



Regulations (Standards - 29 CFR) - Table of Contents

- **Part Number:** 1910
- **Part Title:** Occupational Safety and Health Standards
- **Subpart:** Z
- **Subpart Title:** Toxic and Hazardous Substances
- **Standard Number:** 1910.1025 App C
- **Title:** Medical surveillance guidelines

INTRODUCTION

The primary purpose of the Occupational Safety and Health Act of 1970 is to assure, so far as possible, safe and healthful working conditions for every working man and woman. The occupational health standard for inorganic lead⁽¹⁾ was promulgated to protect workers exposed to inorganic lead including metallic lead, all inorganic lead compounds and organic lead soaps.

Footnote⁽¹⁾ The term inorganic lead used throughout the medical surveillance appendices is meant to be synonymous with the definition of lead set forth in the standard.

Under this final standard in effect as of March 1, 1979, occupational exposure to inorganic lead is to be limited to 50 ug/m³ (micrograms per cubic meter) based on an 8 hour time-weighted average (TWA). This level of exposure eventually must be achieved through a combination of engineering, work practice and other administrative controls. Periods of time ranging from 1 to 10 years are provided for different industries to implement these controls. The schedule which is based on individual industry considerations is given in Table 1. Until these controls are in place, respirators must be used to meet the 50 ug/m³ exposure limit.

The standard also provides for a program of biological monitoring and medical surveillance for all employees exposed to levels of inorganic lead above the action level of 30 ug/m³ (TWA) for more than 30 days per year.

The purpose of this document is to outline the medical surveillance provisions of the standard for inorganic lead, and to provide further information to the physician regarding the examination and evaluation of workers exposed to inorganic lead.

Section 1 provides a detailed description of the monitoring procedure including the required frequency of blood testing for exposed workers, provisions for medical removal protection (MRP), the recommended right of the employee to a second medical opinion, and notification and recordkeeping requirements of the employer. A discussion of the requirements for respirator use and respirator monitoring and OSHA's position on prophylactic chelation therapy are also included in this section.

Section 2 discusses the toxic effects and clinical manifestations of lead poisoning and effects of lead intoxication on enzymatic pathways in heme synthesis. The adverse effects on both male and female reproductive capacity and on the fetus are also discussed.

Section 3 outlines the recommended medical evaluation of the worker exposed to inorganic lead including details of the medical history, physical examination, and recommended laboratory tests, which are based on the toxic effects of lead as discussed in Section 2.

Section 4 provides detailed information concerning the laboratory tests available for the monitoring of exposed workers. Included also is a discussion of the relative value of each test and the limitations and precautions which are necessary in the interpretation of the laboratory results.

TABLE 1

	Effective date					
	Mar 1, 1979	Mar 1, 1980	Mar 1, 1981	Mar 1, 1982	Mar 1, 1984	Mar 1, 1989 (final)
Permissible airborne lead levels by industry (ug/m(3) (1))						
1. Primary lead protection..	200	200	200	100	100	50
2. Secondary lead protection.....	200	200	200	100	50	50
3. Lead-acid battery manufacturing.....	200	200	100	100	50	50
4. Nonferrous foundries.....	200	100	100	100	50	50
5. Lead pigment manufacturing.....	200	200	200	100	50	50
6. All other industries.....	200	50	50	50	50	50

Footnote(1) Airborne levels to be achieved without reliance or respirator protection through a combination of engineering, work practice and other administrative controls. While these controls are being implemented respirators must be used to meet the 50 ug/m(3) exposure limit.

I. MEDICAL SURVEILLANCE AND MONITORING REQUIREMENTS FOR WORKERS EXPOSED TO INORGANIC LEAD

Under the occupational health standard for inorganic lead, a program of biological monitoring and medical surveillance is to be made available to all employees exposed to lead above the action level of 30 ug/m(3) TWA for more than 30 days each year. This program consists of periodic blood sampling and medical evaluation to be performed on a schedule which is defined by previous laboratory results, worker complaints or concerns, and the clinical assessment of the examining physician.

Under this program, the blood lead level of all employees who are exposed to lead above the action level of 30 ug/m(3) is to be determined at least every six months. The frequency is increased to every two months for employees whose last blood lead level was between 40 ug/100 g whole blood and the level requiring employee medical removal to be discussed below. For employees who are removed from exposure to lead due to an elevated blood lead, a new blood lead level must be measured monthly. A zinc protoporphyrin (ZPP) is required on each occasion that a blood lead level measurement is made.

An annual medical examination and consultation performed under the guidelines discussed in Section 3 is to be made available to each employee for whom a blood test conducted at any time during the preceding 12 months indicated a blood lead level at or above 40 ug/100 g. Also, an examination is to be given to all employees prior to their assignment to an area in which airborne lead concentrations reach or exceed the action level. In addition, a medical examination must be provided as soon as possible after notification by an employee that the employee has developed signs or symptoms commonly associated with lead intoxication, that the employee desires medical advice regarding lead exposure and the ability to procreate a healthy child, or that the employee has demonstrated difficulty in breathing during a respirator fitting test or during respirator use. An examination is also to be made available to each employee removed from exposure to lead due to a risk of sustaining material impairment to health, or otherwise limited or specially protected pursuant to medical recommendations.

Results of biological monitoring or the recommendations of an examining physician may necessitate removal of an employee from further lead exposure pursuant to the standard