the following results are obtained:

(1) Production males 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. = 282, $p$ is less than .01; $t = 2.48$, d.f. = 292, $p$ is .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 $\mu$g/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 test 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 cumulative toxic effects 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 employed in the track maintenance, haematocrit, hemoglobin, protoporphyprin and calculated PEP concentrations as well as motor nerve conduction velocity of the peroneal nerve. Both groups of workers wore respirator 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 45.2 and the six nonburners was 49.0 as compared to the control mean of 54.69 m/sec ± 5.56. This study demonstrates again the relationship between neuropathy and motor conduction velocities, and indicates slowing of MNCV at blood lead levels well below 80 $\mu$g/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

<table>
<thead>
<tr>
<th>Patient</th>
<th>Motor Nerve Conduction Velocity, M/sec</th>
<th>Screening Blood Lead Level, ug 100 gm</th>
<th>Before Edetate Disodium Calcium Therapy, ug/Liter</th>
<th>24-Hour Urine Lead Excretion Level</th>
<th>u mole of Lead/u mole of Edetate Disodium Calcium</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>43.5</td>
<td>23</td>
<td>10</td>
<td>260</td>
<td>0.094</td>
</tr>
<tr>
<td>6</td>
<td>45.0</td>
<td>14</td>
<td>20</td>
<td>180</td>
<td>0.152</td>
</tr>
<tr>
<td>7</td>
<td>38.5</td>
<td>23</td>
<td>10</td>
<td>700</td>
<td>0.422</td>
</tr>
<tr>
<td>15</td>
<td>38.5</td>
<td>17</td>
<td>20</td>
<td>50</td>
<td>0.076</td>
</tr>
<tr>
<td>20</td>
<td>31.0</td>
<td>41</td>
<td>10</td>
<td>683</td>
<td>0.566</td>
</tr>
<tr>
<td>21</td>
<td>38.0</td>
<td>33</td>
<td>16</td>
<td>1,036</td>
<td>0.957</td>
</tr>
<tr>
<td>22</td>
<td>40.5</td>
<td>83</td>
<td>14</td>
<td>695</td>
<td>0.297</td>
</tr>
<tr>
<td>25</td>
<td>44.5</td>
<td>41</td>
<td>--</td>
<td>Not chelated</td>
<td>-----</td>
</tr>
<tr>
<td>17</td>
<td>40.0</td>
<td>22</td>
<td>--</td>
<td>Not chelated</td>
<td>-----</td>
</tr>
<tr>
<td>10</td>
<td>47.5</td>
<td>29</td>
<td>--</td>
<td>Not chelated</td>
<td>-----</td>
</tr>
</tbody>
</table>
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 .01). The 24-hour collections before and during edetate disodium calcium provocations of lead show no significant deviations from the usual levels in urine and blood. The authors conclude:

Although the urine concentrations of lead in children with neurological damage are significantly raised when compared with controls and with lead workers, the authors stress that the research cited has withstood the test of time. The evidence is insufficient to determine whether the effects of lead exposure are similar in children and adults. The authors also note that the findings bear on the possibility of increased body lead burden, a possible relationship exists between the MnCCV slowing and the quantity of lead excreted in the urine after edetate disodium calcium provocations. The four children with provoked urine excretion of greater than 800 µg of lead all had MnCCV reductions 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 those showed lead concentrations in urine exceeding the mean for control subjects.

The individual differences in MnCCV values among the 13 risk groups and control subjects were not at issue in many of the studies that have gathered, it becomes clear that subclinical neuropathy, measured by nerve conduction velocity, is not increased in children exceeding the mean for control subjects.

Based on the studies described in detail and others only referenced, OSHA concludes that 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 levels are 50 µg/100 ml or greater. OSHA also noted that the slowing of MnCCV 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, particularly 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 have also shown MnCCV reduction in lead exposed workers and others.

The principal issue raised with respect to the reduction of MnCCV 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 by, or accompanied by, the disease progression by a relatively mild, apparently reversible stage of disease which is characterized by the MnCCV reductions described herein. Based upon the voluminous evidence in the record concerning MnCCV reduction OSHA believes that these reductions do constitute material impairment and do represent early indicators of a neurological disease process. The existing state is characterized by varying subjective and/or objective symptoms that may not at first unambiguously 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 such a pathological condition. 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 MnCCV were of concern for several reasons: (Tr. 1885, 1903, 2108, 3040, 6577). However, industry testimony failed to dispute the significance of these acute effects as manifestations of neurological disease which, with continued exposure, may progress to clinical neuropathy. The same cannot be said of the MnCCV reductions described in the record. 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 tissues.

OSHA notes 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 morphological anomaly 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 segmentation 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 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, and, in general, a slight decrease 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 areas" 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. 512, 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 shows slight evidences of demyelinating neuropathy, in many of them showed changes of denervation type. Furthermore, nervous tissue is slow and at times incomplete 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. However, this slowing should not be regarded as an early indicator of clinical neuropathy. The same phenomenon is a possibility which has to be kept in mind when dealing with human exposure to lead. When nerve conduction velocity shows abnormally slow nerve conduction velocities in two nerves, better histological alterations are also considered as being suspect of increased body lead burden, a possible relationship exists between the MnCCV slowing and the quantity of lead excreted in the urine after edetate disodium calcium provocations. The four children with provoked urine excretion of greater than 800 µg of lead all had MnCCV reductions 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 those showed lead concentrations in urine exceeding the mean for control subjects.
system is safe, since the pathological signs in the hematopoietic system appear at significantly higher Pb B levels than nervous system signs. (Ex. 5, p. 5)

Dr. Kenneth Brind' of NIOSH divided response to environmental exposure into five categories: normal, physiologic change of uncertain significance, pathophysiologic change, morbidity (fairly severe disease), mortality. (Tr. 1785-1801). He places nerve conduction 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 look for, would be the decreased nerve conduction velocities in which case, in adults we begin to see this as blood lead levels rise above 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 vary is fairly limited. That's not to say that there couldn't be some reversion 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 changes in the pathophysiologic category. 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 be fairly 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 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.

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The record of this proceeding contains numerous examples of clinical symptoms behavioral changes and re-...
The pathogenesis of lead-induced nephropathy has long been debated. A sequence of histological changes extending from the occurrence of lead-induced intranuclear inclusion bodies to diffuse interstitial fibrosis and renal failure has been described in animal studies by Goyer. 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 response 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, and it is increasingly more difficult 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 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, Lills 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 creatinine and persistent urea retention, which usually was accompanied by high blood pressure, developed in those workers who were chronically poisoned. Lills et al. concluded that the functional impairment resulted from a marked vasoconstriction of the renal blood vessels (perhaps indicating the generalized vasoconstriction 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 urinalysis 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 Lillis et al., where 18 cases of clinically verified chronic neuropathy were found, the mean blood lead level was approximately 80 μg/dl, with a range of 42 to 141 μg/dl for the whole series. (Ex. 118E.)

Another study by Lillis 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 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. 96, 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 Lillis, Fischbein et al. reported the results of a clinical field study from two secondary lead smelters during the rulemaking hearing. They examined 168 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>
<thead>
<tr>
<th>Duration of lead exposure</th>
<th>Number examined</th>
<th>BUN levels</th>
<th>&gt;21 mg/100 ml</th>
<th>&gt;25 mg/100 ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 10 yr.</td>
<td>137</td>
<td>Number</td>
<td>19</td>
<td>13</td>
</tr>
<tr>
<td>More than 10 yr.</td>
<td>20</td>
<td>Percent</td>
<td>11</td>
<td>55</td>
</tr>
<tr>
<td>Total</td>
<td>157</td>
<td></td>
<td>29</td>
<td>18</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Duration of lead exposure</th>
<th>Number examined</th>
<th>Creatinine levels</th>
<th>&gt;1.2 mg/100 ml</th>
<th>&gt;1.4 mg/100 ml</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 10 yr.</td>
<td>137</td>
<td>Number</td>
<td>19</td>
<td>14</td>
</tr>
<tr>
<td>More than 10 yr.</td>
<td>20</td>
<td>Percent</td>
<td>9</td>
<td>43</td>
</tr>
<tr>
<td>Total</td>
<td>157</td>
<td></td>
<td>28</td>
<td>18</td>
</tr>
</tbody>
</table>

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 (Lillis, 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 85 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 (Lillis, 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
Evidence has been reported concerning the development of nephropathy 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 possibility is vasoconstriction affecting the afferent renal arterioles predominantly. (Ex. 24 (Lillis 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 30 to 135 mg/100 ml, with 44 (83 percent) greater than or equal to 60 mg/100 ml and 19 (36 percent) greater than or equal to 80 mg/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

<table>
<thead>
<tr>
<th>Worker numbers</th>
<th>BUN</th>
<th>Creatinine</th>
<th>Years employed</th>
<th>Number of courses of EDTA</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>March</td>
<td>May</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>—</td>
<td>44</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>26</td>
<td>30</td>
<td>1.1</td>
<td>23</td>
</tr>
<tr>
<td>3</td>
<td>20</td>
<td>28</td>
<td>1.2</td>
<td>23</td>
</tr>
<tr>
<td>4</td>
<td>26</td>
<td>24</td>
<td>1.3</td>
<td>7</td>
</tr>
<tr>
<td>5</td>
<td>21</td>
<td>27</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>23</td>
<td>27</td>
<td>1.0</td>
<td>13</td>
</tr>
<tr>
<td>7</td>
<td>21</td>
<td>23</td>
<td>1.2</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>25</td>
<td>—</td>
<td>1.2</td>
<td>0</td>
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<tr>
<td>9</td>
<td>23</td>
<td>—</td>
<td>1.2</td>
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<tr>
<td>10</td>
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<td>1.0</td>
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<tr>
<td>11</td>
<td>23</td>
<td>—</td>
<td>1.0</td>
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<td>15</td>
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<td>1.2</td>
<td>31</td>
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<td>16</td>
<td>20</td>
<td>23</td>
<td>1.4</td>
<td>12</td>
</tr>
<tr>
<td>17</td>
<td>16</td>
<td>24</td>
<td>1.2</td>
<td>20</td>
</tr>
</tbody>
</table>

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. 60)

Based upon the BUN results in this earlier study NIOSH conducted a follow-up 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 exposure rate. (Table 3)

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

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Duration of lead exposure (years)</th>
<th>Blood lead level (μg/100 ml)</th>
<th>Creatinine clearance (ml/min/1.73 m²)</th>
<th>Urine amylase activity (units/liter)</th>
<th>Lead clearance rate (ml/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>55</td>
<td>7</td>
<td>154</td>
<td>85</td>
<td>0.87</td>
<td>0.07</td>
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<tr>
<td>2</td>
<td>43</td>
<td>20</td>
<td>66</td>
<td>142</td>
<td>0.82</td>
<td>0.82</td>
</tr>
<tr>
<td>3</td>
<td>37</td>
<td>20</td>
<td>35</td>
<td>62</td>
<td>1.48</td>
<td>1.48</td>
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<td>4</td>
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<td>71</td>
<td>72</td>
<td>1.10</td>
<td>1.10</td>
</tr>
<tr>
<td>5</td>
<td>45</td>
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<td>128</td>
<td>1.35</td>
<td>1.35</td>
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<tr>
<td>6</td>
<td>33</td>
<td>23</td>
<td>61</td>
<td>91</td>
<td>2.05</td>
<td>2.05</td>
</tr>
<tr>
<td>7</td>
<td>52</td>
<td>25</td>
<td>61</td>
<td>115</td>
<td>2.50</td>
<td>2.50</td>
</tr>
<tr>
<td>8</td>
<td>38</td>
<td>29</td>
<td>123</td>
<td>109</td>
<td>2.08</td>
<td>2.08</td>
</tr>
<tr>
<td>9</td>
<td>42</td>
<td>21</td>
<td>65</td>
<td>96</td>
<td>1.60</td>
<td>1.60</td>
</tr>
<tr>
<td>10</td>
<td>41</td>
<td>48</td>
<td>108</td>
<td>820</td>
<td>1.29</td>
<td>1.29</td>
</tr>
<tr>
<td>11</td>
<td>42</td>
<td>25</td>
<td>24</td>
<td>89</td>
<td>1.00</td>
<td>1.00</td>
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<td>12</td>
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<td>13</td>
<td>56</td>
<td>109</td>
<td>1.33</td>
<td>1.33</td>
</tr>
<tr>
<td>13</td>
<td>62</td>
<td>18</td>
<td>73</td>
<td>77</td>
<td>1.01</td>
<td>1.01</td>
</tr>
<tr>
<td>14</td>
<td>55</td>
<td>28</td>
<td>58</td>
<td>108</td>
<td>1.25</td>
<td>1.25</td>
</tr>
<tr>
<td>15</td>
<td>33</td>
<td>20</td>
<td>92</td>
<td>105</td>
<td>1.04</td>
<td>1.04</td>
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<tr>
<td>16</td>
<td>51</td>
<td>45</td>
<td>84</td>
<td>105</td>
<td>2.04</td>
<td>2.04</td>
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<td>55</td>
<td>43</td>
<td>65</td>
<td>0.80</td>
<td>0.80</td>
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<tr>
<td>18</td>
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<td>4.5</td>
<td>58</td>
<td>112</td>
<td>1.14</td>
<td>1.14</td>
</tr>
</tbody>
</table>

Normal range: 60–91 μg/100 ml for blood lead level, 50–1,300 ml/min/1.73 m² for creatinine clearance, and 10–40 units/liter for urine amylase activity.

**Rules and Regulations**

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 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 a plant that fired ranges, and one prepared solder cans 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:

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

2. 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 complicated lead nephropathy, the urinalysis is usually entirely normal.

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 dialysis 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. 1737–39)

In these 18 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 (5) 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-
The blood lead levels of these workers is leading, 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 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 where 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 a kidney to obvious limitations in activity. Loss of a kidney or an equivalent loss of kidney function means the lead worker is unable 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. 1744-1760.)

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 line in 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. 1744-1760.)

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It can therefore be anticipated that at least 10 percent of the American lead workers have occupational lead nephropathy. This line in 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. 1744-1760.)
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 this, 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 blood lead level on that worker and having some information about 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 idea 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 lead 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 continuously 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 60, for 2 years, may be very similar to what the 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 when looking at the patient, very few very many 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 physicians 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 appears to agree 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 particular I would like to mention hypertension, a very important problem, well recognized in this country.

Hypertension 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 possible excess mortality from hypertensive vascular disease in the battery manufacturing population as well. The Mt. Sinai group also noted in increased prevalence 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. Physicians may exclude a diagnosis of lead nephropathy because of the lack of clinical indicators. This compels the physician to make a diagnosis of essential hypertension unrelated to lead. OSHA believes that hypertension and renal disease are confounding. 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 single parameter", but rather the reduction in GFR was supported by comparable reductions in effective renal plasma flow (ERPF). In nine patients, the diagnosis of lead nephropathy was confirmed and other possible etiologies further excluded by renal biopsy.

As a result 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. As we noted in medical studies, loss of function is commonly considered disease. Moreover, many diseases are relatively asymptomatic, particularly early phases, e.g., hypertension, diabetes, etc.

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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 satisfactorily with less than 50 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. 237, 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 his experience in 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.

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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:

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

(2) Amonolayer changes, atrophic damage and loss of proximal tubular brush borders were absent in all but the most severe case.

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

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

(5) Aminocadura 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 8, No. 7 (1): Re "Intranuclear inclusions." The disappearance of intranuclear inclusions in lead nephropathy has been noted by et al. in some subjects (Brit J Indust Med 31:113, 1974), and by Goyer in experimental animals (Lab Invest 32:149, 1975).

Ibid (2): Histologic changes in proximal tubules consistent with lead nephropathy were present in all ten biopsied kidneys.

Ibid (3): The absence of multiple renal defects detectable by clearance methodology undoubtedly reflects only the limitations of this physiologic technique.

Ibid (4): The PAH transport defect may be a transient phenomenon peculiar to acute lead poisoning.

Ibid (5): Amonolayer has been demonstrated only in large groups of lead exposed individuals compared to unexposed subjects.

Ibid (6): Normal renal concentrating ability is characteristic of early lead nephropathy and helps distinguish this disease from other renal disease. (Ex. 218, 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. 217, pp. 1-5)

Hine:
On page 8 of his presentation, Dr. Wedeen refers to measurements of GFR in 41 unselected lead workers. Dr. Wedeen and coworkers have presented important data that demonstrate that lead induced renal disease is prevalent. This is in contrast to the previously held positions that lead nephropathy or any other lead related kidney disease is rare, if not nonexistent. Dr. Wedeen has more than adequately supported the claim that lead induced renal disease is a common cause of end-stage kidney disease. (Ex. 218A, p. 6)

Wedeen:
The importance of negative control data cannot be emphasized too strongly. (Ex. 218A, p. 6)

Hine:
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 excluding other possible causes of decreased renal function in these cases. (Ex. 217, pp. 2-3)

Page 8, No. 7 (2): Re "Intranuclear inclu­sions." The absence of multiple renal defects detectable by clearance methodology undoubtedly reflects only the limitations of this physiologic technique.

Page 8, No. 7 (3): Re "Intranuclear inclu­sions." The absence of multiple renal defects detectable by clearance methodology undoubtedly reflects only the limitations of this physiologic technique.

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Page 8, No. 7 (5): Re "Intranuclear inclu­sions." The absence of multiple renal defects detectable by clearance methodology undoubtedly reflects only the limitations of this physiologic technique.

Page 8, No. 7 (6): Re "Intranuclear inclu­sions." The absence of multiple renal defects detectable by clearance methodology undoubtedly reflects only the limitations of this physiologic technique.
CHRONOLOGY OF POTENTIAL ADVERSE EFFECTS OF
JOB EXPOSURES ON REPRODUCTION OR
ON THE ABILITY TO HAVE NORMAL,
HEALTHY CHILDREN

PRIOR TO CONCEPTION
- 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.

AT CONCEPTION
- Difficulties in conceiving a child (for example, by interference with sperm's ability to fertilize the egg)

DURING PREGNANCY
- Miscarriage, stillbirth, cancer, disease, or birth defects—as a result of substances crossing the mother's placenta and reaching the developing fetus

ON THE NEWBORN
- Toxic effects on development of baby as a result of chemicals transmitted to child in mother's breast milk

ON THE CHILD
- Toxic effects on development of child from exposure to substances inadvertently brought home on parents' work clothes

FIGURE 1.
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 paint industries who purchased lead paint felt lead poisoning as 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)

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 levels 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 more 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. In addition, OSHA has concluded that the fetus is not as well protected as the mother. 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 evidence of the effects of lead on reproduction and development. OSHA will follow the outline described by Hickox 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) post-natal and developmental.

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 menes, amenorrhea, dysmenorrhea 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. 594) 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 challenged, and 8 months later the left ovary was removed. Fetal development 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.
cessed. The sexual skin color developed 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 germ cells, 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 and conception failed to occur for 1-12 months to less than 140 female printshop workers, exposed to lead. The study of this phenomenon increased.

Panova reported a study on the reproduction of male and female textile workers (Ch. 11). In a group of 20-25-year-old workers, the percentage of abnormal spermatogenesis increased when blood lead levels were noted in the males; testicular hypertrophy and asthenospermia (decreased number of spermatozoa), teratospermia (malformed sperm), asthenospermia (decreased motility) and hypospermia (decreased number of spermatozoa) were found. 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 impotence. Dr. Ioana Lancranjan who demonstrated altered spermatogenesis with teratospermia (malformed sperm), asthenospermia (decreased motility) and hypospermia (decreased number of spermatozoa) and with increased absorption of lead by lead-poisoned rabbits also found reductions in litter size, in weights of offspring, and in survival rate.

Panova reported a study on the reproduction of male and female textile workers (Ch. 11, Ref. 354). The workers were grouped on the basis of clinical and toxicological test results. In the first group the workers questioned, 131 were married, 62 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 fatigue, and 10 complained of difficulties having children. A 22 percent increase in abnormal pregnancies was discovered after 62 of the workers' wives were administered the questionnaire, appearing suggestive of a relation 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) in an increase of about 50 percent, after initial occupational lead exposure (Ex. 233).

The most important study indicating paternal reproductive effects presented during oral testimony was that of Dr. Ioana Lancranjan, who demonstrated altered spermatogenesis with teratospermia (malformed sperm), asthenospermia (decreased motility) and hypospermia (decreased number of spermatozoa) and with increased absorption of lead by lead-poisoned rabbits. The workers questioned, 131 were married, 62 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 fatigue, and 10 complained of difficulties having children. A 22 percent increase in abnormal pregnancies was discovered after 62 of the workers' wives were administered the questionnaire, appearing suggestive of a relation to include their wives' miscarriages.
lead level). A control group of 25 men without occupational exposure was also included.

Lancerjan 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 (Lancerjan 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?

Ms. MILLER. And 50 percent were infertile?  
Ms. MILLER. What do you mean by infertile?

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

* p<.001 from 12) Lancranjan et al.
** p<.01
The data suggests a dose-response relationship for altered spermatogenesis and teratospermia. The abnormal spermatozoa included binucleated, bicellular, amorphous and tapered forms. Reversibility of the 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 germinai epithelium of the seminiferous tubules of the testes. The aim of this study was to establish a dose-exposure effect through interference with the hypophysal-ampitutary 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. The first criticism offered concerning the Lancranjan study, OSHA opposes. The LIA, on the other hand, supports Lancranjan's lead-exposed group was comprised 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 determinant to sexual functions. 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 condition of the children they 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 to 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 know such a declaration. I am not able to 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. 605-607).

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. 335, 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 teratosperma 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 recommendations 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 lead levels and the sperm studied. (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, asthenosperma, 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 this 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...
Ms. MILLER. What might such a level be?

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

Ms. MILLER. 30 micrograms?

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

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

Dr. LANCRAJAN. 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, Olner studied pregnancy outcome among the wives of males employed as house painters, many of whom suffered from lead colic. Of 467 deliveries, 23 per cent (107/467) were stillborn as compared to a stillbirth rate of 9 per cent in the entire town. (Ex. 28 [LancranJan et al.], Ref. 21.)

Lewin also reviewed the reproductive histories of "healthy" women who were married to lead workers. Out of 22 pregnancies, 6 were aborted, 12 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. 8).

It is 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. 23). 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 (Ref. 18 [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 teratogenic 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 [15], Ref. Forni et al.). Dr. LancranJan has suggested that percentage of early 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 determine the teratogenic 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 lymphocyte 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 AIA. Demonstrates that lead does induce human chromosomal changes in somatic cells.

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
cytes not only of workers with clinical lead poisoning but also of subjects with preclinical lead intoxication with no clinical symptoms 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 plumism is not evident these chromosomal changes may be present in a varying degree of concentration. Such decreased chromosomal alterations are present in cultured lymphocytes of workers exposed to lead for 18 months.

In a storage battery plant was carried out in one 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. PO-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 have taken place after some months of lead exposure.

While many studies demonstrate that lead can cause chromosomal abnormalities, a study by Sperling (Ex. 72, appendix 3 (5)) found no increased chromosomal abnormalities 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 shipbuilding 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-exposed group compared to the controls.

However, chromosome aberrations 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 disease. Therefore, we can suppose that such alterations might be present in large populations of workers exposed to lead (Ex. 6/53, p. 479).

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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 cell production or mortality at the subcellular level, 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, embriototoxic 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 limits these effects of lead from occurring.

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 (Ziellnus, Wibowo); Ibid., Ref. Fahim; Ibid., Ref. Haas; Ibid., Ref. Baglam; Ex. 95, Ref. 32; Ex. 60a; Ex. 233; Ex. 338). Correlation coefficients between lead in the umbilical cord and maternal blood lead levels have been reported as high as 0.94 (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 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 once 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 more 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 severe lead poisoning in women using salt as an abortifacient or severe industrial poisoning cases. The cases came to attention because of the death of the mother, and if not 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 injured 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 could be expected that 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).

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

While Dr. Hunt maintains that specific teratogenic effects cannot be demonstrated by the time the fetus is exposed to lead at birth, he suggests that amniotic fluid sampled at the time of birth may be used to study lead levels in the fetus. OSHA agrees with the conclusions of Dr. Hunt and his collaborators: 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 by the hematopoietic system. The indications of anemia include pallor, weakness, easy fatigability, and anemia may be overlooked. This symptom may also be misinterpreted or overinterpreted as indications of other conditions.

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 both in children (Ex. 95, Ref. 301) and adults (Ex. 6 (70)) at blood 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 precursor in the liver cells, ALA in serum, and ALA and CPP in urine.

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(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 65 μg/100 ml. In general, neurologic effects, including hyperactivity, behavioral disorders, 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 the 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(9)) 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 that the 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 μg/100 ml). Of 84 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 concluded that women exposed to a highly leaded water supply had an increased risk 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 35 normal subjects (25±8.9 vs. 20.5±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 this future defect.

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 little 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 elimination 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)).

OSHA further finds there is conclusive evidence that exposure of the fetus and infant to lead induces neurological damage manifested by behavioral disorders, motor conduction velocity decrements, deficiency in IQ, subjective CNS 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±9 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 Zahnert. (EPA Criteria Document, Ch. 11, Ref. 225.) Hyperactivity was found to be associated with exposure to lead. (EPA Criteria Document, Ch. 11, Ref. 236.) Baloh (Ex. 6(41)) 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 45 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 groups. Decreased nonverbal cognitive and perceptual motor skill decreases, as well as low grade motor neuropathy in children with blood lead levels of 40-65 μg/100 ml and a decrease in IQ, reading ability, and hyperactivity ratings failed to display any differences in the two groups.

Landssdown (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 levels over 40 μg/dl.

Another followup study on the same group (Ex. 6 (99), Ref. McNeill and Ptasnik) failed to demonstrate neurological and psychological abnormalities.
The evidence describing adverse effects from lead exposure to the fetus and newborn was uncontested 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, 1154-58; NOW 2678) (see also NIOSH 1321, 1892). 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 in the mother reaches 30 or 40 \( \mu g/100 \) g. (Exhibit 2, at 45930) (see also Lundquist 4506). (Ex. 335, p. 23.)

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.

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. 36.)

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 newborn. (Ex. 225.)

Scientific theory openly admits the difficulties involved in the extrapolation of these data to precise standards, such as the removal was, in the case of the newborn, the relative modulation of the pregnant woman and the unproven possibility of mobilization of lead from the skeleton during pregnancy. Nonetheless, the body of evidence from the hearings has convinced OSHA that blood lead levels must be kept below the 30 \( \mu g/100 \) ml range to adequately protect the developing fetus. (Tr. 6470; 71; 170; 1155-56; 4682; 605; 567; Tr. 647.) OSHA is mindful of the statement of the Center for Disease Control which considers blood lead levels of 30 \( \mu g/100 \) ml to be elevated in children. (Ex. 52 (15).) OSHA will consider the blood lead level of a pregnant woman to be elevated if it exceeds 30 \( \mu g/100 \) 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. 6464.) Blood lead levels 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 abnormalities in workers exposed to lead. Based on these studies, OSHA concludes that male exhibit lead-induced reproductive effects at 30 \( \mu g/100 \) 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 it has previously been the public health approach for females. (Ex. 27 (131, p. 10.)

Historically in many developed countries, "occupational exposure of adult females to lead is forbidden by law," (Ex. 24 (Zielhuis, Wirbouw), 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 demonstrates 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 progenitors 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 treatment of male employees 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 \( \mu g/100 \) ml is required to adequately protect the fetus.

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

6. Altered spermatogenesis, teratogenesis, atherosclerosis, and hypopituitarism are evident in workers exposed to lead. Blood lead levels of these workers were apparently as low as 30-40 \( \mu g/100 \) ml.

7. 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 40-50 \( \mu g/100 \) 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 both men and women of the number of chronic diseases, such as nephritis and other hypertensive diseases. Additionally, the standard mortality ratios (SMR's) observed for all causes of death in the smelter and battery workers were 107 and 89, 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 is generally 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 than those found. Cooper and Gaffey argue that while the mortality experience of the cohort is without foundation and is refuted by the data presented in the proposal, the mortality experience of the cohort is without foundation and is refuted by the data presented in the proposal.

Another critical factor in evaluating the 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 who were studied was ascertained at the end of 1970. Since nearly 1,400 of the 2,252 smelter worker deaths occurred before 1960 and nearly 1,760 of the 4,800 battery worker deaths occurred 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 limited 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 of 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. LILA 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. 45938.)"

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 subpart, 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 rests on the mortality study of lead workers carried out by Cooper, Tabershaw, and Gaffey. The study, as presented by the following 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 study 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 83 percent of the battery deaths. It is unfortunate that this plant alone was not evaluated in this study except from death certificates. The detailed information available through the company has not provided a good deal more, such as exposure levels, race distribution, etc.

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 before 1946 and 84 percent of the deaths occurred in workers employed greater than 10 years. As a result, the evaluation of cause of death by subperiod and 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 usefuly 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 calculation. 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 by exposure time and by 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 work categories is unlikely to be productive.

With these criticisms in mind, the study still deserves attention. A major question, however, is what 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 the magnitude and extent.

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 the shortcomings and the weakness of 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", there is a somewhat larger excess in smelter workers which is more strikingly distributed by employment history: those employed before 1946, SMR=118 and those employed after 1946, SMR=76; those employed less than 10 years, SMR=92; and those employed greater than 10, 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. The important fact, however, is that the 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=52; 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 distributed in a direction that is consistent.
with an association with work in smelters (see table 1).

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</table>

Other Hypertensive... | 475 | 238 | 314 | 400
Chronic and Unspecified and Nephritis, Renal Sclerosis | 258 | 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 mortality 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 types of lead exposure in 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=78), 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 subgroups 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 are unreported by the authors in the Final Report) 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 consistent with this observation 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 allow one to attribute to lead exposure an 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 the study population were from "Other Hypertensive Disease" and "Chronic Nephritis or Renal Sclerosis." Other conditions (e.g. acute and unspecified nephritis and renal sclerosis) are noted in the table as well, but it is clear that the excesses noted in the earlier reports are no longer a significant problem.

The overall impression of this study is that excess mortality from chronic renal disease and possibly excessive hypertensive vascular disease are seen in the study populations. There appears to be an increased risk of death associated with low lead exposure that 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 consistently have exposures under the permissible limit. However, OSHA reiterates, however, that mortality studies are notoriously insensitive measures of risk.

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

1. Air to blood relationship. The proposed lead standard reduces the permissible exposure limit from 200 µg/ m³ 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 µg/m³). 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 marker 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.

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 the 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 of physiological lead transport and the application of the Bernard Model for prediction of blood lead levels represents an accomplishment heretofore unseen in efforts 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. OSHA 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 level lead measurements to determine compliance with the permissible exposure limit. Further discussion of the permissible exposure limit is found in that section.

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. 5329) 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 and air lead levels by means of linear regression techniques. Note that this does not mean that only linear relationships were developed. The least squares technique was also applied to transformation of the variables, such as 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 that 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 and correctness of the independent variable, \( a \) 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 that the observed coefficients be different, 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 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 use a linear relationship observed between blood lead and air lead exposure and not 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 dependences 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 lead correlation than equation (2), attempting to fit the data to equation (2) will have the following effects: First, it will systematically bias the regression coefficients a and b, that is, it will make a and b inconsistent with the causal relationships 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 levels 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 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,
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but even for a more or less correct co-
efficient, its statistical significance
could be affected such that no rela-
tionship might be detected with a rea-
sible 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 in-
clude the effect of previous lead body
burden. In determining current blood
lead levels, as will be discussed in a
later section, there is much experi-
mental evidence to suggest that during
continuous exposure to lead, the lead
levels in various organs of the body in-
crease slowly over time, and that blood
lead levels probably never reach equi-
lbrium. Thus, if we attempt to predict
the blood lead levels for a sample of
workmen with different tenures, stud-
ies which did not include a term for
job tenure would overpredict blood
leads given a certain exposure for
workers with short employment histo-
ry, and underpredict blood leads for
workers with long job tenure. Regres-
sion lines, calculated from mixed-
tenure population would then have
lower slopes and larger intercepts at 0
exposure than would actually be ob-
served if job tenure were taken into
account.

Similarly, none of the studies includ-
ed 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 un-
derestimation of the slope coefficient
in the dependence of blood lead on
exposures.

There is little doubt that this kind
of specification error affects the ac-
curacy 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 10 $\mu g$/m$^2$/hr, an average blood lead
level of greater than 40 $\mu g$/100cc
would be observed (Ex. 234(422)). Such
a finding is clearly at odds with nu-
merous observations of blood lead
levels in populations without occupa-
tional lead exposures. It is clear that
the true lead in the environment is
under 25 $\mu g$/100g and is very probably
under 20 $\mu g$/100g for most areas. Stud-
ies which predict behaviors of this
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 much with pre-
dictions based upon a model to be
discussed below. This should not be
surprising, since the better studies pre-
sumably do give a fairly accurate pic-
ture of the effects of particulate expo-
sure 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 indu-
tries is probably one reason why many
of the studies generate apparent rela-
tionships between blood lead and air
lead that disagree. The application of
the Bernard model developed during
hearings on Medical Removal Protec-
tion and discussed below to error by the
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
$$\hat{\beta} = \frac{\beta \pm \sqrt{\frac{2}{\sigma^2} + \frac{1}{\sigma^2_v}} \cdot \text{var}(x)}{\text{var}(x)},$$
where $\sigma^2$ is the variance of the measure-
ment error, $\sigma^2_v$ the variance of the measure-
ment error in the independent variable.

In most of the studies of blood lead-
air lead relationship, the mean of many
different air lead measurements
was taken, thus minimizing the contri-
bution of outliers to error by the
total variance of the dependent vari-
able. Only in the case of the Delco-
Remy study, where single measure-
ments of air lead measurements
air lead and blood lead levels were paired,
would the inaccuracies in air lead mea-
surement 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, mea-
surement error would be a problem.

Another type of measurement error,
distinct from the classical "errors in
variables," results if the present aver-
age 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 evi-
dence in the record that air lead levels
have fallen significantly in the lead in-
dustry 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 probably that a
number of sources of error may sig-
ificantly affect the accuracy of any
incremental benefit prediction con-
ducted using any one study of air lead-
lead blood correlations. The major
sources of error are:

The unjustified causal interpreta-
tion of coefficients of simple regres-
sion results based on observational
data relating to specific populations at
one point in time. This causal inter-
pretation is unjustified because of
specification errors inherent in the
design of these studies. Important var-
iables 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 re-
results individually is appropriate to-use
in predicting the effect of air lead re-
duction throughout the entire indus-
try. (This does not mean that it is not
possible to derive a generally applica-
table model, however.)

Most of these studies include no ex-
plicit theoretical justification for the
use of any particular fit to the data, whether linear or nonlinear.

Errors in the measurement of air lead levels could also introduce such differences and 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 exposure 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-concentrations of 0.3-9 μg/m³. Air lead levels were measured three times each year. 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, it was assumed that air lead concentration was only 9 μg/m³. 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. 86; 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 lead level predictions made using the AZAR regression equation agree with the findings of several other studies, most notably the Hammond study.

In light of more recent studies conducted in occupational settings it is no longer likely that such a claim could be made. In the absence of 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 μg/m³. The extrapolation of the relationship based on observation at blood lead levels between 0.3 and 9 μg/m³ to levels as high as 300 μg/m³ could not be considered a significant use of the data. 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 Xehoc in the 1940's and 1950's. (Ex. 533.)

In the first study, two subjects were exposed to particulate lead 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 μg/m³ and the other to a particulate concentration of 150 μg/m³. Both workers' blood levels appeared to rise 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. 532), who observed 29 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. The workers were analyzed to determine 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-linear relationship which fits the data much better than Williams' regressions. (Ex. 466). Williams himself reported that he detected a large systematic error in all lead measures just after the study was published (Ex. 234F). All of these criticisms 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 fact 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 inclusion of the Williams study in any general model of blood lead-air lead response. For example, even if the controls are removed the regression equation is Y = 0.07x + 1.2x^2 which is not inconsistent with the other studies. Note however, that the Zhao regression model is consistent with OSHA's examination of error sources, i.e., Y intercept biased high.

In 1976, Buncher et al. (Ex. 385) 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 stack 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 errors 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 conducted 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 at 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 samplers. A large, 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. 234, 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/100 cc. 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 numbers of workers exposed to particulate lead levels much greater than 200 μg/m3, and 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 employee. 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. 59) 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 97 μg/m3 had an average blood lead level of 51.8 μg/100 g.

The results of these studies provide data necessary for the development of a comprehensive model of blood lead response to occupational particular 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 provide is a "snapshot" 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 (600-500 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 is, whether produced 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 of 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 µg/m³) air lead exposures.

The remaining studies agree reasonably well, considering the difference in location, methodology, and differences 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 accounts 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 for use in constructing a model for particulate exposure was adapted to account for the differences between industries. The guides used in constructing the model were extensively reviewed by Dr. Dale Hattis in his letter to Richard Gross of 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, 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 correlation studies from the occupational 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 model for particulate exposure.

- 1. The Bernad 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 mammalian 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 responding 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 or out of a compartment from the blood depends upon a number of factors, such as whether or not that particular organ specifically takes up or metabolizes the substance in question. Bernard also observed 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 conditions.

In the course of the rulemaking process, representatives of the lead industry presented 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 the Center for Policy Alternatives (Jan. 13, 1978 (Ex. 468A)). 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 conditions.

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 autopsies and 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 of lead in long-tenured workers, and an overestimation 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 lead 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 experimentally observed behavior of the substance in question. 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 ciliary 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.

One of the major uncertainties concerning the use of the Bernard model in predicting medical 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 µg/m³ of particulate enter a worker's body 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. Of medical basal 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 exposure to be located relatively closer to sources of lead emission into the workplace atmosphere than their fellow workers with smaller air lead exposures. Because the larger lead particulates 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 AMA study (Ex. 437) show that for six locations, whose average total particulate exposure is about 1,000 µg/m³, the average amount of particulate of diameter less than 1.1 micron is only 21 µg, with only two locations (Bronx refinery) having small particulate levels of about 12.5 µg/m³. Thus it appears that, at least in the smelting industry, the vast majority of particulate above 12.5 µg/m³ 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, for extremely 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 300.6 µg/m³, only 5.6 percent, or 20.2 µg/m³, 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 µg/m³² 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 µg/m³, about 18 µg/m³² was smaller than 1 micron, and the other, which had a total particulate level of 121.3 µg/m³², only 12.5 µg/m³² 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 µg/m³² 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-air lead correlation. (Ex. 485). 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-bone lead correlations, but disagrees 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 distributions 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 used by the presence of many long-term workers in the sample.

A final criticism which has been made of the application of the Bernard model to predict occupational blood lead levels is that the model predictions do not correspond to the classic clinical observations of Kehoe on the blood level response to controlled exposures to lead particulates. (Ex. 5(83)). The major discrepancies were:

One of the subjects exposed to particulate lead (F.C.) exhibited blood levels that rose and then began to decline to 150 µg/m³ 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 0.06 micron, produced by the combustion of tetraethyl lead in a small propane burner. Subjects were exposed to lead 8 hours/day, five 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, with all of the previously observed 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 80 percent efficiency. At the other extreme, 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 setup 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 80 percent. All of then simply runs the models, assuming the experimental subjects breathe 9.6 m³ 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 to tell how much of the variance in blood lead levels is due to individual differences of the response of blood lead to particulate exposure. It was difficult to decide what value of blood lead to start the simulation with. At value of between 23 and 35, the model predicts 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 not detect no significant difference between the blood lead levels of this subject during the control period and during the period exposure.

To summarize: The Bernoull 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 (S.W.) 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 above-mentioned 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 μg/100 g would be a more accurate estimate 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 determine 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, $\sigma_i$ in equation 1 in reality becomes the sum of two or more kinds of variations,

$$\sigma_i = \sigma + \epsilon_i$$

In this case, $\epsilon_i$ is the error due to long term variation in individual response to lead exposure, and $\sigma$ is the error term due to other sources, such as short term fluctuations in individual levels (weekly, cyclic, etc.) and measurement error.

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 (G.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 value of 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 not detect no significant difference between the blood lead levels of this subject during the control period and during the period exposure.

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If long term variation in individuals' susceptibility are not the only source of variability in blood lead levels, then, $\sigma_i$ in equation 1 in reality becomes the sum of two or more kinds of variations,
Using raw data from the Delcor-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 $e_1$ and $e_2$ are independent of each other. 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). $^3$ The standard deviation is the square root of the variance.

$$\text{Var}(e) = \text{Var}(e_1) + \text{Var}(e_2) \quad (3)$$

Thus, we can estimate the standard deviation of $e$, the observed variation in blood lead levels, from the variances of $e_1$ and $e_2$.

An attempt to estimate $e_2$, 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 66D, departments at the Delcor-Remy plant were divided, according to the mean observed blood lead levels for all of the male employees, into groups of departments with average blood lead levels that were equal (group 1) or different (group 2) with average blood level between 40-41.9; group 2 = departments with average blood lead levels between 42.0-43.9. It was assumed that the standard deviation for each department was the same for the long and short term. The short term individual fluctuations and the standard deviation for each department were calculated. For each department within the five groups pooled, and subjected to a 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

<table>
<thead>
<tr>
<th>Departmental average blood leads</th>
<th>N (workers)</th>
<th>Average std. dev.</th>
<th>X$^2$ (for normality)</th>
<th>Prob. (H$_0$=false)</th>
</tr>
</thead>
<tbody>
<tr>
<td>40-41.9</td>
<td>307</td>
<td>5.48</td>
<td>0.05</td>
<td>-0.55</td>
</tr>
<tr>
<td>42-43.9</td>
<td>312</td>
<td>6.67</td>
<td>0.67</td>
<td>-0.5</td>
</tr>
<tr>
<td>44-45.9</td>
<td>315</td>
<td>5.21</td>
<td>0.76</td>
<td>-0.5</td>
</tr>
<tr>
<td>46-47.9</td>
<td>349</td>
<td>4.87</td>
<td>14.46</td>
<td>-0.05</td>
</tr>
<tr>
<td>48-51.3</td>
<td>135</td>
<td>4.91</td>
<td>1.76</td>
<td>-0.05</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5.46</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*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 $\mu g/100$ 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 $\mu g/100$ g (variance = 53.6). The $X^2$ value (10 d.f.), for this distribution, tested against a normal distribution with $\sigma = 7.32$, 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 ($\sigma^2 = 0.004812$), 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 5 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.

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:

$$\text{Var}(e_2) = \text{Var}(e_1) + \text{Var}(e_2) = 29.8 + 53.6 = 83.4 \quad (4)$$

The standard deviation of $e_2$ is thus 9.14 $\mu g/100$ 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. 45), 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 $e_2 (e_2=5.45)$, we get an observed standard deviation of 10.4 $\mu g/100$ 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 $\mu g/100$ g. Two laboratories did extremely poorly, with standard deviations of 10.0 and 15.5 $\mu g/100$ g. All the rest had standard deviations less than 3.5. The standard deviation for the 13 best laboratories ranged from...
Air lead exposure is useless in predicting individual blood lead levels. The data on blood lead-air lead correlation is so weak that it does 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 that the 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 use of 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 variability of blood lead levels would be, the answer would depend upon several factors. A lower bound estimate would involve using an 8a, σ = 5.46, since it is probably 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 8b = 8a + 8c (σ = 9.1-10.4) would be a good estimate, assuming reasonably measurable errors. If one wishes 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 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 because the relations 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:

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 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 population 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 the 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 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 health in an adequate approximation of the response of blood lead to air lead dependence downward.

Thus, arguments about which study of blood lead-air lead correlation most accurately predict the magnitude of 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 Bernhard 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. Air lead measurements for the "map shot" observation are 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 200 \( \mu g \)/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 healthy workers with weak responses to high 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 blased 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 paired 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 relationship, it is likely that the result 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 choose one over the other.

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 one 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. 458 A), to the studies indicating why he believes that none of these studies provides convincing evidence that the assumed physiological response to lead exposure in animals is nonlinear over appropriate ranges of lead exposures.

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 \( \mu g \)/100 g in an individual with a normal blood volume of 5 liters. (The concentration would be 1.9 \( \mu g \)/100 g instead.)

This approach misunderstands 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 the R1 pool is defined to be the level of blood lead which correspond more or less to that level circulating in the blood stream 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 \( \mu g \)/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 not 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 \( \mu g \)/lead/day. It was assumed that the total intake of lead from all sources, including airborne lead as well, was 55.2 \( \mu g \)/day. This 35.2 \( \mu g \)/lead actually absorbed into the body to produce the equilibrium pool levels in equivalent to a dietary exposure of about 150 \( \mu g \)/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 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
Hattis has already demonstrated that physical justification for this part of the model fit the observed data. The assumptions which were used only been enough to be absorbed at high efficacious particulate exposures include at emission of "large" lead particulate exposure. that there will be a perfect correlation between lead particulate sources which may be exist in real workplaces where there are multiple tendencies for workers with smaller total lead exposures. Be- cause the larger lead particulates will tend to settle out from the atmosphere faster than smaller particulates, works 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-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 particulate exposure and proportion of "large" lead particulate exposure. Lundquist also argued 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 theo-
levels which provide a certain margin of safety below the known harmful levels. (Ex. 2, p. 45933.)

The issue of whether the PEL for lead should be lower than the proposed 100 µg/m² 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 µg/m² 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 µg/m²); i.e., as low as blood 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) and were discussed in 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 µg/m² was not protective of these reproductive functions. Reading of the cited comments and studies indicate that adequate protection could require an air level as low as 20–50 µg/m².

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 PELs, possibly as low as 50 µg/m², 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 100 µg/m², a limit is needed. Therefore, the PEL has been set at 50 µg/m³.

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 an appropriate margin of safety as well as to provide maximum protection against the effects of lead exposure, the blood lead level of lead workers must be kept below 40 µg/100 g. A maximum blood lead level of 60 µg/100 g corresponds to a mean blood lead level of about 40 µg/100 g. (Ex. 2, p. 45933.)

During the hearings there was testimony which argued for establishing a reasonable margin of safety against clinical manifestations of lead toxicity. OSHA recognizes a more conservative PEL than 50 µg/m² 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 µg/m².

A PEL of 50 µg/m² 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 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 µg/m² represents the intersection between maximum health benefits and feasibility.

The permissible exposure limit is based in 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 µg/100 g. A discussion of whether clinical effects occur at blood lead levels below 80 µg/100 g.
The remaining sections in the PEL which follow health effects are benefits of the PELs 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 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. 45545.)

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, and quantification of individual susceptibilities and exposures in the working population is impossibly imprecise. 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, of course, 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 may 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. 45545.)

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 term and "clinical" 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 working as a basis for understanding the 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 such as first unduly alarm the victim or prevent a physician with clear-cut diagnosis. Nevertheless, this early developmental stage of disease is a pathological state and is probably 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 undue delay in detection and intervention velocities, elevation of enzyme inhibition products from hemal impairment, decrease in hemoglobin levels, CNS symptoms, neurobehavioral effects, and reduced kidney function 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 propose 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 or welfare. 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 various 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 occur 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 hemo­globin in peripheral blood. In contrast, increases 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 (ALA) 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 effects 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 in 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 problem, and is 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, which produce overt symptoms of toxicity would also undergo behavioral changes similar to the sensory, motor, and other alterations characteristic of 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 symptoms of lead poisoning are, initially at least, rather vague; irritability and other mood changes predominate in the early stages and can be difficult to interpret. Long biologic half-life results in slow buildup of toxic levels in the body, the symptoms may seem to diverge between the beginning of exposure to a chronically noxious environment and the development and progression of the symptoms of lead poisoning. (Ex. 95, p. 183.)

In 1973 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. Kuchenreiter. We've heard words like abnormal, damage, disease or subclinical disease, toxic poisoning, and asymptomatic poisoning 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 control in the work environment, for the three systems, neurological, hemotological, and renal.

Dr. Ensmo. I think I'd like to present a conceptual framework first and then go back to each of these in detail. I think I'm talking to several of 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 across the triangle, 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 left's call normal. The next box, somewhat smaller, let's call physiologic change of uncertain significance. The next line left's call physiological change. In other words, something that we think is a change that is very closely associated with disease but may not, and in and of itself, be called disease. I would think that morbid hypertensive change would represent fairly severe disease and finally, the tip of the triangle or the very 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 increase in the physiological change. While there may not really be a clear cut distinction, conceptually there is. Because that's where we begin to increase in the pathophysiology and pathologic change. Conversely, there will be a broader number of people who may not be effected to the point that they develop a disease.

I think in this particular scheme, it would be important to keep a sharp line drawn between the concept of physiological change of uncertain significance and pathologic change. While there may not really be a clear cut distinction, conceptually there is. Because that's where we begin to increase in the pathophysiology and pathologic change. Once the ZPP or the FEP begin to increase exponentially, at a 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 understand what it means yet but that I's not strictly speaking, normal and that's an indicator of a pathologic process or a disease process.

- Mortality in this case, I would define as hemoglobin levels also call pathophysiologic changes going upward to 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 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's probably a good example and that's the tip of the iceberg so to speak, would be mortality.

During his testimony Dr. Teitelbaum echoed many of Dr. Bridbord's conclusions: and Physicians have had little difficulty identifying advanced lead intoxication among the more obvious cases of these societies. The problem always has been how to recognize early lead intoxication among the more subtle cases. It's very real. If any single question is common to all physicians who have observed and treated lead intoxication throughout history, it 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 without 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 adults we begin to see this as blood lead levels rise about 50. One reason why I think that is pathological 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 repair done in some of these indicators but clearly there is a limited capacity to repair damage once such damage has occurred.

I think the net effect on the nerve conduction velocity is we're still measuring a fairly simple function and that to perform complex functions requires some integration of many of the nervous systems. An electrical analogue might be a good example and that has to involve a certain amount of feedback and cumulative damage.

Certainly, once someone has had wrist drop, unequivocally that's morbidity and that's a very distinct disease entity. In the case of the kidney system, it's 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 a kidney, are not very good. At least the routine indicators that we have. As far as I'm concerned, 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 40 of 60 would not be more appropriate. (Tr. 1796-1802.)
has been how to prevent the occurrence of lead intoxication and how could one recognize it early enough to prevent death or permanent 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 effects were available, and when the engineering controls for the prevention of lead intoxication exist, that we should still be in a quandary as whether it is possible, to prevent lead poisoning before any action was taken to protect the workers. Rather, we must focus our attention on the point at which preventive medicine is an absolute necessity to eradicate this industrial disease can be accomplished on a cost-effective basis.

Our present technologic sophistication permits us to divide the patients who have abnormal lead absorption into two categories. One group has overt lead intoxication, lead poisoning. These patients have absorbed so much lead that a clinical diagnosis of lead effect of lead poisoning and to treat these persons who have suffered lead intoxication.

The remaining issue in their cases, is the threat of injury when it occurred. This group of patients is not lead poisoned. Such patients have long since passed the stage where any attempt to wait for overt disease, to hope for their complete recovery. Rather, we must focus our attention on the point at which preventive medicine is an absolute necessity to eradicate this industrial disease can be accomplished on a cost-effective basis.

The proposed standard falls short of the absolute prevention of lead effects, even in the context of our present imperfect knowlidge of the disease and, no lead exposure would be permitted. However, as a realistic concession to human frailty, it permits exposures of 100 micrograms per meter squared to day and that the 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 Hammond explained when testifying for OSHA, "subclinical" or "subclinical" is the usual terminology for an effect that does not appear to have an effect on health, per se. (Diamond 300-01.)

Before considering the significance of the various biological effects which occur at different blood-lead concentrations, two preliminary observations are in order. 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". The functional capacity test 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. This is true despite the fact that the 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 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. 3355, 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 statute 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. §§ 655(b)(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. ** * * 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 be no 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 employer's neglect 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 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 real 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 experience, exposure 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.)

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 intoxication 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, tremors, numbness, disorientation, hyperactivity, and colic.

Damage to the central nervous system in general and the brain (encephalopathy) in particular is the most severe clinical form of lead intoxication. The most severe and 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 seizure followed by coma, cardiorespiratory arrest and death. There is a tendency toward the occurrence of weakness of extensor muscle groups; i.e., motor involvement. Tremor and weakness are either progress to palsy, often observed as a characteristic "wrist drop" or "foot drop" and is a manifestation 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 lead exposure. The evidence is that prolonged exposure can increase the risk of nephritis, mental deficiency, premature aging, and high blood pressure. (Ex. 94, 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, and 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 hereofore thought to be inconsequential. The main research topics which have been addressed are early biochemical changes in the synthesis of the respiratory pigment, hem and early effects on the nervous system including behavioral and peripheral nerve effects. Studies on the prevention 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 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 changes, overt symptoms (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 occurrence 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 work shift 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 pathological changes, reversible 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 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 building block of the red blood cells necessary for oxygen transport throughout the body. The 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 fatigue as a result of a 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. Piometta has suggested that heme impairment in the cells would occur when the cells are in a condition in each cell similar to that to 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 neurologic damage could conceivably occur prior to anemia as a result of heme synthesis impairment in the brain. For example, Piometta 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 the 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 intermediate 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 deltaaminolevulinic acid (ALA), occurs at this PbB level: 11 percent of adult males are excreting more than 10 mg/l.

The build up 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 argued 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 to 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 essential to the process of hemoglobin synthesis, they are also vital to the function of all cells since heme is ubiquitous in the human.

OSH A 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 poisoning as manifested in disorders in the nervous, renal, and hematopoietic 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 would manifest extensive health 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 lead poisoning, 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, Plomelli suggested that hemopoietic effects such as anemia are not the most significant clinical effect of heme synthesis disruption but rather as 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 50 μg/100 g. (Tr. 5(23)). 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 worker. (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. Seechl (Ex. 5(23)) 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 Plomelli's suggestions that changes in heme synthesis in the blood forming (hematopoietic) system reflect changes that occur in other tissues. The work of Fishbein 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). Fishbein also noted anemia in 37 percent of these same 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 lead poisoning, 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.
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 adherence to 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 toxicological 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 reference to the hematopoietic system, OSHA believes that the effects of lead are a complex progression through 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.

Fomelli 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 is not producing subjective evidence of impairment of health, unless they reach the extreme depression in severe lead intoxication associated anemia 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 adapted to suboptimal health, If changes have not occurred slowly enough and also because we 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. These 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. 97, 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 of great help in understanding 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 effect of lead on the nervous system range from acute intoxication coma, cardiorespiratory arrest and fatal brain damage to mild symptoms, subtle behavioral and electrophysiological changes associated with lower level exposure. Although the severe effects of lead have been known for some time, only in the last several years has evidence accumulated which demonstrates neurological 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 also apparent 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 previously recognized. In particular, Lilis et al. (Ex. 24, 50) has demonstrated central nervous system symptoms (tiredness, fatigue, nervousness, sleeplessness or somnolence, or anxiety) in 56 percent of workers with blood lead levels below 80 µg/100 ml. The percentage blood lead levels were 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 symptoms had disappeared within 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. Lilis testified that above 60 µg/100 g "one may expect florid lead poisoning, full blown lead poisoning, or something worse. 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.) Reg. 6 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. concluded that at blood levels of 70 µg/100 g demonstrated impaired psychological performance among workers with low exposure to lead. Henninen's work is particularly significant insofar as no single blood lead concentration had ever exceeded 70 µg/100 g.

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 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.
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. OHSA 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, Behe, Mostafa et al., Gerald et al., Guadaglici et al., Arakl, W. R. Lee, Repko, Lillis, Fishbein 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 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 98 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 to which 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 g.

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. 84) 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 co-workers. This work has been described by an industry spokesman, Dr. Malcolm, as being "immaculate." (Tr. 2076) Based upon extensive evidence in the record from Seppalainen and others, OHSA 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 affected. However, apart from a few complaints of numbness of the arms, 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, however, have not been discouraging, and the presence of nervous system damage is an early stage in the development of kidney disease among lead workers.

Seppalainen testified that "to progress the source and therapy initiated, the worker may still experience impairment. In a recent paper describing his results Dr. R. Baloh, a neurologist at U.C.L.A. equates the nervous system damage as evidence to suggest the only reliable way to treat nervous system damage from inorganic lead is chelation therapy and/or removal from further exposure. This is not particularly surprising, however, since experience with other heavy metals intoxication has been similar. Nervous system damage from arsenic and mercury respond 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 (T), 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.) OHSA 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 among lead workers. 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 OHSA 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 hemolytic 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 co-workers, the health hazard evaluation by NIOSH at Eagle Picher Industries, Inc., and the research in secondary smelters by Lillis, Fishbein et al. demonstrated that lead exposure is a key etiologic agent in the 100 milligram level. (Tr. 155.) OHSA agrees that "occupationally exposed workers. Clearly, too little attention has been given
to lead-induced renal disease in recent years, and while OSHA recognizes that this is a concern, 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 levels are valuable in the diagnosis of renal disease development. Dr. Bribord questioned Dr. Wedeen on the issue of chronicity of exposure and blood lead levels.

Dr. Bribord. 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 occur after few months of exposure and the blood lead level may remain the same for the next 20 years, despite the individual is continuously accumulating lead in the body.

Dr. Bribord. Would you think that the chronicity of lead exposure, apart from precisely what blood lead was an adequate indicator below 80 or above 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. Bribord. 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 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. 1755-1766.)

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 serious. This is important because the worker has lost the functional reserve, the safety, provided by two normal kidneys. If one kidney becomes damaged, the other is not likely to rely upon. The lead worker with 50 percent loss of kidney function has no such security. Loss of one 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 permanent damage has occurred. The lead standard must be directed towards limiting exposure so that occupational lead nephropathy does not occur (Tr. 1747-1758.)

And OSHA agrees with Dr. Richard Wedeen, that "40 μg/100 ml is the upper acceptable limit" (Tr. 1771) and with Dr. Bribord 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 at an inadequate measure of occupational exposure (though most agree the best available single measurement) they have no way of 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 lead workers are less likely to produce healthy sperm and that there is conclusive evidence for 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 progeny of animals fed with diets containing lead (Ex. 29 (30), p. 430.)

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 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 stillbirth. 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 uncontested and there were no refutations. 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 15-14 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.
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 affect 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, cardio-respiratory or CNS arrest. There have been reported cases of acute encephalopathy similar to those in infants who are 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 lead 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. Beattle 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 a significant relationship between blood lead concentration and mental retardation. Mean blood lead for the retarded children was 25.5 µg/100 ml. While lead levels are present in the maternal blood during pregnancy, they 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.

With 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 50 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 50 µg/100 g. In general, 30 µg/100 g appears to be reasonably protective 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 compara­
tively low levels. Given this in utero 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 brain, 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 raises particularly difficult issues when establishing this final standard. OSHA recognizes that a PB 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 lead blood lead levels 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 ml 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, 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 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 or 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 indicates that for many record, including their rapid growth, children may be susceptible to lead intoxication at lower blood lead levels than adults. The U.S. Public Health Service considers this and other factors when it recommends, 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 were devoted to this issue. Participants in the hearings argued that, given the data demonstrating adverse effects on male reproductive abilities and potential genetic defects 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. Steilman 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 men. (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. Steilman and Hunt that women of childbearing age exposed to lead are not more susceptible to adverse effects on their reproductive capacity than are male workers. There can be no doubt that the reproductive ability of both males and females is adversely affected 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 \( \mu g/\text{m}^3 \) alone will not maintain all workers below 30 \( \mu g/\text{m}^3 \). The mean blood lead level of workers uniformly exposed to 50 \( \mu g/\text{m}^3 \) will be approximately 35 \( \mu g/100 \text{g} \), and the population blood lead distribution is predicted to be: less than or equal to 30 \( \mu g/100 \text{g} \), 30 percent; 30-40 \( \mu g/100 \text{g} \), 40 percent; greater than or equal to 40, 30 percent. When full compliance is achieved with the 50 \( \mu g/\text{m}^3 \) PEL through engineering and work practice controls, 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 (EPA) 1.5 \( \mu g/\text{m}^3 \) Action Level 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 workplace exposure by employees with blood lead 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 the PEL. When compliance is achieved in a particular plant, however, there will no doubt be many areas throughout the industrial operation where the lead levels will be substantially below the PEL—therefore further reducing the blood lead levels of the aggregate workforce. However, even taking these mitigating factors into account, there will often be a substantial percentage of workers whose blood lead levels exceed 30 \( \mu g/100 \text{g} \). 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 reduce reproductive risks. Use of these procedures by concerned employers and 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 \( \mu g/\text{m}^3 \) 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 with 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 employee 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 could be exposed to levels of lead which are not currently recognized as excessive. An employee might also be provided with the opportunity to use a 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 can also recommend implementation of 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 benefits due to the need to be temporarily removed from lead exposure, or otherwise limited in any manner by the standard. The medical surveillance program also offers employees the opportunity to obtain, from a physician, 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, consequently, 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. Erickson testified that women of child-bearing age had been excluded from employment because "the response of industry has been to 'protect' women workers from lead's reproductive hazards by refusing them the opportunity, upon request, of forcing them to prove that they can no longer bear children" (Ex. 60(a)). There was also testimony which dem-
constrains that women have and do work in production areas of battery manufacturing (Tr. 1245, 4057, 4058, 4059, 5520, 5529, 5530, 5531).

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 productive risk on a case-by-case basis rather than 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 committee to evaluate employment opportunity overrides the health considerations, then there should be a program which would assure that the female knows the risks, that the employer is protected from liability, and that information is obtained which help us to better 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 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 it will not change the basic standards. 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 3068-70.)

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 developed a comprehensive standard which will minimize 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.

a. 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 is based on the question of 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 acute 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 clear-cut 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 will 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 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 important, relevant evidence 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(T2), p. 19.)

Experience and the accumulation of voluminous data have spoken for themselves, in proclaiming that an eight-hour workday can be entirely eliminated given the current 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 PBC 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. 85M.)

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 μg/m3 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 24 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 does not proceed to overt disease. Evidence from many sources makes it obvious that chronic low level lead exposure is widespread 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" (CERES Ref. 52). There are numerous studies showing effects on children and adults below 80 

\[ \text{pg/dL} \] 

100 g. Statements which are clearly at odds with current data raise serious questions about Dr. Kehoe's overall view. Medical and public health specialists are concerned that lower levels of lead are hazardous while industry spokesmen maintain that evidence for low dose effects is faulty and far from persuasive.

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 sequence of lead doses sufficient to produce clinical symptoms 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 exposure. Pediatricians and public health specialists are concerned that lesser levels of lead are hazardous while industry spokesmen maintain that evidence for low dose effects is faulty and far from persuasive.

I am one of those who believe that a substantive 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 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 electroencephalograph, He found that 10 of the 20 were failing in school, had significant problems in perceptual motor function or were severely behaviorally 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. Sadly, this was the burden of my research in 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 lead exposure. Dr. Byers at the Amsterdam meeting in 1972 recommended an individual limit of 35 micrograms per deciliter and a group average of 29 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 impairments 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. 1077-78)

There are important differences during the time that the blood brain barrier is being laid down, in that certain enzymes are sensitive at six. I think the point that I was trying to generate in that argument was that in my pediatric experience, when I started training in pediatrics, we used to say that children over six or seven were at high risk for the lead poisoning, and now we have been talking about children of 30, 40, 45, or 40, and I think the same argument holds. The medical examiner's experience and a 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 aminolevulinate 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 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 

\[ \text{g/dL} \] 

and those whose blood lead concentrations were more than 60 

\[ \text{g/dL} \] 

Even though the population studies have been substantially exposed above the newly proposed TLV of 0.10 mg/m3, there have been only a few of the described cases related to the lead exposure, and few, in the opinion of the attending physician, have required chelation therapy for the repair of the body burden of lead.

Based on these findings, it is our opinion that the current blood lead standard of 60 

\[ \text{g/dL} \] 

can be kept, unless more new data will support the OSHA proposal. Also, the OSHA policy with respect to 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 

\[ \text{g/dL} \] 

We believe that implementation of this proposal of OSHA could not add any further dimension to the less rigorous protection program employed by ASARCO. (Journal of Occ. Med, 20, pp. 1960.)

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 creat, but there were only 229 determination of the ratio of BUN to creat.

The disparity between these numbers is not explained. 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 were a number of omissions which I believe were 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 mentioned in the papers 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. 1950-1959).

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 this then was publishing studies and doing research in the 1930s, 1940s, 1950s, 1960s, 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. SAMPSON. 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 the same men remained 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. SAMPSON. 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 that produced data. I am saying that this is his statement.

Mr. SAMPSON. So you have never read a paper of his that produced data. Are you 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 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. BRADBOR. 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 his 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 was based upon 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 lead level was only 80 µg/100 g, or 0.08 mg percent, and the diagnosis of lead poisoning was therefore missed for 2 years. Indeed, it at first appeared that the gallbladder surgery he had undergone was indeed the cause of his abdominal pain because lead colic, like normal blood 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 level of 80 µg lead is easy and more economical than the long and expensive process of validating studies showing effects below 80 µg/100 g. Industry representatives argued studies showing effects below 80 µg/100 g do not address the issue of cause and effect and are not scientifically valid since they are not longitudinal in nature. In a cross-sectional study, for example, measurements of cause and effect are sampled at a given point in time—when you talk about scanty published data and then you go on to discuss Dr. Williams' opinions even if they were not supported by data. 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.

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Mr. SAMPSON. So you have never read a paper of his that produced data. Are you 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 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. BRADBOR. 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 his 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 was based upon 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 lead level was only 80 µg/100 g, or 0.08 mg percent, and the diagnosis of lead poisoning was therefore missed for 2 years. Indeed, it at first appeared that the gallbladder surgery he had undergone was indeed the cause of his abdominal pain because lead colic, like normal blood 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 level of 80 µg lead is easy and more economical than the long and expensive process of validating studies showing effects below 80 µg/100 g. Industry representatives argued studies showing effects below 80 µg/100 g do not address the issue of cause and effect and are not scientifically valid since they are not longitudinal in nature. In a cross-sectional study, for example, measurements of cause and effect are sampled at a given point in time—when you talk about scanty published data and then you go on to discuss Dr. Williams' opinions even if they were not supported by data. 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.
levels, and efficiency of other biological indicators have a crucial role. OSHA has sustained 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 small 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 rigorous of cross-examination. OSHA recognizes that blood lead level measurements are not necessarily accurate representations of past exposure, but wishes in mind the agency does have the evidence for "clinical effects" below 80 \( \mu g/100 \text{g} \). There is in fact evidence of signs and symptoms (morbidity) at levels as low as 40 \( \mu g/100 \text{g} \). That evidence is discussed in detail in the health effects section.

2. Benefits. The dramatic reduction in blood lead levels over 40 \( \mu g/100 \text{g} \), as shown below, is a measure of the increment derived from the PEL of 50 \( \mu g/100 \text{g} \). OSHA has concluded that based on the health effects data in the record blood lead levels should be maintained below 80 \( \mu g/100 \text{g} \) as the extension. 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 signs and symptoms effects of lead exposure (e.g., neurological damage, kidney damage, etc.). However, the available data does not allow meaningful quantitative estimation of the differences between the different forms of health damage likely to be achieved by lowering worker air exposures and blood lead levels. A beneficial comparison is possible, but various methods 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. A 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 ranges over 40 \( \mu g/100 \text{g} \) 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 change in the entire distribution of the blood lead levels in the population.
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 μg/m³. The "Results" section will utilize the Bernard model and Assumption C, 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>
<thead>
<tr>
<th>Table 1.—Best Point Estimates of Ultimate Equilibrium Benefits of Reducing Air Lead Exposures</th>
<th>[Blood level standard Deviation=6.5 μg/100 g]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long-term average air lead exposure</td>
<td>Total number of workers</td>
</tr>
<tr>
<td>Current Compliance Level</td>
<td>&gt; 100 μg/m³</td>
</tr>
<tr>
<td></td>
<td>50-100 μg/m³</td>
</tr>
<tr>
<td>Compliance With 200 μg/m³</td>
<td>&lt; 50 μg/m³</td>
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<tr>
<td>Compliance With 100 μg/m³</td>
<td>&gt; 50 μg/m³</td>
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<tr>
<td>Compliance With 50 μg/m³</td>
<td>&lt; 50 μg/m³</td>
</tr>
<tr>
<td>Incremental Benefits</td>
<td></td>
</tr>
</tbody>
</table>

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.
- Miscellaneous environmental conditions affecting physiological processes (heat, humidity, other chemical and physical stressors).
- Variation in the work week (over-time, 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 variability 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 μg/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 appropriate 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-
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,” “c,” and “d” compliance levels compared to the baseline defined by the “a” compliance level. (See figure 2.)
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)

Number of Workers (1,000's)

Blood Level

a. Current Compliance level with 200·ug/m³ Air Standard

Over 60 ug/100g
50-60 ug/100g
40-50 ug/100g
Over 40 ug/100g

b. Compliance with 200 ug/m³ Air Standard

Over 60 ug/100g
50-60 ug/100g
40-50 ug/100g
Over 40 ug/100g

c. Compliance with 100 ug/m³ Air Standard

Over 60 ug/100g
50-60 ug/100g
40-50 ug/100g
Over 40 ug/100g

d. Compliance with 50 ug/m³ Air Standard

Over 60 ug/100g
50-60 ug/100g
40-50 ug/100g
Over 40 ug/100g

*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-lead levels. We can anticipate that about 41,622 workers would have blood-lead levels over 60 \( \mu g/100 \text{g} \), and about 79,569 would have blood-lead levels over 40 \( \mu g/100 \text{g} \), in the absence of other remedial measures. Achievement of the "a" compliance level would reduce the number of workers over 60 \( \mu g/100 \text{g} \), but would leave the number of workers in the 50-60 \( \mu g/100 \text{g} \) and 40-50 \( \mu g/100 \text{g} \) range substantially unchanged. Achievement of the "a" compliance level would be expected to make reduction to about 2,500 in the number of workers over 60 \( \mu g/100 \text{g} \), and would be expected to produce some reduction in the numbers of 50-60 \( \mu g/100 \text{g} \) blood-lead level range to 14,000. The "d" compliance level would reduce the total number of workers over 40 \( \mu g/100 \text{g} \) to slightly under 28,599, as compared over 79,569 for the "a" scenario. The integral benefit of "a" versus "d" is in terms of workers over 40 \( \mu g/100 \text{g} \) would be 50,970 and for workers over 60 \( \mu g/100 \text{g} \) the benefit would be 32,270. These are clearly substantial reductions in the numbers 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 benefits of "a" and "d" 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 these indices. Employers covered by the standard should be required to conduct environmental monitoring and shall 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 in such an enforcement mechanism (even if based on the proposed permissible limit of 100 \( \mu g/\text{m}^3 \)) will not accomplish the objectives of protecting workers' health.

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 \( \mu g/\text{m}^3 \) 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 \( \mu g/100 \text{g} \). The OSHA action level is an air-lead level of 30 \( \mu g/\text{m}^3 \). The LIA proposal sets 80 \( \mu g/100 \text{g} \) as the appropriate exposure limit for compliance purposes with a blood-lead action level of 60 \( \mu g/100 \text{g} \).

c. OSHA will continue to place primary reliance on engineering and work practice controls for compliance purposes while environmental monitoring is utilized to verify and enforce 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 sum, OSHA has decided to place primary 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 allows a more effective monitoring 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 it would be a deviation of 60, 70, or 50 \( \mu g/100 \text{g} \) would be excessive and would not protect workers health adequately. It would be infeasable to require controls to maintain blood-lead levels at the level of 40 \( \mu g/100 \text{g} \) and below. Rather when all controls are implemented 30 percent of all workers PbB will range from 40 to 60 \( \mu g/100 \text{g} \). 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. Therefore, OSHA believes a PbB above 60 \( \mu g/100 \text{g} \) is excessive but a PbB level in an individual worker between 40 to 50 \( \mu g/100 \text{g} \) may be the result of excessive exposure.

3. A biological standard is not only unnecessary but 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 lead determinations would differentiate between the two situations.

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 unsubstantiated. OSHA believes there is no doubt that an air to blood relationship exists and is best described by the CPA application of the Bernard model.

6. Although both biological and air monitoring are subject to errors OSHA believes that the uncertainties
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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 associated with lead exposure, it appears necessary to establish a comprehensive air and biological monitoring program. (Tr. 5739.) Similar testimony was presented by most other witnesses, including those appearing on behalf of Government and Industry (OSHA 1330-31), industry (AMAX 1703; Cominco 2226-28; Cole 3167-68; Caplan 3868; Globe Union 4312; General Battery 4551) and labor (Teamsters 2203-04; McBride 2961-62, 2973; Woodcock 5940).

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 can be minimized 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 emission 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 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. Implementation of engineering controls 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 context of 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 means of controlling the exposure to lead, 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, the blood lead level 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 could be implemented which will lower the PbB levels of more than 70 percent of all lead exposed workers to below $40/\mu\text{g}/100\text{ g}$. In fact, many industries will be able to achieve an even higher percentage of workers below $40/\mu\text{g}/100\text{ g}$.

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 workers will have a PbB greater than $60/\mu\text{g}/100\text{ g}$, 5 percent will be between 50 and $60/\mu\text{g}/100\text{ g}$ and 23.3 percent will be between 40 and $50/\mu\text{g}/100\text{ g}$ for a total of 29.3 percent. The mean PbB for the entire population will be approximately $35/\mu\text{g}/100\text{ g}$. Assuming compliance with $100/\mu\text{g}/\text{m}^2$ 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 believes that individuals whose PbB is above $60/\mu\text{g}/100\text{ g}$ or the worker will be removed from that job to one in which the air lead level is below $50/\mu\text{g}/\text{m}^3$ (see MRP section). When full compliance with $50/\mu\text{g}/\text{m}^3$ is achieved, the number of employees whose PbB is above $60/\mu\text{g}/100\text{ g}$ will be virtually eliminated. However, there will be a sizable number of workers above 40 at $50/\mu\text{g}/\text{m}^3$ (29.3 percent) and there would be at $100/\mu\text{g}/\text{m}^3$ (50.7 percent). It is estimated that ultimately a PbB level of 40 will result in there being 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 $40/\mu\text{g}/100\text{ g}$, 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 $60/\mu\text{g}/100\text{ g}$. Therefore, there is a good 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 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 initial exposure equilibrium is defined as the short run time frame. The period after the population reacts to ultimate equilibrium exposure is defined as the long run time frame. The period after the population reaches its ultimate exposure equilibrium. There is no precise PbB which reflects compliance purposes is meaningless, since the preferred value of $40/\mu\text{g}/100\text{ g}$ cannot be achieved because of feasibility constraints and because OSHA expects that even with the optimum controls, 5 percent of all workers will not have PbB between 40 and $60/\mu\text{g}/100\text{ g}$. There is no precise PbB which reflects implementation of controls. The percentage of workers whose PbB levels are between 40 and $60/\mu\text{g}/100\text{ g}$ 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. 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 lead levels will rise until equilibrium is reached. However, prior to equilibrium their blood lead levels would have been lower and higher exposure conditions would not have been identified if the OSHA compliance official had relied on biological monitoring. For example, if a worker with no prior exposure had a blood lead level of 2111$g/100\text{ g}$ (134$\mu\text{g}/\text{m}^3$) his blood lead level (using assumption C—Bernard model) would rise as follows: 6 months—$49/\mu\text{g}/100\text{ g}$; 1 year—$55/\mu\text{g}/100\text{ g}$; 2 years—$60/\mu\text{g}/100\text{ g}$; and 4 years—$69/\mu\text{g}/100\text{ g}$. If OSHA had established $60/\mu\text{g}/100\text{ g}$ as a compliance level (assuming no MRP removal) this particular worker would not exceed it for 2 years although his blood lead level would still exceed the PEL. Therefore, 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. 282.)

Citing the work of Dr. Chisolm, 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. Rosch 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 a better indication than the analysis of body excretions or blood to be analyzed. Even with those few substances where biological samples can be useful indicators of absorption of a contaminant, 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. 4169), First (Tr. 2519) and Stewart.
I would not put my primary emphasis on biological monitoring for this reason that is the after the fact (Tr. 2008).

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. 2010).

OSHA should not adopt a compliance strategy which might cause a worker's loss of employment. In the example of the former worker, the standard is cut 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 use a given-corp control. 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. It is adequately possible 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 standard. OSHA should not adopt a compliance mechanism which might be circumvented, if only by a 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 insufficiency 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, even considering the testimony to the participation of unions since a high percentage of employee participation would be necessary for the success of such an invasive procedure as biological monitoring. Unless seriously 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 as the only measures that have their usefulness and both should be employed. Just as important, both have significant limitations and, therefore, disadvantages. Rather than tesomely arguing the relative usefulness of each of these measurements at these hearings we 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 the 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 environmental monitoring. Biological monitoring, including blood lead determinations and the sensitive and practical indices of toxic exposure 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 and residually available time to equilibrate following such exposure. Even for this purpose, the validity and sensitivity of blood lead tests have been recently challenged. Blood lead levels also reflect incremental nonexposure—nonoccupational exposures. Additional 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. Contrarily, the recently developed ZPP test is highly sensitive, provides immediate results, and can be performed by relatively unskilled personnel with furthermore being highly economic and practical.

Finally, blood lead levels underestimate exposure and lead body burdens at higher exposure levels. Blood lead levels were measured to have their usefulness and both should be employed. Just as important, both have significant limitations and, therefore, disadvantages.

above approximately 40 micrograms per 100 gms as stated in OSHA's regulations 4715-16. That figure of 40 micrograms per 100 gms corresponds to an air lead level of about 60 micrograms per cubic meter of air. Thus, a four-fold increase in blood lead was associated with a tenfold or greater increase in chelatable lead. In other words, blood lead is a very sensitive 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 ARSACO 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.

The 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. 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, different 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 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 the petitioner's expert testimony, to ascertain whether they should be charged with another workplace in the plant. In the fact, the plant manager had no direct involvement with the monitoring system. The employer was "not 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-
Given the fact that the act has been in effect for 7 years, this record of compliance raises serious doubts about the contention that 200 lead industry appears to be in compliance not on what is asserted to be possible in the future but rather 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 focus for enforcement. The standard should not be enforced by blood lead criterion. The practice of using the worker as a monitoring device for clean or at least safe air, but is not a substitute for a healthful working environment.

Lead should be treated as other occupational health hazards with adverse health effects, it directly relates it to the chemicals concentration in the air. In the case of lead, the blood lead measurement is a better indicator of exposure. 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 theOHSANature 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 years.

Dr. Brau: 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 workers to submit themselves to blood tests. (Tr. 1545-46).

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 open-ended as it is perceived to be designed to protect and who are crucial to its implementation. Under the Act the burden for a safe and healthful workplace is on the employer, not the employee. OSHA believes a strategy designed to determine adequate 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; 6946-651 348 exhibit 246 at 4.) OSHA believes this statement is entirely with 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 Invasive nature of the technique.

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 but rather on what it has observed. History of the industry's effort to achieve compliance 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 μg/ 

m³; ** (Ex. 349)

RULES AND REGULATIONS

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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 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 be repeated 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 relationship. 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 level at a given point in time, to predict effects. It is concluded that the weight of the lead-blood correlations with rela­

useful to supplement the empirical air to blood relationships derived from physiological models of lead transfer to the body. The quantity of lead in the blood. Most clinical measures of lead toxicity have been related to blood lead determinations. One of this issue in the difficulties and 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 have different limitations. 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. For example 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 performed. 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 measurements of the employee's exposure (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 employee.

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 determinations. One of this issue in the difficulties and 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 have different limitations. 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. For example 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 performed. 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 measurements of the employee's exposure (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 employee.
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 mountable 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, 2912, 2994, 2972-73, 4127, 3749-9, 6980-2.) NIOSH wholly supported establishing a PEL based upon air lead levels.

Mr. Belczewski. Mr. Baier, in the absence of a standard on worker exposure levels and sole reliance on blood lead determinations, 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 of one that would be based on its blood lead level only. There is the necessity of being based on a lead blood. I mean, if that was the sole source of information, as we pointed out before, it simply tells you that you have been overexposed. It is the source of being overexposed, exposure. I don't know what, the incentive would be.

Mr. Belczewski. Does anyone else care to respond to that?

Dr. Bumsom. Yeah, my personal feeling would be that that would tend to be a disincentive toward the development of engineering controls and it would trend 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. Belczewski. Do you feel that a promulgated standard, based solely on blood lead levels, would encourage the indiscriminate use of respirators?

Dr. Bumsom. I think that it has that potential. Yes sir. (Tr. 1457-8.)

OSHA considers the point of Mr. Belczewski and Dr. Bumford extremely important; that is, how a biological monitoring program based on biological monitoring which further renders it even more important. The agency will not attempt to address this debate in detail since OSHA believes that debate is premature. (Tr. 352, 3104, 3260, 6476.) The agency will not attempt to address this debate in detail since OSHA believes that debate is premature.

Statements of Mr. Belczewski about the usefulness of biological monitoring are probably correct in that biological monitoring is not very useful in monitoring control practices although there is no reason to believe that it is useless. This makes it possible to recognize this important interrelationship between good work practices and good control engineering accounts for the accuracy of biological monitoring because 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 shortcoming 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 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 development of high concentrations of lead in blood or urine.

Reliance on routine air sampling represents an important input to a conscientious engineering control program because it 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. In the event of an overexposure (as would be the case where one is using biological monitoring that requires weeks of exposure before reaction to a single exposure 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 standard is calculated 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 far less subject to human errors of interpretation. Of equal importance is the informed, well trained, and responsible work force because 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 far less subject to human errors of interpretation. Of equal importance is the informed, well trained, and responsible work force because control standard for conducting such surveys.

For the reasons cited above, OSHA will place primary reliance on its PEL of 50 μg/m$^3$ as determined by environmental monitoring.

b. 100 μg/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 provide significant protection against the effects, clinical or subclinical, and the mild symptoms which may occur at blood lead levels below 60 μg/100 g, it is necessary to set an airborne level which will limit lead blood level to 60 μg/100 g. A maximum blood lead level of 60 μg/100 g corresponds to a mean blood lead level of about 40 μg/100 g, will result in a range in workers of approximately 20 μg/100 g at the lower limits to 50 μg/100 g at the upper limits. Having determined the maximum blood lead level which the protection of employees and public 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 μg/m$^3$ as determined by environmental monitoring based on a 40-hour work period. This 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 exposure. 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 use the lower permissible exposure limit for 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. 45939-39.)

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 goal with 60 \( \mu g/100 g \) as a maximum. OSHA believes 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 34.8 \( \mu g/100 g \) 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 \( \mu g/m^3 \).

<table>
<thead>
<tr>
<th>Blood lead level</th>
<th>Percent, workers</th>
</tr>
</thead>
<tbody>
<tr>
<td>50 ( \mu g/m^3 )</td>
<td>0.5</td>
</tr>
<tr>
<td>50 to 60 ( \mu g/m^3 )</td>
<td>6.5</td>
</tr>
<tr>
<td>60 to 70 ( \mu g/m^3 )</td>
<td>23.3</td>
</tr>
<tr>
<td>&gt;70 ( \mu g/m^3 )</td>
<td>29.3</td>
</tr>
</tbody>
</table>

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 \( \mu g/m^3 \). This contrasts with 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 "on the basis of the best available evid..."
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, particularly 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 \( \mu g/100 \text{ g} \) would be reduced from 32,777 to 498 and the number of workers whose PbB levels would be reduced below 40 \( \mu g/100 \text{ g} \) is 50,970. To summarize the benefits:

### INCREMENTAL BENEFITS

<table>
<thead>
<tr>
<th>Blood Relationship and Benefits</th>
<th>Workers Removed</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 60 ( \mu g/100 \text{ g} )</td>
<td>13,957</td>
</tr>
<tr>
<td>50-50 ( \mu g/100 \text{ g} )</td>
<td>22,575</td>
</tr>
<tr>
<td>40-50 ( \mu g/100 \text{ g} )</td>
<td>8,961</td>
</tr>
<tr>
<td>≤ 40 ( \mu g/100 \text{ g} )</td>
<td>45,511</td>
</tr>
</tbody>
</table>

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):

<table>
<thead>
<tr>
<th>Blood Lead</th>
<th>Number of Workers Removed</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 60 ( \mu g/100 \text{ g} )</td>
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<td>45,511</td>
</tr>
</tbody>
</table>

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 \( \mu g/100 \text{ g} \) or less but allowing for a range up to 50 \( \mu g/100 \text{ g} \) 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 \( \mu g/100 \text{ g} \).

Referring again to the table, it is seen that the midpoint of this range, 40 \( \mu g/100 \text{ g} \), would be predicted by air lead values 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 NLRIH 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 \( \mu g/100 \text{ g} \) (Ex. 86D). Second, considering the range of values expected around mean PbB levels in this range, the upper limit would be greater than 50 \( \mu g/100 \text{ g} \) (Ex. 60). Third, as suggested by Epstein, some allowance should be made for a margin of safety (Ex. 66). 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 \( \mu g/100 \text{ g} \) (Ex. 363 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 60 \( \mu g/m^3 \). The results are as follows:

<table>
<thead>
<tr>
<th>Blood Lead Standard Distribution</th>
<th>40 ( \mu g/m^3 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥ 60 ( \mu g/100 \text{ g} )</td>
<td>13,957</td>
</tr>
<tr>
<td>50-50 ( \mu g/100 \text{ g} )</td>
<td>22,575</td>
</tr>
<tr>
<td>40-50 ( \mu g/100 \text{ g} )</td>
<td>8,961</td>
</tr>
<tr>
<td>≤ 40 ( \mu g/100 \text{ g} )</td>
<td>45,511</td>
</tr>
</tbody>
</table>

OSHA has determined 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 \( \mu g/100 \text{ g} \) where possible and in fact preferably below 40 \( \mu g/100 \text{ g} \) 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
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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:

   a. Introduction.
   b. Medical Removal Protection as a Means of Effectuating the Medical Surveillance Sections of the Lead Standard.
      -Rules played by medical surveillance in the standard.
      -Summary of the need for MRP.
   c. Medical Removal Protection Benefits as a Means of Allocating the Costs of Temporary Medical Removals.
      -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.
   d. 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/100 mL average blood level removal trigger 1 year after the effective date of the standard.
      -60 μg blood lead level removal trigger 2 years after the effective date of the standard.
      -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.
      -Immediate removal due to physician determinations.
      -Quantification of potential MRP costs.
      -Economic impact on less heavily impacted segments of the lead industry.
      -Economic impact on small manufacturers.

3. Summary and Explanation of the Medical Removal Protection Sections of the Standard.
   a. Temporary Medical Removal and Return Criteria.
      -Elevated blood lead levels.
      -Final medical determinations.
   b. Removal From Work At or Above the Action Level.
   c. Return of an Employee to His or Her Former Job Status.
   d. The Implementation of Temporary Medical Removals.
   e. Employer Flexibility as to Removal and Return Pending a Final Medical Determination.
   f. Definition of Medical Removal Protection Benefits.
   g. Duration of Medical Removal Protection Benefits.
   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.


The final standard requires that an employer temporarily remove from lead exposure in excess of 30 μg PbA/m^3 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) or the PEL of 50 μg PbA/m^3 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. 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 to temporary medical removal, OSHA views this protective mechanism as an indispensable 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 individuals in circumstances where other protective means have 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, employ­
ers should be able to protect most
workers by persistent dedication to
maintaining all elements of an effect­
ive respiratory protection program.
OSHA also anticipates that the major­
ity of affected employers will con­
tinuously comply with all provisions of the
inorganic lead standard such that tem­
porary removal of most workers is un­
necessary. 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 med­
cal removal, and the final lead stand­
ard includes a mandatory temporary removal provision for this reason.
The record evidence developed in
the lead proceeding demonstrates that
temporary removal of workers at sub­
stantial risk of sustaining material im­
pairment is a protective mechanism
recognized by and acceptable to both
management and labor. Many lead
firms have existing medical surveil­
ance programs incorporating the temporary
removal of any worker whose medical
condition meets specific criteria. (Ex.
157, p. 10; Ex. 158, p. 68; Ex. 386, p. 14;
Ex. 401B, p. 16; Ex. 402B(D-1), p. 4;
76; 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(29) (section 8.) The 1975 pro­
posed inorganic lead standard con­
tained provisions which essentially
prohibited an employer from keeping
any employee at existing exposure to
lead if such exposure posed an in­
creased risk of material impairment to
health. (40 FR 4893A (1975) (to be
and 1910.1025(1)(iii)(II).) Temporary
removal was not mandated, but obvious­
ly 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 explicit­
ly stated its intention to mandate
temporary removal of workers at risk
(42 FR 46547 (1977)). Subsequently,
both industry and labor representa­
tives readily endorsed the proposition
that temporary removal is an appro­
priate means of protecting workers
found to be at risk of sustaining mat­
erial impairment to health. (See, Ex.
354 responses to 42 FR supra, Quest­
ion 6: “Should employees be permit­
ted to remain on their job despite the
risk of material impairment of their
health?”)

2. Medical Removal Protection Bene­
fits.
   a. Introduction.
   b. Medical removal protection as a
   means of effectuating the medical sur­
   veillance sections of the lead standard.

The final standard requires that an
employer maintain the earnings, se­
iority, and other rights and benefits
of any worker temporarily removed
from current lead exposure due to the
risk of sustaining material impair­
ment until such time as medical surveil­
ance proves the absence of any signifi­
cant health risk. OSHA defines the
term "removal protection (MRP) ben­
fits," 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 as­
bestos and cotton dust standards. (As­
bestos, 29 CFR section 1910.1001(d)(2)(iv)(c)
(1977), Industrial Union Depart, AFL-CIO v. Hodgson,
160 U.S. App. D.C. 17, 461 F.2d 1230 (1972).)

The MRP program, as codified in 29 CFR
section 1910.1043(D)(2)(iv)(v) (1978) to be
For both of these reasons, the following sections of this
attachment discuss at great length the
reasons why OSHA has 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 ex­
plained in depth. OSHA has included
the length of this descriptive process in re­
sponse to the specifics of industry op­
position 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 nec­
essary price of doing business.

The final inorganic lead standard
contains a MRP provision for two rea­
sons. First, OSHA views MRP as the
most effective device for maximizing
meaningful worker participation in the
medical surveillance program pro­
vided by the standard. Second, since
temporary medical removal is funda­
mentally a protective, control mecha­
nism, 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 indi­
vidual 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 compel­
lng reasons for the adoption of MRP
provisions.

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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 sur­
veillance provided by the final stand­
ard. Before discussing the need to
maximize meaningful participation, it
is appropriate to emphasize the crucial
roles that medical surveillance will
play in preventing the health of work­
ers exposed to lead. Section 6(b)(7) of
the Act specifies numerous elements
that, where appropriate, must be in­
cluded in an OSHA occupational
health standard. These elements in­
clude the requirement of cautionary
labels, the prescription of protective
equipment and control procedures, the
provision of environmental monitor­
ning, and the specification of medical
examinations and other biological
tests. These elements along with the
rest of the OSHA Act demonstrate
OSHA’s mandate to promulgate com­
prehensive occupational health stan­
dards. Even where a lead-related occu­
pational health program cannot
succeed unless all elements ac­
complish their intended purpose.

Medical surveillance is a crucial com­
pONENT 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 pri­
mary control measures, such as engi­
nineering controls, to minimize worker
exposure to lead, but only medical sur­
veillance can determine the effective­
ness of these measures in protecting
specific workers. The detection of un­
expected or undesired health effects
can prompt the correction of inoper­
ative or ineffective controls. Timely and meaningful medical sur­
veillance can detect the early, revers­
able stages of occupational lead disease
so that treatment can be performed to
preclude permanent health impair­
ment. Even where a lead-related occu­
pational disease is not reversible, med­
ical surveillance still may serve to
identify workers who merit special
protection so that additional exposure
does not worsen or quicken the devel­
opment of disease. In all situations,
medical surveillance also serves the
important function of informing indi­
vidual workers of their personal
health status. Where a lead-related
disease has been or is being contra­
ed, 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 be included in medical surveillance. The evidence suggests that economic disincentives to worker participation are a problem in the lead industry. 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 that 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 Americans work for one simple reason—to provide for 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 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 occurred:

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. In job security, we have the most of most workers. Their experience demonstrates adequately that if they suffer abnormally on the job, they are removed. That is a fact. In fact, what the Act would eliminate this fear. Instead, the debate over the question of wage retention is essentially, if it is not successful, 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 is to be avoided at all costs, 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:

• • • (Our 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 single reason the worker is in the workplace in the first instance 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 price that that parent or parent of a family would not pay 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 painfully employed; or having their income drastically 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 convinced that the parents would do something, and certainly they would not do something, with one simple reason—to provide for 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 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 occurred:

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5008, 7179-7180, 7184-7186, 7219-7220; Ex. 165, p. 2; Ex. 374, pp. 126-133; Ex. 378, pp. 2-5; Ex. 390A, pp. 84, 105-106, 143; Ex. 432, pp. 5-7, 9, 11-13, 24), 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 Workers of America (Tr. 7282-7283; Ex. 379A, pp. 12-15, 32-33, 1301-1305, 1305, 3023, 3029), United Paperworkers International Union (Tr. 7603-7607, 7610, 7622), Oil, Chemical Atomic Workers International Union (Tr. 8059-8069, 8064, 8068-8069; Ex. 400A, pp. 3-5), United Electrical, Radio, and Machine Workers of America (Ex. 384D, 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), United Mine Workers of America (Tr. 8247-8248, 8434-8437, 8460-8441, 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 C.F.R. section 1910.1017(Ex.9)(1977)). Witnesses testified that there was widespread concern among vinyl chloride workers about potential adverse employment consequences of participation in offered medical surveillance programs provided by OSHA standards. OSHA's health standards are not heretofore contained in MRp. It is thus logical to conclude 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. 390B, p. 11.) 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 opportunity limited." (Ex. 390B, 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. 2) 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 proceedings of Dr. Daniel T. Titelebaum, a physician with extensive experience in occupational toxicology. (Ex. 354A, Ex. 354B, D) Dr. Titelebaum is also Director of Polkon­lab, 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. ** * * 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 on 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 as 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 OSHA in 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 long­run 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, workers who faced the possibility of losing their jobs were more willing to go to great lengths to avoid their worry about losing their job. 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 wouldn't want 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 It is al­so a common fear that worker. have. wheth­er it be textile workers, whether they are coal miners, whether they are steel workers. we run into it all of the time. in my opinion—what 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 very common in other industries. It is a general phenomenon. (Ex. 379A(A), pp. 1305-1306.)

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veillance programs had arisen under both OSHA's asbestos and vinyl chloride standards. (Tr. 7315-7316, 7356-7357; Ex. 3948, p. 12.) Numerous industry representatives described the lead problem existed in general (Tr. 7456-7457, 7468-7469, 7520-7521, 7759-7760, 7861-7882; Ex. 354H, p. 1; Ex. 354L, p. 1; Ex. 354(O), p. 5; Ex. 354U, p. 2; Ex. 354V, p. 1; Ex. 354PP, p. 1; Ex. 354HH, p. 5; Ex. 354P, Art. 2; Ex. 350A, 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. 3118), pp. 1-2), stated that its experience was that workers at risk often decline to participate in medical surveillance programs. (Ex. 3118, p. 11.) The National Association of Manufacturers agreed that the absence of MRP in many industries is a significant 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 for Employers, 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.

While 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 levels of adverse economic consequences. The lead record also amply documents the fact that such fear is often 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—approxi-mately half of the total work force under union contract in the industries examined. (Id., pp. 1-2.) The study reveals that some-50 of the 1724 contracts contain some kind of compensation benefit for health impairment. (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 health or safety reasons. (Id., p. 43.) Some 5 percent of the 1724 contracts contain such provisions, covering 4 percent of the 7,500 lead industry 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. 169, p. 65; Ex. 281, p. 21; Ex. 288, p. 33; Ex. 399, 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. 426, p. 23; Ex. 424, p. 12; Ex. 425, p. 6; Ex. 426, Art. 18C; 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(9), p. 10; Ex. 430D(13), p. 17; Ex. 430D(18), 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 and "MRP" and earnings compensation claims discussion, infra.)

The Bureau of Labor Statistics study, however, shows that the general rule throughout industry is that union workers temporarily removed due to health impairment can 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 form than nonunion 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 provide only 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 whose 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. 400D(9), p. 18) A worker in a battery manufacturing plant suffering 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, corporate policy promises a discharge to any worker unfortunate enough to absorb harmful quantities of lead. The Lead Industries Association has repeatedly proposed that "as a general rule a worker
should not be discharged because he has an elevated blood lead" (Ex. 287); 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)

**An 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 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/m³ 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/m³. (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 PbA/100g were to be encountered. At this primary lead smelter, the possibility of losing one’s job as a consequence 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 of Mining and Manufacturing, 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) when their blood lead levels repeatedly exceed 80 μg PbA/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 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. Indeed, 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/m³ OSHA standard. (Tr. 4720-4735, 1284, 3250; Ex. 3(26), p. 1; Ex. 3(36), p. 2; Ex. 3(44), p. 4; Ex. 3(50), p. 2; Ex. 3(76), p. 2; Ex. 3(69), p. 2; Ex. 3(93); Ex. 3(103), p. 9-10, 85-86, 88, 90, 92; Ex. 3(105), pp. 2-5, App. 1-4; Ex. 3(110), p. 1; Ex. 3(111), pp. 14-18; Ex. 3(127), p. 1; Ex. 4(61), 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 "inadequate reasons for 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 decrease meaningfully participation in inorganic lead monitoring activities. Workers 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, the temporary medical removal provisions 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 provisions 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 lead blood 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 200 μg PbA/100g (Ex. 3250, 50 pg/O0g. (Ex. 137, p. 1), the setting of explicit lead blood level removal criteria would effect the same result. (Ex. 4877, 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 standard’s removal criteria represent a point where many workers begin to experience clinical signs of lead poisoning. (See Tr. 716L.) 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 many firms to decline to voluntarily maintain the earnings of workers having “acceptable” blood levels.
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. This may not be a current 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 the MPPA threshold. The ultimate 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, 5537, 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 about 50 μg PbB/100g. Although OSHA is confident that a diligent company can provide substantial numbers of transfer opportunities which will satisfy the 30 μg PbB/m³ TWA requirement, always far from impossible. 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 industry 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. 425, p. 12; Ex. 428, p. 5; 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 these requirements) and 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 standard during which time medical surveillance programs for 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 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 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 in the course of 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 PbB/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 diagnosis, 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; 106A) are lead agents in nature. If a worker denenks 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 N. Merchant, 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-occupation- al 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 evidences manifested. I estimated that 80 percent of pathological conditions can be detected simply with proper administration and analysis of a medical history, even an abbreviated one.

Administration of such a history as part of a continuing integrated health care program is generally agreed to be well worth more in the prevention of disease than the extremely expensive laboratory tests given under the rubric of annual physical examinations. (Tr. 8214-8215; Ex. 404A, p. 2)

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 cases 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 is purely contrived. The patient doesn’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 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 on meaningful participation of 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 is significant only if a 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 1959 (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. 92-503, 86 Stat. 155 (1972) and Federal Coal Mine Health and Safety Amendments, Pub. L. 95-164, 91 Stat. 1290 (1971)) Experience under this program demonstrates that economic disincentives do adversely affect worker participation in medical surveillance programs, even where job transfers 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 transfers to low dust positions if they 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 37.5 (1977)) The X-rays are performed without cost to participating coal miners (42 CFR 37.3 (1977)), and the X-rays 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, 1969 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 to the position or guarantee that the miner will receive a position that 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. 7388)

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 100,000 miners have been X-rayed at least once, and over an opportunity every several years to develop of Black Lung have exercised the right to be transferred to low dust positions. (Tr. 7416-7417, 7429)
problems had been satisfactorily reported the second round of 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 (T1972) 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. 7384, 7412-7413, 8415), or from the possibility that some miners faced delay in completing X-rays as required by the law (Tr. 7384, 7368), or from the apparently inconvenient circumstances under which the X-rays were sometimes provided. (Tr. 8439-8440) The relative contribution of these factors to the lack of participation is unknown. Both NIOH 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) NIOH 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 concern to some miners. The second 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. 8426-8437) Dr. Lorin Kerr, director of the UMWA Department of Occupational Health (Tr. 8410), however, stated that even then the union had 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 from fear that negative X-ray results would be used against miners filing claims for Black Lung disability benefits (Tr. 8413, 8434-8437) - benefits which at that time were being processed at a very slow pace. (T1972) 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 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. 7429, 7449) 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, 7459), 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 position. (Tr. 7440-7441; 7430, 8412-8437), while still others might see no need to transfer if their transfer rights 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. 390a, 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. 7381, 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. (Tr. 7310, 8410) OSHA is convinced, however, that 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. Fear of adverse economic consequences has apparently significantly affected the decisionmaking of miners eligible to transfer to low dust positions.

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 will depend 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 health 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, let us 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 needed the legality and quality of worker participation in the medical surveillance program, as provided by the final standard.

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, and thus they needed the legality and quality of worker participation in the medical surveillance program. Industry comments are irrefutable 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 medical disposal to temporarily remove workers 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 foregoing 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 they will not be affected by occupational lead exposure or, if the company's existing economic protection policies differ little from MRP. OSHA also submitted 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 the management and the workers. 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 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 appropriately costs of doing business, thus employers should profit because 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 with respect to the economic incentive for employers to comply with the inorganic lead standard. In recent
years, increasing attention has been focused on the desirability of administr
ative regulations incorporating eco
mic incentives to compliance. (See, H. Owen and C. Schultz, “Setting Na
tional Priorities, The Next Ten Years,” 494-477 (1976); C. Schultz, “The Public Use of the Private Inter
est,” Harpur’s, May 1977, at 43-49.)

OSHA is convinced that MRP is essen
tial to maximize not just participation in
but meaningful participation. Absent
MRP, many workers will fear the pos
sible adverse economic consequences of
participation, therefore they will re
sist or refuse to participate in a mean
gorful fashion. Voluntary partici
pation cannot and will not change this
fact so long as the economic con
sequences 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 fa
ther a child should consider consulting an
examining physician in the course of
the next periodic physical examina
tion offered under the standard, or
then request an immediate physical exam ination to verify the ab
sence of MRP cannot serve to
prevent stillbirths, miscar
riages, and birth defects among the
potential children fathered by male
lead workers. Without MRP, many
workers will fear that if they come
forward and workers planning to fa
ther a child should consider consulting an
examining physician in the course of
the next periodic physical examina
tion offered under the standard, or
then request an immediate physical exam ination to verify the ab
sence of MRP cannot serve to
prevent stillbirths, miscar
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potential children fathered by male
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workers will fear that if they come
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potential children fathered by male
lead workers. Without MRP, many
workers will fear that if they come
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examining physician in the course of
the next periodic physical examina
tion offered under the standard, or
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prevent stillbirths, miscar
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potential children fathered by male
lead workers. Without MRP, many
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examining physician in the course of
the next periodic physical examina
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riages, and birth defects among the
potential children fathered by male
lead workers. Without MRP, many
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forward and workers planning to fa
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examining physician in the course of
the next periodic physical examina
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potential children fathered by male
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potential children fathered by male
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tion offered under the standard, or
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potential children fathered by male
lead workers. Without MRP, many
workers will fear that if they come
forward and workers planning to fa
ther a child should consider consulting an
examining physician in the course of
the next periodic physical examina

mual surveillance offered under the
standard. Second, OSHA could have
mandated that temporary medical re
movals occur only at the election of in
dividual workers. And, third, OSHA
could have permitted the use of respi
ratory protection in lieu of temporary
medical removals. The agency decided
to exclude MRP as a means of effectuating
any of these possibilities for the follow
ing reasons.

Mandating that workers participate in
medical surveillance.—This alterna
tive to MRP would require employers
to compel all employees to participate in
offered physical examinations and
biological monitoring. To meet this ob
ligation, employers would have to dis
cipline and ultimately discharge any
worker who refused to submit to medi
cal surveillance. Under this theory,
compulsion would be used to override
any reluctance a worker might have
concerning participation. Since partici
pation would apparently be assured,
there would seem to be no need to in
clude 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 remo
vals. Temporary medical removal is
fundamentally a protective, control
mechanism, and OSHA has deter
mined that the costs of this control
mechanism should properly be borne
by employers. This judgment is unre
lated to whether or not workers volun
tarily participate in medical surveil
lance, 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 suc
cess of the standard’s medical surveil
lance program depends. Mere partici
pation is not an end in and of itself.
The quality-of participation is crucial
due to the special nature of lead po
isoning. 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 essen
tial to maximize not just participation in
but meaningful participation. Absent
MRP, many workers will fear the pos
sible adverse economic consequences of
participation, therefore they will re
sist or refuse to participate in a mean
gorful fashion. Voluntary partici
pation cannot and will not change this
fact so long as the economic con
sequences of participation continue to
be of concern.
Mandating that temporary medical removals occur only at the election of the employer, due to 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. OSHA believed that providing workers the power to choose removal 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 specialized respiratory protection. These results are inconsistent with the preventive purpose of the Act, and thwart the level of health protection which temporary medical removals achieve. In its view, MRP would preclude any alternative to MRP which reduces the effectiveness of temporary medical removal as a protective mechanism.

Permitting the use of respiratory protection, including 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 respiratory protection 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 specialized respiratory protection, 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 alternative to MRP, 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.

To conclude, respirators are not an acceptable alternative to MRP 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 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 dictate 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' take 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 60 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.

**Possibility 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.
### Table C-1

**OBSERVED BLOOD LEAD DISTRIBUTIONS IN THE BATTERY INDUSTRY**

<table>
<thead>
<tr>
<th>FIRM</th>
<th>Percent of Workers in Given Blood Lead (ug/100g) Range</th>
<th>Total Number of Workers</th>
<th>Exhibit Number</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;40</td>
<td>40-49</td>
<td>50-59</td>
</tr>
<tr>
<td>Health Research Group</td>
<td>31.9</td>
<td>50.5</td>
<td>16.2</td>
</tr>
<tr>
<td>Delco-Remy</td>
<td>38.6</td>
<td>46.6</td>
<td>13.2</td>
</tr>
<tr>
<td>Chloride</td>
<td>1.5</td>
<td>9.1</td>
<td>31.8</td>
</tr>
<tr>
<td>UAW</td>
<td>16.0</td>
<td>26.0</td>
<td>18.1</td>
</tr>
<tr>
<td></td>
<td>10.1</td>
<td>18.0</td>
<td>19.0</td>
</tr>
<tr>
<td></td>
<td>7.0</td>
<td>67.0</td>
<td>16.0</td>
</tr>
<tr>
<td>NIOSH Study</td>
<td>33.3</td>
<td>37.6</td>
<td>22.4</td>
</tr>
<tr>
<td>Estee</td>
<td>10.6</td>
<td>21.7</td>
<td>30.7</td>
</tr>
<tr>
<td>Bell City</td>
<td>5.6</td>
<td>22.2</td>
<td>11.1</td>
</tr>
<tr>
<td>Battery Systems</td>
<td>64.4</td>
<td>22.2</td>
<td>8.9</td>
</tr>
<tr>
<td>General Battery</td>
<td>15.9</td>
<td>19.0</td>
<td>24.6</td>
</tr>
<tr>
<td>Gould</td>
<td>7.5</td>
<td>32.5</td>
<td>27.5</td>
</tr>
<tr>
<td>Teledyne</td>
<td>19.3</td>
<td>25.0</td>
<td>20.7</td>
</tr>
<tr>
<td>Trojan</td>
<td>30.8</td>
<td>31.9</td>
<td>23.3</td>
</tr>
<tr>
<td>Plates</td>
<td>13.4</td>
<td>21.6</td>
<td>37.1</td>
</tr>
<tr>
<td>Prestolite</td>
<td>--</td>
<td>--</td>
<td>33.3</td>
</tr>
<tr>
<td>C and D Battery</td>
<td>61.8</td>
<td>32.7</td>
<td>5.3</td>
</tr>
<tr>
<td>Globe-Union</td>
<td>--</td>
<td>--</td>
<td>3.3</td>
</tr>
<tr>
<td>Voltmaster</td>
<td>15.6</td>
<td>34.5</td>
<td>31.3</td>
</tr>
<tr>
<td>Total N</td>
<td>743</td>
<td>1040</td>
<td>761</td>
</tr>
<tr>
<td>Total Percent</td>
<td>20.2</td>
<td>28.3</td>
<td>20.6</td>
</tr>
<tr>
<td>D.B. Associates</td>
<td>--</td>
<td>69.2</td>
<td>14.7</td>
</tr>
</tbody>
</table>

*Note: The table provides percentages of workers in a battery industry with observed blood lead distributions. The data includes various firms along with the percentage of workers in different blood lead ranges, total numbers of workers, and corresponding exhibit numbers.*
<table>
<thead>
<tr>
<th>FIRM/PLANT</th>
<th>&lt;40</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>70-79</th>
<th>&gt;80</th>
<th>Total Number of Workers</th>
<th>Exhibit Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASARCO</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3(106)</td>
</tr>
<tr>
<td>East Helena</td>
<td>18.6</td>
<td>40.5</td>
<td>38.7</td>
<td></td>
<td>2.2</td>
<td>274</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Omaha</td>
<td>16.3</td>
<td>48.5</td>
<td>33.2</td>
<td></td>
<td>2</td>
<td>295</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glover</td>
<td>27.8</td>
<td>55.0</td>
<td>13.2</td>
<td></td>
<td>4</td>
<td>151</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whiting</td>
<td>38.4</td>
<td>52.0</td>
<td>9.6</td>
<td></td>
<td>0</td>
<td>125</td>
<td></td>
<td></td>
</tr>
<tr>
<td>El Paso</td>
<td>43.3</td>
<td>45.2</td>
<td>11.3</td>
<td></td>
<td>0.1</td>
<td>876</td>
<td></td>
<td></td>
</tr>
<tr>
<td>St. Joe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3(103)</td>
</tr>
<tr>
<td>Herculaneum</td>
<td>13.9</td>
<td>19.0</td>
<td>23.4</td>
<td>25.9</td>
<td>13.1</td>
<td>4.6</td>
<td></td>
<td>407(B)</td>
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<td>(76)</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>22.8</td>
<td>18.6</td>
<td>23.4</td>
<td>19.9</td>
<td>13.2</td>
<td>2.1</td>
<td></td>
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<tr>
<td>(77)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>J.W. Lorio</td>
<td>0</td>
<td>25.0</td>
<td>29.2</td>
<td>33.3</td>
<td>12.5</td>
<td>0</td>
<td></td>
<td>122(A)</td>
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<tr>
<td>Bunker Hill</td>
<td>25.6</td>
<td>16.7</td>
<td>25.2</td>
<td>18.7</td>
<td>7.3</td>
<td>6.5</td>
<td></td>
<td>299</td>
</tr>
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<td>(NIOSH)</td>
<td></td>
<td></td>
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<tr>
<td>Total N</td>
<td></td>
<td>(1415)</td>
<td>(835)</td>
<td>(75)</td>
<td>(3177)</td>
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<tr>
<td>Total Percent</td>
<td>26.8</td>
<td>44.5</td>
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<td>D.B. Associates</td>
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<td></td>
</tr>
<tr>
<td>Study Percent</td>
<td>19.0</td>
<td>21.0</td>
<td>25.0</td>
<td>20.0</td>
<td>11.0</td>
<td>4.0</td>
<td></td>
<td>N=3055</td>
</tr>
</tbody>
</table>

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### TABLE C-3

**DISTRIBUTION OF BLOOD LEAD LEVELS IN THE SECONDARY SMELTING INDUSTRY**

<table>
<thead>
<tr>
<th>SOURCE</th>
<th>&lt;40</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>70-79</th>
<th>&gt;=80</th>
<th>Total Number of Workers</th>
<th>Exhibit Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>California</td>
<td>17.1</td>
<td>61.6</td>
<td>20.2</td>
<td>1.1</td>
<td>99</td>
<td></td>
<td></td>
<td>86C</td>
</tr>
<tr>
<td>4 Smelters</td>
<td>17.9</td>
<td>15.4</td>
<td>33.3</td>
<td>33.3</td>
<td>78</td>
<td></td>
<td></td>
<td>86B</td>
</tr>
<tr>
<td></td>
<td>15.4</td>
<td>15.4</td>
<td>43.6</td>
<td>25.6</td>
<td>39</td>
<td></td>
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</tr>
<tr>
<td></td>
<td>3.4</td>
<td>17.2</td>
<td>6.8</td>
<td>72.4</td>
<td>29</td>
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</tr>
<tr>
<td></td>
<td>7.9</td>
<td>15.8</td>
<td>44.7</td>
<td>31.6</td>
<td>35</td>
<td></td>
<td></td>
<td>&quot;</td>
</tr>
<tr>
<td>Indianapolis</td>
<td>1.3</td>
<td>21.8</td>
<td>48.1</td>
<td>28.8</td>
<td>156</td>
<td></td>
<td></td>
<td>23 (Lillis)</td>
</tr>
<tr>
<td>ASARCO</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3 (106)</td>
</tr>
<tr>
<td>Perth Amboy</td>
<td></td>
<td>24.3</td>
<td>0</td>
<td>0</td>
<td>115</td>
<td></td>
<td></td>
<td>&quot;</td>
</tr>
<tr>
<td>Newark</td>
<td></td>
<td>36.1</td>
<td>15.8</td>
<td>0.8</td>
<td>133</td>
<td></td>
<td></td>
<td>&quot;</td>
</tr>
<tr>
<td>Keystone</td>
<td>51.4</td>
<td>21.6</td>
<td>27.0</td>
<td>0</td>
<td>0</td>
<td>37</td>
<td></td>
<td>430G-1</td>
</tr>
<tr>
<td>Total N</td>
<td></td>
<td>(212)</td>
<td></td>
<td>(178)</td>
<td>(116)</td>
<td>(724)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Percent</td>
<td></td>
<td>29.3</td>
<td></td>
<td>24.6</td>
<td>16.0</td>
<td>100</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Lead Industry Association Study**

- 38
- 24
- 19
- 19

*N=?” 354(AA)
TABLE C-4
BLOOD LEAD DISTRIBUTIONS IN OTHER LEAD INDUSTRIES
Percent of Workers in Given Blood Lead (µg/100g) Range

<table>
<thead>
<tr>
<th>INDUSTRY/SOURCE</th>
<th>&lt;40</th>
<th>40-49</th>
<th>50-59</th>
<th>60-69</th>
<th>70-79</th>
<th>&gt;80</th>
<th>Total Number of Workers</th>
<th>Exhibit Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pigments</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D.B. Associates</td>
<td>1.9</td>
<td>15.1</td>
<td>47.2</td>
<td>35.8</td>
<td>53</td>
<td>26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>King</td>
<td>26.4</td>
<td>14.7</td>
<td>44.1</td>
<td>11.8</td>
<td>2.9</td>
<td>0</td>
<td>34</td>
<td>234(22)</td>
</tr>
<tr>
<td>Zinc Smelting</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>St. Joe</td>
<td>76.7</td>
<td>16.7</td>
<td>5.3</td>
<td>1.3</td>
<td>0</td>
<td>0</td>
<td>150</td>
<td>3 (103)</td>
</tr>
<tr>
<td>Josephkton</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steel</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bethlehem</td>
<td>58.8</td>
<td>17.6</td>
<td>11.8</td>
<td>5.9</td>
<td>5.9</td>
<td>0</td>
<td>17</td>
<td>44, 237</td>
</tr>
<tr>
<td>Printing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R.M. Banknotes</td>
<td>91.3</td>
<td>8.7</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>23</td>
<td>38B</td>
</tr>
<tr>
<td>Lead Products</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NL Industries</td>
<td>78.2</td>
<td>18.0</td>
<td>3.8</td>
<td>2501</td>
<td>394C</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NL-Hoyt Plant</td>
<td>10.7</td>
<td>0</td>
<td>17.9</td>
<td>14.2</td>
<td>25.0</td>
<td>32.8</td>
<td>28</td>
<td>23(NIOSH)</td>
</tr>
<tr>
<td>Soldering</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Western Electric</td>
<td>100.0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>37</td>
<td>3(79)</td>
</tr>
<tr>
<td>Lead Chemicals</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eagle Pitcher (NIOSH)</td>
<td>5.9</td>
<td>5.9</td>
<td>17.6</td>
<td>5.9</td>
<td>23.5</td>
<td>41.2</td>
<td>17</td>
<td>113</td>
</tr>
<tr>
<td>Eagle Pitcher (Data)</td>
<td>15.4</td>
<td>48.1</td>
<td>36.5</td>
<td>52</td>
<td>38(C)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

FEDERAL REGISTER, VOL. 43, NO. 225—TUESDAY, NOVEMBER 21, 1978
The tables paint a bleak picture of existing worker blood lead levels, but the situation can and should rapidly improve. Several factors, all of which will be impacted by the new standard, currently exist to accommodate the lead industry in the past. For one year thereafter. This 80 pg blood level removal trigger is imposed immediately upon the effective date of the standard, and continues for one year thereafter. Thus, the economic impact of the 60 pg 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 under this standard to be reemployed in excess of 80 pg to be transferred to positions having an air lead level (without regard to the use of respirators) below 100 pg/m³. Abundant transfer opportunities already exist to accommodate these removals. (See, Ex. 26, pp. 3, 4, 5; Ex. 334, Tables 8, 9; Ex. 404B, Att. B; Ex. 407B, Ex. C; Ex. 430G-1, Tables 3, 4.) Greater transfer opportunities will also be present as the new standard is implemented.

The costs to employers of transferring small percentages of their workforce will be insignificant. As part of the lead proceeding, the Center for Policy Alternatives estimated that the one year direct cost of an 80 pg removal trigger (ignoring costs already regularly absorbed by existing industry transfer programs) to be as follows: (Ex. 439A, Table 7.1).

<table>
<thead>
<tr>
<th>Industry</th>
<th>Direct costs assuming transfers occur</th>
</tr>
</thead>
<tbody>
<tr>
<td>Battery Manufacturing</td>
<td>$500,553</td>
</tr>
<tr>
<td>Primary Lead Smelting</td>
<td>44,209</td>
</tr>
<tr>
<td>Secondary Lead Smelting</td>
<td>320,018</td>
</tr>
<tr>
<td>Inorganic Pigments Manufacturing</td>
<td>359,100</td>
</tr>
</tbody>
</table>

The Center for Policy Alternatives termed these costs "so low on an absolute scale that it is unlikely that the 80/60 (first year 80 pg) 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 290 pg/m³ standard.

70 pg blood level removal trigger 1 year after the effective date of the standard.—One year after the effective date of the standard, a 70 pg blood 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 and 92 pg need decline no more than 10 pg in 12 months—less than 1 microgram per month. As explained earlier, major blood lead 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 pg 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.
is clear that those workers initially removed with blood lead levels in excess of 80 $\mu$g should have declined to below 60 $\mu$g before the end of the first year of the standard, thus would be unaffected by the 70 $\mu$g removal trigger.

The Center for Policy Alternatives modeled the consequences of exposing workers having blood lead levels between 70 and 79 $\mu$g (average 75 $\mu$g blood lead level) to 50 $\mu$g/m$^3$ 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 $\mu$g/m$^3$ air lead exposure: (Ex. 439B, Table Ad. 2).

<table>
<thead>
<tr>
<th>JOB TENURE (yrs)</th>
<th>0-1</th>
<th>1-5</th>
<th>5-10</th>
<th>10-20</th>
<th>OVER 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVERAGE PbB</td>
<td>41.3</td>
<td>42.9</td>
<td>54.4</td>
<td>55.7</td>
<td>55.5</td>
</tr>
<tr>
<td>PERCENTAGE OVER 60 $\mu$g</td>
<td>2.4%</td>
<td>8.9%</td>
<td>16.4%</td>
<td>21.8%</td>
<td>27.1%</td>
</tr>
</tbody>
</table>

Assuming a normal distribution about each of these averages with a standard deviation of 9.5 $\mu$g, one can apply standard statistical tables to achieve the percentage of each job tenure group which would equal or exceed 70 $\mu$g 1 year after the effective date of the standard:

<table>
<thead>
<tr>
<th>JOB TENURE (yrs)</th>
<th>0-1</th>
<th>1-5</th>
<th>5-10</th>
<th>10-20</th>
<th>OVER 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>PERCENTAGE OVER 70 $\mu$g</td>
<td>0.2%</td>
<td>1.7%</td>
<td>5.1%</td>
<td>7.5%</td>
<td>11.5%</td>
</tr>
</tbody>
</table>

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 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. The 5-percent figure means the following: Of 100 workers with blood lead levels between 70 $\mu$g and 79 $\mu$g at the effective date of the standard, less than 5 workers should continue to exceed 70 $\mu$g 1 year later. Tables C-1 to C-3 indicate that workers with blood lead levels between 70 $\mu$g and 79 $\mu$g 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 $\mu$g.

60 $\mu$g 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 $\mu$g 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 $\mu$g and 60 $\mu$g will decline to below 60 $\mu$g. Two years is an adequate period of time to accomplish this goal.

<table>
<thead>
<tr>
<th>JOB TENURE (yrs)</th>
<th>0-1</th>
<th>1-5</th>
<th>5-10</th>
<th>10-20</th>
<th>OVER 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVERAGE PbB</td>
<td>41.3</td>
<td>42.9</td>
<td>50.7</td>
<td>52.9</td>
<td>54.2</td>
</tr>
<tr>
<td>PERCENTAGE OVER 60 $\mu$g</td>
<td>2.4%</td>
<td>8.9%</td>
<td>16.4%</td>
<td>21.8%</td>
<td>27.1%</td>
</tr>
</tbody>
</table>

The weighted average for a total worker population (using job tenure distribution as before) would be 4 percent. The foregoing suggests that of all workers originally having blood lead levels between 70 $\mu$g and 79 $\mu$g at the start of the standard, much less than 14 percent would exceed 60 $\mu$g 2 years later. Since workers now between 70 $\mu$g and 79 $\mu$g 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 $\mu$g.

The preceding "much less than 3 percent" figure would only be affected slightly by workers originally having blood lead levels between 60 $\mu$g and 60 $\mu$g. The previous paragraph's calculations indicated that less than 14 percent of these workers would exceed 60 $\mu$g after 1 year. One would expect most of these workers still above 60 $\mu$g to be very close to having a 50 $\mu$g blood lead level. One extra year should be sufficient for these few workers to decline one or two additional micrograms so that only extremely few individuals would still exceed 60 $\mu$g.

Six-month 50 $\mu$g average blood lead level removal trigger 4 years after the effective date of the standard. The standard provides that the 60-$\mu$g 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-$\mu$g removal trigger comes into force to shift the blood lead levels of employees still between 50 $\mu$g and 60 $\mu$g down below 60 $\mu$g. Two years were provided instead of some shorter period since the rate of worker blood lead level declines between 60 $\mu$g and 60 $\mu$g will likely be somewhat slower than the rate of declines between 70 $\mu$g and 60 $\mu$g. In total, however, a full 4 years is provided for all segments of the lead industry to achieve worker blood lead level distributions which would be 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 levels within lead plants necessarily effect immediately. OSHA is convinced that this trigger for temporary medical removal of 50 pg/m³ air lead levels 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 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.

**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.)

**Table C-5**

<table>
<thead>
<tr>
<th>Industry</th>
<th>Number of production employees</th>
<th>Average annual straight time earnings and benefits</th>
<th>Total cost exposed employee ($)</th>
<th>Cost per production employee ($/hr)</th>
<th>Cost per exposed employee ($)</th>
<th>Percentage of annual cash flow (percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary lead smelting</td>
<td>2,500</td>
<td>14,978</td>
<td>38.21</td>
<td>38.21</td>
<td>50</td>
<td>0.072</td>
</tr>
<tr>
<td>Secondary lead smelting</td>
<td>3,330</td>
<td>13,269</td>
<td>1.938</td>
<td>1.938</td>
<td>50</td>
<td>0.015</td>
</tr>
<tr>
<td>Battery manufacturing</td>
<td>17,800</td>
<td>16,727</td>
<td>1.264</td>
<td>1.264</td>
<td>50</td>
<td>0.002</td>
</tr>
<tr>
<td>Inorganic pigments manufacturing</td>
<td>2,915</td>
<td>15,053</td>
<td>2038</td>
<td>2038</td>
<td>50</td>
<td>0.008</td>
</tr>
</tbody>
</table>

**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:**

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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 cost of MRP has been lessened 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 plants with an MRP cost—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 µg/m² 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 industry have contended that MRP will have a far greater economic impact on smaller lead firms (Tr. 7460-61; Ex. 3540), p. 4; Ex. 385, pp. 11-12; Ex. 71A, p. 14). We would agree that in some instances companies such as small battery manufacturers might have lost 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 the large business 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 devices 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. Other smaller 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.**

---

**RULINGSB AND REGULATIONS**

Direct annual costs if 1 percent of work force is continuously laid off, and 1 percent is continuously on transfer status

<table>
<thead>
<tr>
<th>Industry</th>
<th>Total cost</th>
<th>Cost per exposed employee</th>
<th>Cost per production employee</th>
<th>Percentage of annual cash flow (percent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary lead smelting</td>
<td>$374,947.60</td>
<td>$169.16</td>
<td>$148.94</td>
<td>0.32</td>
</tr>
<tr>
<td>Secondary lead smelting</td>
<td>552,720.60</td>
<td>152.48</td>
<td>148.94</td>
<td>0.32</td>
</tr>
<tr>
<td>Battery manufacturing</td>
<td>2,331,824.10</td>
<td>151.34</td>
<td>142.22</td>
<td>0.75</td>
</tr>
<tr>
<td>Organic pigments manufacturing</td>
<td>551,191.20</td>
<td>175.60</td>
<td>119.25</td>
<td>0.35</td>
</tr>
</tbody>
</table>

The new lead standard will hopefully foster a higher level of concern for worker health by the overall lead industry. The new lead standard. Battery manufacturers might have little MRP expense to these firms. Lead level removal triggers in either lower blood lead level distributions. The remainder of the lead industry. The new lead standard-battery manufacturing, primary and secondary lead smelting, and plants with an MRP cost—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 µg/m² 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 does not feel that a short-term blood lead level elevation in excess of 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 situations, OSHA has determined that temporary medical removals are essential in situations where long-term blood lead levels are likely to significantly exceed 40 µg/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, and 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 after removal, and 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 and the absorbed lead. Some workers will take many months to decline from 60 μg/100 g of whole blood, with elevations above 40 μg/100 g of whole blood, and to reduce the effect of blood lead level measurement variability. The standard provides that the 6-month average removal trigger is 50 μg/100 g of whole blood average 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, indicates that the higher blood lead level is substantially above 40 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 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 significantly above the worker's longterm 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 a blood lead 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 trigger.

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 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 could easily be justified to provide a margin of safety in case a worker's blood lead level began to climb again. 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's 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-
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 also include temporary medical removals due to elevated blood lead levels as part of their medical surveillance programs. (Ex. 28, 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 76 μg (Tr. 7849-47, 8456; Ex. 354(III), p. 8) and 60 μg (Tr. 6202, 7629, 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. 452, p. 12; Ex. 452, pp. 52-55; Ex. 452, pp. 12-15.) Medical records indicate that 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 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 1910.1029(2)(3)(i)(B)(1.)) A similar exam was required every other month during pregnancy if the employee's blood lead level declined below 50 μg/100 g of whole blood. (40 FR 45934 (1975), to be codified in 29 CFR section 1910.1029(2)(3)(i)(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 a specific immediate symptom 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 anomaly which presents a risk to health or safety. Due to elevated blood lead levels, this removal criteria mandates that an employer remove a worker if the medical examiner recommends the employee at increased risk of material impairment 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. The use of temporary medical removal and return upon MRP mandates that an employer implement, and act in accordance with these limitations so as to protect worker health. The requirements that an employer follow such recommendations was included as 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 if those workers experience recognized symptoms of lead poisoning. Employers 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 may occur in a worker having a blood lead level below the removal criteria, or a worker acquires a temporary non-work 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 industry urged OSHA to provide voluntary transfers at the request of any male or female worker desiring to parent a child in the near future. (Tr. 691-92, pp. 1706-71, 1709-70; Ex. 148, pp. 16-16; Ex. 452, p. 60.) Instead of an automatic transfer opportunity, OSHA has determined that questions concerning reproduction can best be addressed first by primary control measures. The protective 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 alternative medical measures or temporary medical removals due to elevated blood lead levels, most of which workers will also be necessary.

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 industry parties and 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. 63-65; 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 45394 (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 in the MRP provisions or elsewhere in the Code, OSHA relies on informed medical judgment in the numerous contexts in which a particular lead exposure may be unusually hazardous to a particular worker.

OSHA's approach parallels that of ESB, battery manufacturer (Ex. 354(U), p. 1), who submitted the following comment:

- * (Blood lead levels are the primary criterion for removal under ESB's present medical surveillance program. That program also is effective in detecting and removing from lead exposure 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 the level 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 exist and place the determination as to whether an individual should be removed from his job in the judgment of a knowledgeable physician. (Ex. 354(D), 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 after 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. 767-81, 1829-30, 4721-32, 4901, 7905, 7797-99; Ex. 3(711), p. 10; Ex. 3(1055), 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 to be condemned 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 employees 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 that the removal be from work having an exposure to lead at or above the action level. The standard, however, permits lower lead exposure levels in the first instance, such lead exposure is to be computed with regard to the use of respirators. The lead record demonstrates that adverse health effects from lead exposure below the action level are also consistent with OSHA's intent to provide greater protection than that already at risk of material impairment to health even to workers not already at risk of material impairment to health. Workers removed as a result of their work experience, pose risks to some exposed employees. 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 would 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. 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 would 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 minimize 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 MTT’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. 413.) 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 has indicated that the position the worker would likely hold just before removal occurs is below the action level. The worker might work shorter hours at his or her former job, or work the daily time weighted average exposure 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 is a form of transfer). 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 the various alternatives be a prerogative of the employer unless this flexibility is altered by some counterbalancing obligation. A removed worker is provided no automatic right to veto an employer 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 statements in the lead record with identifiable durations, 3 have a 1-year term (Ex. 381(C); Ex. 300B (Texaco); Ex. 428), 8 have a 2-year term (Ex. 381(C); 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. 168; Ex. 261; Ex. 368; Ex. 385; Ex. 381(C); Ex. 383; Ex. 389; Ex. 400B (GATX); Ex. 400B (Kawasaki); Ex. 401(B); Ex. 404B(D)(4); Ex. 404B(D)(4); Ex. 404B(D)(6); Ex. 423; Ex. 424; Ex. 425; Ex. 427; Ex. 415(A); Ex. 415(B); Ex. 430(D)(1); Ex. 430(D)(23)).

c. Return of an employee to his or her former job status

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 has indicated that the position the worker would likely hold just before removal occurs is below the action level. The worker might work shorter hours at his or her former job, or work the daily time weighted average exposure 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 is a form of transfer). 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 the various alternatives be a prerogative of the employer unless this flexibility is altered by some counterbalancing obligation. A removed worker is provided no automatic right to veto an employer 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.

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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 union job bidding and transfer rights (T. 7462, 7471-73, 7511-12; Ex. 354(F), p. 2; Ex. 354(W), p. 2; Ex. 354(Y), p. 3; Ex. 383, pp. 15, 16, 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 was able to resolve difficulties presented by MRP. (T. 7634, 7789-90; contra, T. 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 confidence that MRP would not significantly disrupt industrial relations. (T. 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 a finding.


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 Amvax, Inc. (Ex. 351, p. 6) 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 to 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.)

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 stackers would have job skills peculiar to lead-related industries, and thus would likely not find al-
termed 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, thereby circumventing the standard. 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 regard to the control costs of temporary removal and then hire worker A back again with MRP benefits, then the lead standard has been complied with. OSHA does not intend to become involved in 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 dispute 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 review. Rather than expiration of the review period, 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 otherwise 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 earlier 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 laid off workers would 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 must lead 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 permit uncertainty in this area, the standard provides 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 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 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 he had not been laid off. 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 maintain the removed 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 week 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 will transfer to low 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-
iority is somewhat redundant since the phrase 'earnings and other employment rights and benefits' would be adequate to encompass all possible seniority interests. OSHA therefore explicitly concluded that the seniority language in new collective bargaining contracts may prove desirable, but major revisions should be unnecessary. Hearing participants agreed that at any given moment an employer is working, hiring, and firing workers and the worker will want to maintain a removed worker's seniority rights. (Tr. 7659-71.) Mr. Tomayko verified that due to their importance seniority rights are clearly spelled out in 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, pp. 68; Ex. 261, p. 28; Ex. 356, p. 7; Ex. 7664, p. 52; 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, (Art. 7649) and for 25 years director of the United Steelworkers of America, (Tr. 7662-64) also testified as to the importance of seniority to workers.

The standard requires that MRP and seniority interests can be smoothly integrated by affected employers, workers and collective bargaining representatives. The phrase 'earnings and other employment rights and benefits' would be consistent with this approach. The phrase 'employment rights' would avoid any implication, this rejects suggestions by the lead industry that the provision of MRP benefits would 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 MRP hearing 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.

OSHAA's rejection of an available position precondition is not primarily based on economic considerations, but rather on several other factors including the adsverse 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 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, employers would 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 the 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 would 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 MRP hearing 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.

OSHAA is convinced that MRP and seniority interests can be smoothly integrated by affected employers, workers and collective bargaining representatives. The phrase 'employment rights and benefits' may prove desirable, but major revisions should be unnecessary. Hearing participants agreed that at any given moment an employer is working, hiring, and firing workers and the worker will want to maintain a removed worker's seniority rights. (Tr. 7529-30, 7670-71.) Mr. Tomayko verified that due to their importance seniority rights are clearly spelled out in 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, pp. 68; Ex. 261, p. 28; Ex. 356, p. 7; Ex. 7664, p. 52; 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.
anteeing that all positions had an air lead exposure at or above the 30 g action level. Some long-term workers would achieve this result simply by waiting for OSHA to compel the implementation of engineering controls.

1 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(F), 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.

2. The lead record contains considerable evidence concerning the dynamics of blood lead levels. In excess of 80 µg/100 g to 60 µg/100 g, evidence from a variety of sources indicates that workers transferred to lower exposure jobs could accomplish such declines in 3 to 5 months. (Ex. 354(U), pp. 2-3; Ex. 354(AA), pp. 23-29; Ex. 354(DD), pp. 3-4; Ex. 354(HH), p. 10; Ex. 397(AA), 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 µg/100 g. (Ex. 354(U), p. 5.) It may take 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 relationships.)

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).

<table>
<thead>
<tr>
<th>Years exposed to lead:</th>
<th>Mean PbB before strike</th>
<th>Mean PbB after 4 months</th>
<th>Average PbB decline per month (µg/100 ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>More than 20 (n=17)</td>
<td>68.4</td>
<td>55.8</td>
<td>3.2</td>
</tr>
<tr>
<td>Less than 20 (n=21)</td>
<td>67.5</td>
<td>47.1</td>
<td>5.1</td>
</tr>
<tr>
<td>PbB before strike:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than 60 (n=10)</td>
<td>45.5</td>
<td>37.4</td>
<td>2.8</td>
</tr>
<tr>
<td>Between 60 and 80 (n=10)</td>
<td>70.3</td>
<td>55.2</td>
<td>3.8</td>
</tr>
<tr>
<td>Over 80 (n=9)</td>
<td>82.2</td>
<td>50.2</td>
<td>7.0</td>
</tr>
</tbody>
</table>

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 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 exposure 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. 492A, p. 4-13.)

<table>
<thead>
<tr>
<th>Number of years of lead time required to decline from 60 µg/100 g to 40 µg/100 g exposure prior to removal</th>
<th>Transfer to low exposure position</th>
<th>Layoff with no occupational exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Months of removal*</td>
<td>Average PbB decline/month</td>
<td>Months of removal*</td>
</tr>
<tr>
<td>0.55</td>
<td>0.7</td>
<td>28.6</td>
</tr>
<tr>
<td>3.4</td>
<td>3.3</td>
<td>5.1</td>
</tr>
<tr>
<td>9.0</td>
<td>7.7</td>
<td>5.0</td>
</tr>
<tr>
<td>16.0</td>
<td>11.9</td>
<td>1.8</td>
</tr>
<tr>
<td>23.5</td>
<td>14.5</td>
<td>1.4</td>
</tr>
</tbody>
</table>

*Assuming 30 days/month.
As with the Richer data, the MTI 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 MTI center for policy alternatives work provide an adequate basis for the 15 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 occurring 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 1978 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 that was obtained the day after retirement. The data can be presented as follows (PbB in µg/100 g): (Ex. 99(D), p. 83.)

The first four retirees excreted lead at a rate much slower than the average rate 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 9 years of removal to decline to below 40 µg/100 g. If, however, the same American lead worker excreted lead comparably to subject IH, 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 readily 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 lead worker's 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.

A worker whose blood lead levels will not decline to acceptable levels presents unique circumstances. Continued removal of a worker in these cases, continued removal of a worker will serve no major purpose since the information gone to the worker 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 future 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 far below the standard. 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 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 follow-up medical surveillance subsequent to return, and

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Years of prior lead exposure</th>
<th>PbB prior to retirement</th>
<th>PbB after period of retirement</th>
<th>Years on retirement</th>
<th>Average PbB when PbB decline/year measured</th>
</tr>
</thead>
<tbody>
<tr>
<td>JN</td>
<td>65</td>
<td>17</td>
<td>68</td>
<td>43</td>
<td>2</td>
<td>12.5</td>
</tr>
<tr>
<td>JB</td>
<td>71</td>
<td>22</td>
<td>65</td>
<td>58</td>
<td>5.5</td>
<td>10.4</td>
</tr>
<tr>
<td>MB</td>
<td>64</td>
<td>1</td>
<td>73</td>
<td>30</td>
<td>4</td>
<td>10.3</td>
</tr>
<tr>
<td>MB</td>
<td>64</td>
<td>2</td>
<td>73</td>
<td>30</td>
<td>4</td>
<td>10.3</td>
</tr>
<tr>
<td>AS</td>
<td>67</td>
<td>27</td>
<td>63</td>
<td>51</td>
<td>1</td>
<td>12.9</td>
</tr>
<tr>
<td>IH</td>
<td>67</td>
<td>22</td>
<td>68</td>
<td>42</td>
<td>0.5</td>
<td>5.5</td>
</tr>
</tbody>
</table>

The standards are 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 readily 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 lead worker's 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.

A worker whose blood lead levels will not decline to acceptable levels presents unique circumstances. Continued removal of a worker will serve no major purpose since the information gone to the worker 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 future 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 far below the standard. 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 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 follow-up 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 subsequently 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 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 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.

1. 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 provide 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 followup medical surveillance. 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 used 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. It is likely that over the years many have maintained an ability to maintain his blood lead level exactly at 40 µg/100 g, and thus participate 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 action. 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's 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.

1. 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), p. 3; Ex. 453, 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 the MRP program mandates 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. 397, pp. 32, 39; Ex. 358.) 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. 752.)

MRP, in stark contrast to workers' compensation law, is solely a preventive health program. MRP is activated before any disease results from 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 develop. 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. 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 such 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 and would also allow an employer to file 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 required to continue to provide MRP benefits pending disposition of any filed workers' compensation claim, subject to a claim for 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 Workmen's Compensation Law conducted a detailed study of employer supplements to workers' compensation. (“Employer Supplementation of State-Required Workers' Compensation,” National Commission on State Workmens' Compensation Laws, Supplemental Studies for the National Commission on State Workmen's Compensation Laws (1972) (hereinafter cited as “Supplemental Studies”).) The lead record contains such provisions appearing in collective bargaining agreements. (Ex. 158, p. 68; Ex. 386, pp. 44-45; Ex. 369, p. 4; Ex. 400(B), p. 58; Ex. 401(B), p. 39; Ex. 404(B)(D)(2), p. 17; Ex. 404(B)(D)(5), 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. XXI, Sec. 55; Ex. 450(D)(14), amendment to Art. XXII, adding new sec. 4; Ex. 450(D)(16), Art. XIII, sec. 1; Ex. 450(D)(17), p. 45; Ex. 450(D)(25), Art. XX; see, also, Tr. 2211, 7682-63, 7835-31, 8220, 8235-37; Ex. 354(D), p. 2; Ex. 365, pp. 33, 42-45, 62; Ex. 379(A)(15).) 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. 266 (table XI-A1.).) There is no evidence in the lead record, in the National Commission study, in treatises or articles on workers' compensation law, or on the law of the States, 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 employer need only establish that the worker actually received no earnings at all. To the extent that a removed worker may be affording by the standard will be afforded by the standard will be limited due to occupational exposure, this may occur, but they must be accompanied by 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 final proceeding. The relationship of lead contact to the effects of lead exposure on an employee's health remains a subject of concern for employers and employees. To the extent that an employer is removing or limiting workers due to occupational exposure to lead, this is likely that some employers would do this in the expectation of avoiding removal with MRP benefits. In this instance, the employee would be entitled to MRP benefits in the event the employer failed to negotiate the removal criteria more protective than those established by this final standard.

Some situations might arise, however, where any attempt by the employer to remove or limit workers due to occupational exposure to lead, (Ex. 354(AA), pp. 29-35.) The Lead Industries Association (LIA) and the American Occupational Health Nurses' Association (AOHNA) 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 engaging in such conduct, 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, 1059-60, 7306-08, 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 submission is that employers might somehow be incapable of 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 enforcing 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 chance of complete success where they are created by a process of consultation and cooperation between an employer and its employees. MRP and employer conditions "Not the Fault" of the employer. The LIA and other employers argue that MRP benefits should not be provided where the employer was not obligated to temporarily remove a worker 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 substantially aggravated by occupational exposure. The LIA offers no suggestions, however, as to how these situations could be administratively isolated from cases where occupational exposure is the basis for the removal of a worker. This issue
of lead should prove no more a problem in the future than it has been in the past. (Tr. 7720, 7476-77, 7625-27.)

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.” Accordingly, the Secretary must take into account the economic impact of the proposed standard. (E.O. 11218.) The final report from the ALC makes no pretense of conducting economic impact analysis of major government regulations. (E.O. 11218, 11949, 12044.) OSHA makes such analyses available to affected parties for comment 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 been advised by the following factors: the need to temporarily remove 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 an magnitude comparable to the substantial occupational exposure faced by many lead industry workers. (Tr. 3104-05.) OSHA recognizes that some people may develop permanent medical conditions which are not caused by 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 lives 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 threat of 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 non-work-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 street job exposure or 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 lead-exposed area—no matter how safe that area may be for others—if the employer has reason to believe that such exposure would significantly harm the employee’s health. Whether an employee has a preemployment condition such as anemia or later develops high blood lead levels because of hobbies or other outside activities, he should not be subjected to exposures which, although not harmful to others, are not safe for him. (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 not a punitive health mechanism. MRP in no fashion “punishes” 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 an magnitude comparable to the substantial occupational exposure faced by many lead industry workers. (Tr. 3104-05.) OSHA recognizes that some people may develop permanent medical conditions which are not caused by 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 lives 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 threat of 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 non-work-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:

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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 OSHA 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 on a draft of an IHE study performed for CRA. For the primary smelting industry, it relied also on an IHE study performed for LIA ("Final Report: Engineering Cost and Feasibility Study, Proposed OSHA Lead Standard," Ex. 29 (2A)). 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 Amad Leom Company's Missouri smelter (Ex. 3 (10B)). In addition to the CRA 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 procedures (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 CRA and CPA 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. 281) submitted to the record were considered along with all relevant comments and testimony.

2. Guidelines for determining feasibility. The OSHA Act has been characterized by the courts as technology-forcing legislation. Society of Parts Industries, Inc. v. OSHA, 509 F.2d 1301, 1309 (2d Cir. 1975) (vinyl chloride standard); AFL-CIO v. Brennan, 550 F.2d 109, 121 (3d Cir. 1977) (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 obligation to determine 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 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 plants in the same industry? For large and small plants? For the industry as a whole? The OSHA 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) (cokewaste 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 court recognized 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 subgroup 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 OSHA 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 are 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." (SF 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 industries that were not using it. Rather, standards may in effect require "improvements in existing technology" or "the development of new technology." (SF v. OSHA, 509 F. 2d at 1308.) 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 even if it is not mandated by a standard. 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 cokewaste 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 evasive. The case does not, however, stand for the proposition that OSHA cannot in fact require R. & D. to arise 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 purpose 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 technologies are prohibitive or because the technology is not fully developed or both, may become feasible for a longer period of time. Where the technology is 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.

Determining 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 the 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 determining that OSHA’s determination of infeasibility must consider “existing technological capabilities and imminent advances in the art.” Neither OSHA nor the courts noted the extent to which modifying the standard requires 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 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 the needed technology and in a shorter time period than industries without these resources);
2. The degree of change needed and its stage of development (the greater the change needed and the earlier its stage of development, the more time will be needed for compliance);
3. Certainty of product market (increasing demand and the absence of close substitutes would allow earlier compliance; other economic adjustments needed for conversion);
4. Size and complexity of plant or process requiring alterations (large-scale industrial processes may require more innovative and expensive modification); and
5. The experience of recent technological change in similar industries (the ability of similar 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.” (ALF-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.” (In re National Gypsum Co., 500 F.2d 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, OSHA v. Supreme Court 500 F.2d 477 (3d Cir. 1974) the “degree to which economic considerations can temper protective requirements.” 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 reaching an understanding 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. (2) The issue of inter-industry competition. Another economic issue which has been discussed in the case law concerning economic feasibility for each industry is increasing concentration in an industry. (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, 571 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 $1.41/lb. to $12.71/lb. (SPI v. OSHA, 509 F. 2d at 1303.)

Although there are several examples of feasible standards, there has been only one instance in which a standard was considered insecure. 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 "permits the existence" of the industry might be insecure. 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 employers could not comply quickly, a delay in the standard might be inappropriate 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 competitive is more feasible 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. 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 the case of products which are technologically infeasible, it is 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 concerning the feasibility of promulgated standards as they apply to individual firms. Although the issue is one of legitimate concern, it is clearly not positive in assessing the feasibility of standards. On the contrary, the appropriate unit of reference in assessing feasibility is in standard-setting. (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 developments may be necessary. In addition 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 contamination 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, 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 discharges 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 must be either "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 achieve levels in the inhalation sphere 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 6-12 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.) Dr. A'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

**RULES AND REGULATIONS**

(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 through efficient filter systems to prevent atmospheric contamination and reentry of dust laden exhaust to the work areas.

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 vibration, 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, raf ters, and cranes, as well as floors, walls, and machines. Stairs, ladders, platforms, and catwalks should be permanently installed to make all 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 secure reinstalled.

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) The use of enclosed conveying machinery generating airflows that are adequate to draw lead contaminated air from the work area. The design, construction, and installation of exhaust ventilation for the provision of controlled air velocity to draw clean air through the breathing area of workers and to prevent reentry of contaminated air to the work area.

(3) 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 through efficient filter systems to prevent atmospheric contamination and reentry of dust laden exhaust to the work areas.

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 airflow to clean air past a worker, through the contaminated zone, and then to sweep the contaminated material and air 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 any ventilation system, by wind or wind currents generated by the manufacturing processes.

In 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 discharges 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 must be either '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 achieve levels in the inhalation sphere 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 6-12 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.) Dr. A'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, 2338) and Knowlton Caplan of IHE (Tr. 3931–33) also noted time limitations in obtaining quotations on personal protective equipment 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 L.I.A. (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 quickly, 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 a need to implement the technology required, the effort to achieve compliance can be attained, the effort needed to promote widespread adoption, and the resources involved in making the change. In sum, OSHA has attempted to be sensitive to the complexities and various aspects 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 design characteristics of the technology. Such changes may be quick and effective, but they are not always the most cost-effective. 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 of that of a new plant designed with an existing plant not so well 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 are 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 it develops to respond 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.

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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 proposed 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 changes 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 ammonia 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 economic planning horizons; i.e., the time for firms to develop long-run solutions (or 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 long-run 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 long-run 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 long-run 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 industrial area is in industry-by-industry analysis below.

4. Industry analyses and technological conclusions. On the basis of all the evidence accumulated during the rulemaking proceeding, OSHA has determined that:

1. Compliance with the engineering control implementation schedule in paragraph (c)(1) of the standard, with the exception of the continuous reliance on personal 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. 208, p. 5-3)

Primary smelting involves three basic steps—sintering, melting, 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 (coké, fluxes, and recycled slag sinter) into molten lead and slag. Fifteen ton ladles on overhead bridge cranes transport the molten lead to open drawing kettles about 14 feet in diameter. The kettles rest on firebrick settings that keep the lead at the temperatures needed (700° to 1200°F) for drawing. During drawing, the molten lead from the blast furnace is stirred, and the impurities (dross) are collected and frozen 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 μg/m³ 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 the DBA and lead 100 μg/m³ level is unfeasible 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 the Third Circuit. (Tr. 3971-72; 795, 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 unfeasible because the Fairfield steel mill, used by OSHA as an example of the feasibility of the 150 μg/m³ level, 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. 5769;
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in whole to achieve a healthful work-
place.

Hydrometallurgical production methods are likely to be commercially viable within the 10-year limit, how-
ever, less comprehensive forms of proc-
ess redesign and/or adaptation of de-
velopmental projects discussed in the
section may prove to be sufficient. (Tr. 1463.)

The long-term economic conditions
facing the primary smelting industry are detailed in another section. How-
ever, it should be noted that the ex-
tended 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.)

It has been suggested that new technolo-
gies for primary smelting could be
operated at lower cost. The Bureau
expects that new processes will be
more economically feasible. (Tr. 5723.)

Frank Block, research director at the
Reno Metallurgical Research Center
for the Bureau of Mines, described one
such potential development, a hydro-
metallurgical method for recovering
lead from galena concentrate. (Ex. 138;
Tr. 3366-34-17.) This process does not
involve any sintering or smelting
and may require no refinement. It leaves
galena concentrate in a hot solution of
ferric chloride to produce lead chlo-
ride, which, in turn, is electrolyzed to
produce metallic lead. The new pro-
cess generates no sulfur dioxide. It
would be more economical than cur-
tent techniques and could operate at
smaller capacity. It could also be used
with Missouri or Western concen-
trates.

To date, this research has been con-
ducted in the laboratory on a small
scale. Block expects the process to vir-
tually eliminate exposure to lead since
the operation is closed circuit. Al-
though further investigation is needed.
This project is in its early stages of
development, but industry
seems to be very interested in its pro-
gress. It is expected to be fully opera-
ted on a laboratory scale, although there
are some potential problems that may
need to be solved. The Bureau
expects to run a large scale laboratory ex-
periment for a year or 18 months to
enable it to build a pilot plant. The
pilot plant could be built and operated
for 3 years, at which time there
should be enough experience with the
pilot plant to have developed a com-
mercially viable design.

(3) Specific Operations. (a) Concen-
trate Handling and Storage.—Concen-
trates brought from the concentrate
plant to the smelter are sampled to de-
termine their composition. They are
stored in large bins until mixed and
taken in batches as needed. Typically, con-
tainer belts carry the pelletized mixture to the
sintering machine. Exposures exceed 200
µg/m³ at many plants. (Ex. 26, pp. 5-3,
5-10.) The hearing record suggests
that the PEL for lead could be achieved for this
exposure. Typically, conveyor belts are
enclosed. Caplan, for example, found
no problem for the Buick smelter to comply
fully with the 100 µg/m³ standard. (Ex. 3 (106).) Edwin S.
Godsey, Chief Pneum and Dust Recov-
ery 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* Looking at the scale of these op-
erations, it is out of the question to
achieve 50 µg/m³. 

OSHA has concluded that compli-
ance 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. 370, 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 dis-
cussion of specific operations below.

Further, the degree of technological
change necessary to achieve 50 µg/m³
may require development and imple-
mentation of innovative technology,
possibly including alternatives to pyro-
metallurgy. OSHA believes that the
10 years provided in the reconsideration
schedule represent maximum flexibil-
ity for compliance by an industry
which may need, to rebuild in part or

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to 100 µg/m² 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 controls, work practices, and administrative controls may prove adequate. The record does not contain specific evidence about the techniques necessary to achieve a 50 µg/m² 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 µg/m² 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 tuyères are being charged. The passages through which air is blown or injected into the furnace, must be cleared of solidified slag or lead. Workers punch through the tuyère hole with iron bars or pneumatic hammers. (Ex. 3108.) At present, workers engaged in this task wear respirators.

Exposures at existing facilities range from 140 µg/m³ to 24 µg/m³. (Ex. 29, pp. 5-10; Tr. 6506; Tr. 6452.) The hearing record suggests that achieving an 8-hour TWA of 100 µg/m³ 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 µg/m³ 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 µg/m³ 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 tuyères, were ultimately successful, results would probably be on the order of 200 µg/m³. (Tr. 3108, 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 µg/m³ 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 local exhaust control of the "travel vent"; (5) redesign of the tuyère 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 puplits); 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 µg/m³ or 50 µg/m³ 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 reverber­atory furnace is itself a major source of contamination. Exposure levels range from 150 µg/m³ to 2,000 µg/m³. (Ex. 29, 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 µg/m³ level." (Tr. 5908.) Caplan described the adaptation 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. 5968.) 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 charged into a Berzelius® machine (a vacuum drossing machine). (Tr. 5712.) Leach electroy­lisis would, of course, also elim­inate exposures. Other controls include conventional hoods for the lead and matte top holes of the dross reverber­atory 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 dust control during refining are attributable to the nature of the process." (Ex. 300.) Exposures range from 100 µg/m³ to 2,000 µg/m³. (Ex. 29, p. 5-10.) One plant has, however, submitted data which indicated airborne lead levels of 50-100 µg/m³. (Ex. 26, p. 5-10.) It thus appears that a level of 50 µg/m³ 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 dross is a material that re­fining removes anti­mony and other elements and produces a product of lower hardness and strength. Many of the problems in dust control during refining are attributable to the nature of the process." (Ex. 300.) Exposures range from 100 µg/m³ to 2,000 µg/m³. (Ex. 29, p. 5-10.) One plant has, however, submitted data which indicated airborne lead levels of 50-100 µg/m³. (Ex. 26, p. 5-10.) It thus appears that a level of 50 µg/m³ could eventually be met in all refineries with conventional technology.

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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. 3108; Ex. 196, p. 10; Tr. 5696.) If so, the machine offers an approach that could also be implemented in other plants.

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Caplan also suggested that the use of the same controls for refinery operations as for dressing plants. These controls include the Hawley Trav-L-Vent, vacuum conveyors, scrubbers, controls for dust handling, etc. Other controls advocated during the hearing were: (1) Ventilated bins and ventilated enclosures for the dumping and handling of dress; (2) dress hoppers and conveyor systems; (3) fresh air supply islands or operator enclosures (stand by pupils); (4) smooth floors made of concrete or steel; (5) vacuum systems for housekeeping purposes; (6) dilution ventilation; (7) pipe-line systems; and (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.

(1) Crane and heavy equipment. Past experience shows that sufficient air-conditioning equipment can control the cabs of cranes, pay loaders, front-end loaders, dump trucks, sweeping machines, 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 \( \mu g/m^2 \) or 50 \( \mu g/m^2 \) is feasible.

(g) Baghouse, dust handling. Flue dust is a major source of contamination. According to Bergsøe, 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.) 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 hoppers, exposes employees in the area to high concentrations of dust. Periodic exposures between 50 \( \mu g/m^2 \) to 2,000 \( \mu g/m^2 \) have been reported. (Ex. 26, pp. 5-10.) Typically, control of flue dust is achieved by total enclosure in collection hoppers, conveyors, 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 stone.

Bergsøe'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. Bergsøe 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.)

(b) 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.

(2) Summary. The rulemaking record contains uncontested evidence that exposures in secondary smelting operations can be controlled below the 100 \( \mu g/m^2 \) 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 \( \mu g/m^2 \) 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 \( \mu g/m^2 \) standard as "technologically feasible." (Ex. 138D, p. 8) DBA, however, could not control battery breaking or other impurities.

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 \( \mu g/m^2 \)) specifications." (Ex. 339.) Before the implementation of engineering controls, average airborne lead at Keystone Resources was 1,036 \( \mu g/m^2 \). The controls reduced the average to 126 \( \mu g/m^2 \). (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 \( \mu g/m^2 \) level. (Ex. 26, p. 5-10, Tr. 696.)

Attaining these levels, however, may in a few instances require extensive modifications of current processes. IIEH, 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 \( \mu g/m^2 \) 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. 5-10.)

The rulemaking record describes new approaches that may be necessary.
To comply with the PEL, Michael Varner, corporate manager for ASSARCO's department of environmental sciences, and Melvin First, a professor of environmental health engineering at Harvard, discussed the possibility of innovations in dressing, such as continuous vacuum dressing. (Tr. 2387-88; Tr. 6530-31) Svend Bergsoe, president of Paul Bergsoe and Son of Glostrup, Denmark, in detail described the 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 smelts 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 broken, exposed, 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 generally needs 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 30,000 tons per year soft lead, guaranteed to meet a 100 μg/m³ standard. Bergsoe has built plants in many countries and is currently negotiating with American companies. (Tr. 5185-89.)

In addition to Capturing dust, 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 are being discussed." (Tr. 2376.) 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.) Slide 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 μg/m³ 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 μg/m³, NHEI 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. 3.) First described a design for a completely enclosed, ventilated, and remote-controlled system to separate lead from scrap batteries. (Tr. 2337.) Although Finlo'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 battery 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. 26, p. 5-1.) 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 the battery to bring their batteries into the United States. Absent successful mechanization of this process, administrative controls and occasional respiratory protection appears necessary for compliance with the 50 μg/m³ 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. 5-31.) Nonetheless, both DBA and NHEI concluded that such methods would at best achieve the 100 μg/m³ 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, charging by skip hoist, and hood the lead tap. Workers, who are generally required to wear respirators in this area, manually lead the skip hoist with lead, coke, and limestone material. NHEI observed the highest levels of lead in this area, 500-10,000 μg/m³. (Ex. 26, p. 5-31.) Despite such currently high levels, the record indicates that lead exposure in this operation can be kept below 100 μg/m³, 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 plan; and a clean air isolated area for refining furnaces; an isolated, ventilated area for slag handling; and provisions for airline or self-contained breathing apparatus." (Ex. 138D, p. 5-4.) 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. 5164.) Hot metal is tapped into a sump area so that there are no fumes coming into the building during the tapping stage. (Tr. 5160.)

By 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 /µg/m³ 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 flu dust. (Ex. 173.)

(c) Reversing furnaces. Some plants now use reverberatory furnaces to remove antimony from lead bullion. In the plants it studied, IHE 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. 5-32.) Worker exposure at isolated reverberatory furnaces, with administrative controls, probably could be controlled to a TWA of 50 µg/m³.

(d) 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 µg/m³ 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 µg/m³ or 50 µg/m³.

(f) Refining operations. Refining takes place, when metals are melted and treated in hemispherical pots to remove impurities in 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 pouring is required. DBA found that plants currently use hoods only during pouring. (Ex. 26, p. 5-32.)

Despite the technical problems in controlling refineries, IHE concluded that conventional technology could meet a 100 µg/m³ limit. (Ex. 138D, p. 5.) Such technology includes upgrader ventilation systems and hoods during charging and melting (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 transport. (Ex. 26, p. 5-32.) DBA also suggested that ventilation systems will need to be upgraded and hoods provided during charging and melting to meet the interim level. Careful hoisting 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 hoeding 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 µg/m³ level should not be difficult once adequate ventilation, local ventilation, and careful housekeeping are provided.

(h) Baghouse and 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 µg/m³ 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. (Ex. 26, Ex. 138D.) Such modifications in the process should control these operations to the 50 µg/m³ level. Furthermore the flash agglomeration of dust in the Bergsoe process would not be a problem in the 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. (Ex. 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 and maintenance of housekeeping. (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 µg/m³ 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 were shown to be under control, this equipment would need upgrading to meet a 100 or 50 µg/m³ 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 µg/m³. The 5-year period is based on the testimony of First, IHE, and DBA where estimates of 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
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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, only 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 electrolytic 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 dusts, as well as 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 employees’ air levels to below the PEL of 40 pg/m3 or less, 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 µg/m3, (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 µg/m3 standard.” (Ex. 34, 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. 1517.) The UAW asserted that “any operation in the battery industry plant, if controlled once provided with adequate enclosures, exhaust ventilation, or process redesign.” (Tr. 5274.) In his study of 17 plants, Thomas concluded that “the recommendations in the 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, 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. 3685.) 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) Spraying plates in are called vertical array with leakproof connections as well as local exhaust hoisting and control of ventilation flow;

(3) Mechanizing and enclosing wet and dry reclaim facilities;

(4) Enclosing of the ball mill racks and pallets after each cycle; and

(5) Cleaning the floor with a central vacuum system. (Ex. 26(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. 18.)

IHE’s spigot jalousie are designed primarily for larger operations. They assume that production is continuous and that operators remain at each work operation for a full shift, as 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. Miller, 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 Dyno­ lite 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 tested his employees in his plant, “The main controls that were effective for small plants. The record indicates that all the stations for grid casting, stacking, element assembly, battery assembly, and battery filling, only one reading at one location, element stacking, exceeded 100 µg/m3, and it was just slightly over, 110 µg/m3. (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.) 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 at all times every day. In fact, alteration of production schedules or employee rotation might be effective for small plants. Employees in small plants do not work exclusively at one station. As Stuart Manx 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, AFSEP, Inc., and Kermatrol, Inc., testified that they could provide the technology for compliance at 1/25th or reduced costs.—AFSEP, 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 Ker­ matrol, testified that his particulate filtration equipment could “guarantee attainment of a TLV of 50 micro­ grams, and even better, depending on the circumstances.” (Tr. 5211.) Its equipment consists of a layer of negatively charged HEPA (high efficiency particulate air filter).
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(Tr. 5205-5240; Ex. 176, 177.) This system permits cleaning and reuse of HEPA filters, which provide absolute filtration dust collection. Kermitrol recommends the combination of this filtration with the negative pressure enclosure of a "glovebox" glove box and return, wherever material is handled, storage, and scrubulous housekeeping will be attained. (Ex. 6 (45); see also U.S. Air Force study of the toxic residue in the plant have been cited as major causes of preventable lead exposure. (Tr. 503.) 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 pg/m³ standard.

(3) Specific operations. (a) Oxide manufacturing. — At present, exposure levels in this operation, which involves 2 percent of employees, are generally above 200 pg/m³. (Ex. 26.) For plants that manufacture ball mill oxide, available controls can reduce concentrations of lead in air below 50 pg/m³. In the 1994 public health service study, conventional ventilation control, isolation from other work areas, and vacuum collection of spilled oxide attained - of air, ranging between 60 and 100 pg/m³. (Ex. 6 (45).) IHE recommended not only isolating the process, but also establishing dust control at the mill trunion, the classifier, the oversize hopper, the classifier, the oversize hopper, and the vacuum collection systems. The UAW recommended that to maintain levels of 100 μg/m³ in larger plants, IHE endorsed a new design for oxide weigh hopper with a top air exhaust and a cage vent. (Tr. 3053.)

(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 μg/m³ and often in excess of 200 μg/m³. (Ex. 26, p. 5-47.) The record suggests that pneumatic conveying systems can maintain airborne lead concentrations as low as 50 μg/m³, 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 barreled dumping of oxide to 50 μg/m³ 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 barrel systems. (Tr. 5200; Ex. 101, 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. 15-13.) 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. 5375, Ex. 101A, 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. (Ex. 5603.)

(c) Paste-mixing. Lead exposure in paste mixing usually exceeds 100 pg/m³. (Ex. 26, p. 5-42.) The plants surveyed by IHE met the 200 μg/m³ 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 pg/m³, according to IHE, by installing extensive hosing and fa-

the industry to comply by installing extensive hooding and face-to-face exhaust fans. (Ex. 101A, p. 16.) The record suggests that, under certain conditions, approaches for meeting a level of 100 μg/m³ will also achieve a level of 50 μg/m³. In addition to isolation, preventive maintenance, and modifications to permit asiduous housekeeping, conditions needed to attain a level of 50 pg/m³ include enclosing the original access to the paste to test consistency.

(d) Pasting. Employee exposures in pasting operations generally exceed 100 pg/m³. (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 pg/m³ pasting departments in average-sized plants would require not only local ventilation at the feed, take-off end, and back 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 μg/m³ 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 μg/m³ 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 μg/m³, 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 μg/m³. These proposals included separating pallets used to transport grids from those used to transport plates; storing and transporting plates, elements,
oxide-containing scrap in sealed containers; and attaching exhaust systems to tanks for storing and transporting plates. (Ex. 180; p. 8; 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 schemes from the drawing board to assembly may prove the most practical means of reducing levels of lead in air to 50 \( \mu g/m^3 \).

(c) Grid and parts casting. Current exposures in this area, Tr. 5977, of the hearing record indicate 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. 5977.) 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 recommends that separate unit operations be isolated from each other. (Ex. 29(29A), p. 27.) Thomas noted that a downdraft ventilation greater than 100 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 be difficult in both technology and incentive work practices. Until such changes occur, administrative controls with some reliance on respirators may be necessary.

Stacking machines may not always be appropriate for small operations. (Ex. 6(45).) Incentive pay controls or work practices, exposures may go much higher. Incentive pay controls 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. A COS stacker to meet a COS 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 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 dust 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 on a timer and better ventilation 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 entering to the production areas. Consequently, there must be a barrel-washing station adjacent to the pour area. To eliminate dusting while dressing, the dressing operation must be conducted from outside the enclosure with the dressing 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 suggests that if the process is well-controlled, exposures in secondary operations would be below 100 µg/m³. (Ex. 29(29A), p. 37.) OSHA has concluded that a level of 50 µg/m³ 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.

OSH has concluded that conventional engineering controls and good practices are available to the battery manufacturing industry to meet the PEL. The compliance schedule of 2 years for the 100 µg/m³ interim level and 5 years for the PEL is based on both technological and economic factors. Since lead levels in the industry are based on both technological and economic factors, OSHA has concluded that conventional technology in the industry has been shown effective for lowering exposures from melting and pouring to 100 µg/m³. 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 continuous maintenance, employee training, work practices, and personal hygiene.

(3) Specific Operations. (a) Molding.—Because most foundries are small operations, no detailed discussion of the operations is necessary. However, the melting operation is described in detail in the discussion of the pouring operation. In the foundry, a number of related but separate processes are conducted which are important for the control of exposures to airborne lead. These include: (1) Molding and pouring: (2) Melting; (3) Pouring; (4) Finishing; (5) Cleaning metal and; (6) Storing of foundry materials. The following discussion of each of these operations will provide the necessary controls to be used in the foundry for the control of airborne lead.

(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. 28, p. 5-75.)

A combination of local exhaust and general ventilation systems is necessary to control airborne lead to acceptable levels. These ventilation systems have been demonstrated to be effective in 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, 6-90.)

(c) Pouring. Pouring can be performed at several stages. In general, the process is divided into three parts: (1) skimming; (2) pouring; and (3) cleaning. The process of skimming is 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 µg/m³. 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 molten pot. This dross should be deposited in a barrel with a mobile ventilation system that attaches directly over the barrel-washing station. 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 many 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, cements, high-grade paints, and paper pulp.

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 the plants the potential for lead exposure 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 µg/m³ (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, and 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 organic lead pigment. Litharge and other lead forms, however, are sometimes used. Because litharge is a powdery material, 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 µg/m³, although such methods have reduced air-lead levels to 200 µg/m³. 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 µg/m³ level. (Ex. 26, p. 5-96.)

The same conclusion applies to the 50 µg/m³ 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. 297, pp. 28-30). 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 µg/m³. A number of those who 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-paint manufacturing, ceramic glazes, and vitreous enamels. (Ex. 28, pp. 5-113, 5-114.)

(1) DBA concluded that 40 percent of these workers have exposures above 100 µg/m³. (Ex. 26, p. 5-113.) 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>
<thead>
<tr>
<th>Occupation</th>
<th>Total High exposure</th>
<th>Medium exposure</th>
<th>Low exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Welders</td>
<td>16,120</td>
<td>3,000</td>
<td>13,120</td>
</tr>
<tr>
<td>Painters</td>
<td>4,495</td>
<td>4,495</td>
<td>4,495</td>
</tr>
<tr>
<td>Sandblasters</td>
<td>904</td>
<td>284</td>
<td>622</td>
</tr>
<tr>
<td>Leadworkers</td>
<td>1,374</td>
<td>622</td>
<td>130</td>
</tr>
<tr>
<td>Total</td>
<td>22,255</td>
<td>3,808</td>
<td>17,447</td>
</tr>
</tbody>
</table>

(2) Summary. The hearing record indicates that attaining exposures of 100 µg/m³ 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. 1; Ex. 28(30), p. 3; Ex. 3(68), 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 µg/m³ standard.
and brake manufacturing. (Ex. 26, p. 5-119.) 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. (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 55 percent lead and 5 percent tin, is estimated to range from 2 to 30 pounds per vehicle. (Ex. 26, p. 5-135.)

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 pillar, 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. 25, 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. (2) To control exposures, the industry has developed exhaust ventilation, bulb dosing, or local exhaust ventilation. Grinders must also wear air-fed hoods known as hoods. The industry has thus combined engineering controls with elaborate personal protection equipment. The Motor Vehicle Manufacturers Association has 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 a success in reducing airborne lead 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. 16, stated that in his plant, workers 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. 5247-48.)

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 µg/m³ 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. Miler stated that "ultimately, the only solution is engineering the solder out of the car-body by redesign of the body and the final welding. That is, filling out the seam." (Tr. 5249.) Industry has already reduced solder in automobiles by substituting plastics and epoxies. One new line of cars has eliminated the use of solder in production. (Ex. 26, p. 5-135.)

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 Miler, "such exposures should be controlled by efficient local exhaust ventilation with downdraft or low velocity/slow air 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. (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. 1200.)

Unlike Mirer, the short report concluded that at least in some cases exposures may exceed 100 µg/m³ even in a adequately designed, operated, and maintained booth. (Ex. 22, pp. 228-29.) OSHA has concluded that maintaining 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 µg/m³ respirable lead-in-air for eight of its solderers. (Ex. 3(75).) Motorola also reported very low levels, 10-12 µg/m³. (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 µg/m³. Additional ventilation may be necessary at stations where solder is cut and trimmed. Motorola reports levels at these sites of 67 µg/m³ and 73 µg/m³, slightly above 50 µg/m³. (Ex. 3(66).)

1. Solder Manufacturing (1) Introduction.—There are 100 companies operating 120 plants that manufacture solder. The companies manufacture 90 percent of the solders. 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 established 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 temp
perature is kept at 600-850°F, where few fumes are formed, because lead oxide, which ruins the solder product is formed. The process is repeated until the lead is completely melted. 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 10,000 people in the solder 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 pg/m³ in spooling, wire drawing and soldering operations, and kettle areas. (Ex. 65B, p. 42.) One company reported that even with excellent ventilation, lead levels in the casting area reached 200 pg/m³. (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 air through an air nozzle. 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 soldering and wire drawing operations, where lead-air levels range from 100 to 300 pg/m³. (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 shape or by separating the scrap more completely, 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. OSHA 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. OSHA concluded that the likelihood of exposures depends on the amount of lead in the raw materials, the care with which the 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, 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.)

(c) Ink Manufacturing. (1) Introduction.—Approximately 100 of the 479 plants that manufacture inks use lead pigments; 1,000 to 1,500 employees are potentially exposed. During production, 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, fill covering.

Ink manufacturing, the principal lead pigments are lead chromate, molybdate orange, and phosphate red. OSHA estimated the 1976 consumption of these pigments in the captive and noncaptive sectors to be 50 million pounds of lead chromate; 8 million pounds of molybdate orange, and over 2 million pounds of phosphate 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), OSHA 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 in their manufacture, 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 large amount of pig iron and sand in 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. OSHA suggested that most of the lead, which is a nonferrous constituent, enters the process through particular types of obsolete scrap, such as bearings or other parts of automobile engines, pipe fittings, lead-based paints, solders, and leaded glass. (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 50 people; about 20 more than 1,000. (Ex. 65B, p. 24.) Most gray iron foundries, especially the small ones, pick over their scrap before melting, although in large automated foundries heavy machinery may handle the scrap. Also, employees break any lead or other 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, solders, and leaded glass. (Ex. 65B, p. 25.)

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following characterization of exposures: (Ex. 65, p. 38).

<table>
<thead>
<tr>
<th>Number of exposed employees</th>
<th>TWA exposure to lead in air µg/m³</th>
</tr>
</thead>
<tbody>
<tr>
<td>220 to 416</td>
<td>200</td>
</tr>
<tr>
<td>450 to 624</td>
<td>100-199</td>
</tr>
<tr>
<td>250 to 269</td>
<td>65-99</td>
</tr>
</tbody>
</table>

(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 (cans, etc.) will require close attention. OSHA does not anticipate that there will be exposure to lead in excess of the permissible exposure limits associated with 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 chromate is an essential pigment 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 ink containing lead. Such a pigment was used 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 OSHA 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 have ink containing lead. Such products, however, do not pose a major exposure problem. OSHA 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. OSHA 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. OSHA 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 µg/m³.

n. Wire Patening. (1) Introduction.—Wire patening 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 fluxes from the quenching pot 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 µg/m³ limit, very efficient ventilation systems along with necessary housekeeping programs, will be required to control lead exposure. Two companies reported current compliance with the 100 µg/m³ limit in 1972 (Ex. 65B, p. 22). Thus, the 100 µg/m³ level clearly appears feasible.

The Stelmor process, which uses air as the quenching medium, eliminates the molten lead problem. Approximately 25 steel works are now using the Stelmor process, and it appears to be replacing lead wire patening. (Ex. 65B, p. 22.) Through either this new technology or through aggressive implementation of engineering controls, the 50 µg/m³ 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 can is dipped in a molten lead bath or a roller lays solder on the can. A rapidly rotating cloth-covered spindle then wipes off the excess. The cans are dipped, a flux is layered on the top of the solder, and the cans are 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 µg/m³ 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. An upset occurs when the bath is being charged, but 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. 193.)

(3) 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 pg/m³ 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 borosilicate, lead glass or bisilicate. 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 µg/m³ could occur in handling and mixing of frit as well as spray 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 spray operations. (Ex. 22, pp. 221-222.) 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 follow-up 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 µg/m³ level are equally applicable to the 50 µg/m³ 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. Where lead exposure is potentially engaged 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-
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 proposed new lead emission standards. Costs and impacts attributable to the proposed standard were an aggregate of costs associated with environmental monitoring, medical surveillance, training, recordkeeping, operation and maintenance of capital equipment, housekeeping, and other processes. All costs attributable to the proposed standard were included 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. Burton's effort was to provide a specific control efficiency, but not presently being complied with new requirements of the standard should be calculated, but not presently being complied with new requirements of the standard should be calculated, but not presently being complied with new requirements of the standard should be calculated, but not presently being complied with new requirements of the standard should be calculated, but not presently being complied with new requirements of the standard should be calculated.
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 applicable to those industries where the capital costs of process equipment are not reduced with minimal opportunity for recovery of the costs when control strategies involve the modification or replacement of existing production equipment, but if the new process configuration is in conflict with the current process. Given the time and resource factors in collecting the data: but from data sources that are limited and necessary 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 supplying detailed information 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. 6-12). In the secondary smelting industry, all but three of the seven plants studied were company estimates (Ex. 65c, addendum to table 5.15); in the battery segment, the control data came 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 cases 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 overall estimate was not supplied. This was primarily because companies' desires to keep the information confidential for competitive reasons or because companies believed disclosure would result in a compliance activities (Tr. 748; Ex. 65A, p. 4; Ex. 26, pp. 5-59, 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. 

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 the costs. In the primary smelting industry where estimates for each plant were obtained, the "best" estimate for the industry is the aggregate of estimates for each plant (Tr. 626-628). The "low" estimate was based on the lowest per employee and per ton of production cost found in the industry multiplied by the total number of employees and tons of production: likewise, the "high" estimate was based on 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 using the geometric mean of the costs per employee and per ton for each plant in the sample. (See, e.g., Ex. 25, p. 5-40).

The high and low estimates are not considered to be especially meaningful in estimating incremental costs in particular in the primary smelting industry where the best estimate is the total of individual estimates for each plant in the industry. (Tr. 629) 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 industry as old DBA (DBA independently estimated one plant's costs). CRA calculated costs in the secondary smelting industry by extrapolating from eight plant estimates, seven of which were identical to the seven that DBA had used (Compare Ex. 127, Exec. Summ., p. 25, table 5 with Ex. 26, p. 5-50, 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 neither engaged in the concept 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... " (Ex. 127, p. 2-38). 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 of the proposed capital costs (Ex. 127, p. 3-29), CRA stated that its 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. 2-39).

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 a per million dollars concentration of lead. 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. 237) In the primary smelting sector, CRA's figures are lower because of 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 1-4, 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 that CRA engaged in the same forms of 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 46 percent. It is the inclusion of the tax savings and attributable to deductions for annual expenses that accounts for the difference in industry estimates. Working backwards, if CRA's total annualized cost of $28.2 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 $8.259 million for the short term and $5.836 million for the long 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 IHS (Tr. 5371). 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 $10.8 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 µg/m³ 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 µg/m³ PEL as it did for the proposed 100 µg/m³ 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 µg/m³ 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 µg/m³ 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 µg/m³ interim level. (Table 2.)
<table>
<thead>
<tr>
<th></th>
<th>CAPITAL COSTS ($MN)</th>
<th>RECURRING ANNUAL COSTS ($MN)</th>
<th>ANNUAL CHARGE TO CAPITAL (%)</th>
<th>PRE-TAX TOTAL ANNUALIZED COST ($MN)</th>
<th>AFTER-TAX TOTAL ANNUALIZED COST ($MN)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>DBA</td>
<td>DBA</td>
<td>DBA</td>
<td>DBA</td>
<td>DBA</td>
</tr>
<tr>
<td>St. Joe</td>
<td>7.500</td>
<td>10.627</td>
<td>2.211</td>
<td>1.414</td>
<td>N.C. 3.071</td>
</tr>
<tr>
<td>Amax</td>
<td>9.540</td>
<td>8.144</td>
<td>1.814</td>
<td>1.365</td>
<td>N.C. 2.634</td>
</tr>
<tr>
<td>ASARCO (Total)</td>
<td>20.830</td>
<td>19.247</td>
<td>5.833</td>
<td>1.823</td>
<td>N.C. 4.822</td>
</tr>
<tr>
<td>(ASARCO/Omaha)</td>
<td>(4.500)</td>
<td>(4.153)</td>
<td>(1.209)</td>
<td>(0.425)</td>
<td>(N.C.) (0.647)</td>
</tr>
<tr>
<td>(ASARCO/East Helena)</td>
<td>(5.900)</td>
<td>(5.499)</td>
<td>(1.444)</td>
<td>(0.491)</td>
<td>(N.C.) (0.857)</td>
</tr>
<tr>
<td>(ASARCO/El Paso)</td>
<td>(5.250)</td>
<td>(5.483)</td>
<td>(2.060)</td>
<td>(0.498)</td>
<td>(N.C.) (0.752)</td>
</tr>
<tr>
<td>(ASARCO/Glover)</td>
<td>(5.180)</td>
<td>(4.767)</td>
<td>(1.120)</td>
<td>(0.373)</td>
<td>(N.C.) (0.743)</td>
</tr>
<tr>
<td>TOTAL</td>
<td>56.270</td>
<td>47.254</td>
<td>12.507</td>
<td>5.836</td>
<td>12.373 7.363</td>
</tr>
</tbody>
</table>

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

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RULES AND REGULATIONS

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\(^2\) interim level is 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 usually 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 (BPA type) regulations (Tr. 4655-38, 4683-54, 4041-43), and one-third of the total $4.9 million estimate for engineering controls at St. Joe's was for baghouse expansion and renovation, a project that is essentially directed toward control of existing emissions 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. 4607) because ASARCO did not distinguish double counting costs from 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\(^2\) interim level by one-third for all forms of double counting is reasonable.

Total annualized cost estimates are likewise overstated since a major component is the annual charge to capital. Since the capital costs are estimated to be lower, so should the total as is its interest. decrease in estimated interest 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—such as much as 10 percent of the capital costs and 70 percent of annual recurring costs. (Ex. 127, 127.)

OSHA's revised estimate for capital costs is between $32 million and $47 million, the operating costs, reduced pretax, 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) to the revised estimate of pretax, total annualized cost at the 100 \(\mu\)g/m\(^2\) 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 $6,716.48 million and $8,513 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\(^2\) level, based on its total 1975 production, would be approximately $4.6 cent to 0.8 cent per pound. On a per employee basis, it is $2,000 to $2,062 per year.

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. They are custom smelters, smelters, and one is a refinery. The other two smelters are custom smelters, and one is a refinery. The other two smelters are custom smelters, and one is a refinery.

(2) The ability of the firms to shift costs forward into price or backward onto the factors of production; the market power of the primary producers; the market structure in which the primary producers operate; and the factors that affect the market power of 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, Exe. Summ., p. 17.)

The supply of ores and ore concentrates is also diversified. 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 2-18.)

(2) The ability of the firms to shift costs either forward or backward depends on the amount of substitutes for lead in the market 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 are storage batteries, whose prices account for approximately 48 percent of lead consumption in the United States. The demand for batteries is relatively inelastic, being largely a function of the numbers of automobiles and trucks. Since there are presently no substitutes for lead in the production of batteries,
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 in-creasing. Thus, the long run signifi-
cance 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 per-
cent 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 there-
fore, 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 indi-
cated that demand was relatively inelastic (Ex. 127, p. 2-50). It then re-
ported 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 Hei-
ek e'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 of-
fered of why monthly data would pro-
vide more accurate and substantially different estimates of long run elas-
ticity.

Under questioning, Dr. Burrows of CRA repudiated Heineke's work (Cr. 3378). He did point out that previous CRA studies had been aimed at deter-
mining 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 estimate is valid and that CRA's initial observation that long-
term demand for lead in the United States is relatively price inelastic is cor-
rect.

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 permits to explain consumption of lead ingots in the United States on the basis of changes in national income and do-

mestic price. Time lags are explained by contracting procedures common to the industry (causing sev-
eral 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 essen-
tially correct if the market demand was essentially the result of business and consumer decisions in a reason-
ably competitive and free market. However, it ignores several other influ-
ences at work in the market during the period (1949-1965):

a. The Federal Government main-
tained a national security stockpile of lead ingots, purchasing or selling lead in the market at various times on the basis of policy decisions not determined by considerations of the economics of ma-
terial substitution in industrial pro-
duction relative to the price of lead.

b. There are two 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 consump-
tion.

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-
lly of the price or supply of lead, caus-
ing some to substitute other ma-

terials for lead.

These other market forces are not randomly distributed and do not aver-

age out. They could account for a sig-
nificant portion of the price elasticity of demand as measured by Heineke. Fur-

more, 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 significance to the determination of 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 realationship 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 the LME price. In the long run, it was asserted, this would prevent do-
mestic producers from raising prices to recoup the costs of compliance with the lead standard. Several factors could operate to minimize the possibil-
ity of price increases, e.g., an elastic response to a small increase in the dif-
ferential.

World demand has been increasing steadily. In the 10-year period between 1965 and 1974 world consumption of refined lead increased from 3,182,260 to 4,350,300 metric tons an-
ually. (Ex. 127, Table 1-1.) In part this reflects the growth of the auto-
mobile as a primary means of trans-
portation 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 (65 per-
cent) and Asia (25 percent) since 1965 (Ex. 127, p. 1-2). CRA asserts that lead has been produced on a con-
stant or declining cost basis and as-
sumes such will be the case in the future. In order to make this argu-
ment, they constructed a graphic rep-
resentation 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 prices appears to tend toward a rather consistent price range. CRA does not offer an econometric analysis of this price be-

havior. They conclude that it is ex-
plained by long run production at con-
stant 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 as-
sumes 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 re-

sponse to rising prices. Absent the shifts in supply which result from new discoveries, there is every reason to be-

lieve that the normal attribution 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. 1-20.) 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 methods. New Missouri lead belt produces 80 percent of U.S. lead ore, with 90 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 extramarket 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 occurring, 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. 2-10 to 2-13.)

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 prices 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, earlier. Further mines could now be considered marginal.

It should also be noted that increasing smelter charges to meet environmental costs have preceded 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.

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 hierarchy.

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 OSHA 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 45.2 percent, while ASARCO's was 0.740, Amax's 0.818, and Gulf Resource's 1.342. (Ex. 26, 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 $19,646 million, or 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. 345, p. 173.)

Income data was also presented for 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. OSHA's analysis assumed essentially that the costs of compliance with the 100 µg/m³ interim level were to 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 µg/m³ 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. 6-35). 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 may be rather 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. (25) 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 conclusion reprove 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. It remains to be seen whether the cost premium of lead 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, Amex, 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(107), p. 5.)

(3) 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 companies which CRA did not evaluate backward shifting of costs. The extent to which this could be accomplished minimizes the cost impact on the primary producers.

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 favorable and there exists some combination of both, a time frame for 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. 28, p. 27) of the industry.

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.
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 was affected by the regulation 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 firm's 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. From the record evidence as a whole, it appears that CRA compared profit and debt to costs can not be passed back to suppliers. It uses for the Bunker Hill group of companies its profit estimates for the parent company's financial condition. Bunker Hill Co. or on their or, other companies or on others' financial information on the parent company.

The parent company's profitability in 1976 and Amax are still higher than the other firms', but it will be even lower if adjustments were made for the other firms, St. Joe (p. 172) and the result is that the DBA's conclusion that Bunker Hill, which has determined to be reasonable. They found that one of the firms' financial condition. 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, therefore, 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. Therefore, stating that the DBA's profitability would be reduced, DBA was not able to determine whether any primary producer would curtail its lead operation.

From the firms submitting data on their lead operations alone, St. Joe's return on sales was claimed by the Steelworkers to be 94.1 percent, indicating its viability. Bunker Hill's decline in profitability in 1976 to 8.6 percent was attributed to a decrease in lead production 20 percent below capacity and state environmental standards and a "continuing softness in the zinc and by-products markets." 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 misleading because it fails to isolate effects attributable to the proposed OSHA lead standard and bases its conclusions on the simple profit analysis of the proposed OSHA/EPA-type costs equivalent to 1.54 cents per pound of refined lead produced.

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 50 percent of operating capacity (126,000 short tons) will be between $579,000 and $2,016,000 annually.

Looking then at profitability, CRA concluded that if Bunker Hill was forced to absorb $5 million in annual costs, 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 $10.6 million over and above the costs of compliance. Absorbing costs of $5.79 to $2.016 million would cut into profits, but those costs are only 5 percent to 19 percent of the firm's average profits. This mitigates CRA's conclusion that DBA was not able to continue to operate profitably. Similarly, Amax was expected to continue to operate, although absorbing approximately $1.5 million in 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 the Missouri ores and would have to absorb the entire cost of compliance. This would have the lowest compliance cost and the highest remaining processor of non-Missouri ores and would have to absorb the entire cost of compliance. This would have the lowest compliance cost and the highest.

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: monopolistic result as a consequence of the mutually recognized interdependence of the four large producers. This could occur without

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**RULES AND REGULATIONS**

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<thead>
<tr>
<th>Company</th>
<th>Capital costs</th>
<th>Annual costs</th>
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<td>(percent)</td>
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<tr>
<td>ASARCO</td>
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<td>Gulf Resources</td>
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</table>

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The degree of concentration in primary refined lead production is already potentially high enough to achieve: 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 discount entry. These forces would still be operating no matter what the degree of concentration in primary refined lead. 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 pg/m³ level in this industry are roughly similar as indicated below:

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<tr>
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<th>DBA</th>
<th>CRA</th>
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<td>Total capital costs</td>
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<td>Total pre-tax annualized costs</td>
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<tr>
<td>Total after-tax annualized costs</td>
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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 based on actual production costs. 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 collection phase 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. 6-16) 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 pg/m³ standard. In 1976, the estimated cost for a secondary smelter producing 10,000 tons per year to comply with the 200 pg/m³ standard [31] Capital costs were given as $324,200 and annual operating and maintenance costs were $32,650. The capital cost 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 pg/m³ standard. These costs reach 200 pg/m³ may or may not be typical of the secondary smelting industry. However, with the IHE three plant study in the 'battery' industry (Ex. 139, p. 2-7) coupled with energy savings from current battery recycling, 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 anticipated 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 pg/m³ interim level. For the less efficient producers, particularly those energy savings from the Bergsøe process. Those smelters generate over 50 percent of their energy from the burning of the battery cases. Coupled with energy savings from current battery recycling 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 to Compliance.—Cost estimates for compliance in this industry were presented by DBA and CRA. Capital cost estimates, attributable entirely to engineering controls, were $345 and $907 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 CRSA. (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, and individual manufacturers resulting in grossly exaggerated capital 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 200 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 µg/m³. 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 µg/m³ level.

From this, costs were estimated for each operation. As mentioned earlier, the capital costs were used by CRA to obtain compliance estimates for its 63 plant samples and by individual manufacturers who presented cost information.

The capital 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 µg/m³ standard, a substantial amount of the cost should be attributable to meeting the present standard. An indication of the magnitude of this amount 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 over the 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 µg/m³ (even if they were minimally above) would be totally uncontrolled and that entire, new ventilation 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" capacity but in many cases existing dust control systems can be salvaged, adopted, enlarged, or used in various ways. Despite denial (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, Labor 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 µg/m³. Operations where employees' airborne lead levels were slightly in excess of 100 µg/m³ were treated the same as levels 10 or more times 100 µg/m³. (Tr. 3901.) It is clear, and IHE itself suggests, that work practices and housekeeping can reduce air lead levels about 20 percent (Tr. 3924). 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 $3.685 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 existing 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 ($55,000/100,000 cfm). As mentioned above, control systems are estimated at $6.00 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 of OSHA's 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 process—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 500-5000 batteries/day.

New, and substantially less expensive, engineering control techniques may also reduce costs, especially in small plants. As mentioned above, the APSEE system could prove effective and Kermitrol, Inc., has guaranteed its system will be effective to reduce levels to below 50 µg/m³. (Tr. 5217; 5208, 5220-21.)

IHE's cost estimates overestimate true costs because they focused completely on engineering controls. Dr. Firth 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... For 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 industry.

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 exposure (between 205.1 million and $250.0 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 design but do not include costs for retrofit, the Bergso process may be a more cost-effective long-run solution. Given the operating efficiencies Bergso claims for his existing smelters, the entire industry may eventually convert to his process. Bergso 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 to follow labor requirements were estimated by DBA to increase by three resulting in decreases in average productivity of 2.9 percent (Ex. 26, p. 3-33). It should be noted that the Bergso process is much more labor efficient than the current smelting and refining techniques. Bergso testified that only three production workers are required in the smelting and another three for the refinery per shift. (Ex. 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 is based on the assumption 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 unaffected in terms of production, capacity, and competition 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 product-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 oligopolistic in structure 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 70 plants, but the seven 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 48 plants and serve 90 percent of all the batteries sold. (Ex. 26, p. 5-42 (26.) 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 270 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. But the majority 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 (55 firms have less than 20 employees and a total of 2 percent of the workforce) to go out of business due to 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 firms of this size were already quite small.

These factors are expected to continue to put severe stress on the small battery manufacturer without respect to additional costs due to OSHA regulations. Given the operating efficiencies Bergso claims for his existing smelters, the entire industry may eventually convert to his process 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 Bergso process may be a more cost-effective long-run solution.

The seven largest companies operate a total of 70 plants, but their capacity is relatively low (Ex. 127, p. 3-42.) 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 market since they deal in small local markets where they supply retailers directly and, 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, who fixed prices for many years, 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 μg/m³, although further refinement, additions, and modifications may also be necessary. The cost of requiring engineering controls and work practices to be used to reach 100 μg/m³ 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 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 inordinate 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 manufacturers 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 estimate 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 estimate 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’s 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. 6-80 to 6-82.)

DBA assumes 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. 6-82.)

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. 6-84.) 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 trend 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 μg/m³ through relatively simple engineering controls. This conclusion was not disputed, and OSHA has determined that 1 year should be sufficient to implement control devices. A more extensive and refined use of the 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 depressions. 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. Cost 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 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 $31.3 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 μg/m³ level is $17.6-$21.1 million in capital costs and $2.9-$5.6 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 to buyers in lead pigments and that the degree of competition in the industry will decline slightly as marginal 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 μg/m³ 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 µg/m³ milestone in 3 years.

g. Can Manufacturing. The DBA report estimates costs for the can manufacturing industry as a whole as $187.5 million for the approximately 1,000,000 excess workers (Ex. 65B, p. 20). 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, 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 $200,000 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, will be able to keep approximately $20,000,000 or $183/worker per year. (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 5-7% 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 µg/m³ as an 8-hour TWA throughout the industry (Ex. 22, p. 194). According to the Printing Industry of Illinois, air levels in saving operations would never even approach 50 µg/m³ (Ex. 3(255)) and would probably be in the range of 0.2 to 1.4 µg/m³ (Ex. 3(601)). 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 $29,000,000 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 costs for 1,000,000 excess employees would be between $9 million and $25.68 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 chromatographic 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 report 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 the 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 $5.59 per employee and annual expenses totaled $22,256 million or $1,000.12 per employee. Additional labor requirements were estimated to be 1,369.5 man-years, and energy consumption 606,000 kWh.

The impact of these costs is 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-deferred subsity 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 the status quo is definitely threatened at the present level is now 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 therefore be reflected in the acquisition cost of such contracts. (Ex. 3(65B))

1. 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 50 percent of the employees between 50 and 100 µg/m³ and 60 percent over 100 µg/m³, while the low category had 70 percent below 50 µg/m³, 20 percent between 50 and 100 µg/m³, and 10 percent greater than 100 µg/m³). These categories were then assigned 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. This method was 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, five in the medium category.
two in the low category, and two in

very low category. When the aver-
ge 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, it is necessary to make a
reasonable estimate of the potential costs
to the 97 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 µg/m³ PEL, are generally
appropriate for achieving either a
100 or 50 µg/m³ PEL. Capital costs are
for engineering controls, and in these
industries exposure levels are generally
low, necessitating simple and inexpen-
sive enclosure or ventilation sys-
tems. Fugitive emissions are expected to
be negligible once control devices are
installed, so controls which are ef-

te to achieve 100 µg/m³ should result
in lower exposure levels than those
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 esti-

mates have been developed in this way
employ 630,335 potentially exposed
workers. Capital costs for compliance
total $108.4 million, and annual costs
total $28.4 million. On a per employee
basis, this is equivalent to $188 and
$134, respectively. None of these in-
dustries presented evidence or made
claims that economic hardship would
result from the proposed standard.

In addition, there are many other in-
dustries in which lead exposure may
occur; e.g., pipe galvanizing, brick and
tile manufacturing, tanning, and book-
binding. 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 estima-
ted the aggregate price and employ-
ment effects of the proposed standard.
As would be determined from the data
of the national economy, the anticipated
costs were minimal. Nationally, unem-
ployment would be expected to decline
by .08 percent (a net gain of 5,200
jobs) (Ex. 23, p. 6-51) 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 µg/m³
proposal. (Ex. 26, p. 6-65.) The OSHA
revisions would likely reduce these impacts
even further.

Since the cost of achieving 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 µg/m³ standard. It is clear that
evaporative cost estimates in the DBA
study were the possibility of labor
shortages in some of the more va-
cialized health fields. OSHA believes
that such shortages would be tempo-

rarily in nature and concludes that no
permanent disruption of labor mar-

kets will result from the standard.

FOOTNOTES

(1) In SPI v. OSHA, the court quoted with

approval the language from OSHA's pream-
ble that "it is not possible to predict the
degree of improvement to be obtained from
engineering changes until such changes are
actually implemented."

(2) In Atlantic and Gulf Stevedores, Inc v.

OSHRC, the Third Circuit likewise recog-
nized that the feasibility or infeasibility of a
standard may not become apparent until
such changes are actually implemented.

(3) OPL-OIO v. Bobst, 330 F.2d at 122.
The language cited referred to the Secre-
tary's determination on the issue of feasibil-
ity, which the court upheld, and said that
even if the price effects approximation
incorporates understatements of the
real cost of meeting the PEL, there
would be no discernable inflationary
impact on the U.S. economy attribu-
table to the standard.

(4) In Atlantic and Gulf Stevedores, Inc v.

OSHRC, the Third Circuit likewise recog-
nized that the feasibility or infeasibility of a
standard may not become apparent until
such changes are actually implemented.

(5) 509 F.2d at 1309.

(6) 509 F.2d at 1309.

(7) 509 F.2d at 1309.

(8) 509 F.2d at 1309.

(9) 509 F.2d at 1309.

(10) 509 F.2d at 1309.

(11) 509 F.2d at 1309.
Remy is a division of General Motors, and Rinker is used. It is recognized that the short-run costs are the "real" costs to a firm, although they are not the basis for making decisions of whether to stay in business (Tr. 3392-93).

(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. 3392-93).

(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 viability, the aggregate of which may present full cost recovery, the industry would be able to invest in new facilities.

(18) Certain financial information (cost of capital, tax structure) is necessary to make valid predictions of any company's economic viability, the aggregate of which may present full cost recovery, the industry would be able to invest in new facilities.

(19) 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. 30).

(20) OSHA Arsenic Record.

(21) Bureau of Labor Statistics. (See Ex. 127, Table 2-17, n. 1.)

(22) This was figured in the same manner as CRA. (See Ex. 127, Table 2-17, n. 1.)

(23) There was no way to reconcile the various 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. 28, Table 5-3-14.) DBA's total for seven plants is $6,599 million; CRA's total is 1.786.

(24) 723,879 short tons (Ex. 127, Table 2-11).

(25) 3,055 exposed employees (Ex. 28, Table 5.1).

(26) 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).

(27) Certain financial information (cost of capital, tax structure) is necessary to make valid predictions of any company's economic viability, the aggregate of which may present full cost recovery, the industry would be able to invest in new facilities.

(28) There is apparently an error in multiplication in the DBA report. The estimate should total $498,000.

(29) There is little exposure data in this industry, but two companies reported exposures among lead-exposed employees to be between .002 µg/m³ (Ex. 658, p. 19) and .4 µg/m³.

(30) Capital costs are exceptionally low in this industry because engineering controls to control exposure consist primarily of portable local exhaust ventilation units costing about $500.

(31) The Short report's per employee costs are as follows (Ex. 22, pp. 123-125):

<table>
<thead>
<tr>
<th>Category</th>
<th>Capital costs</th>
<th>Annual costs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very high</td>
<td>$10,600/17,875</td>
<td>$2,910/4,785</td>
</tr>
<tr>
<td>High</td>
<td>7,100/5,250</td>
<td>2,650/2,755</td>
</tr>
<tr>
<td>Medium</td>
<td>3,500/2,625</td>
<td>1,550/1,375</td>
</tr>
<tr>
<td>Low</td>
<td>2,500/1,975</td>
<td>850/725</td>
</tr>
<tr>
<td>Very low</td>
<td>250/200</td>
<td>280/280</td>
</tr>
</tbody>
</table>

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. 19.)

(32) DBA's average cost figures for each category are as follows. Operation size was not given.

<table>
<thead>
<tr>
<th>Category</th>
<th>Capital Annual costs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Very high</td>
<td>$17,042/3,116</td>
</tr>
<tr>
<td>High</td>
<td>3,763/1,247</td>
</tr>
<tr>
<td>Low</td>
<td>123/69</td>
</tr>
<tr>
<td>Very low</td>
<td>99/33</td>
</tr>
</tbody>
</table>

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 lead 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 6th day of November, 1978.

Eula Bingham,
Assistant Secretary of Labor.

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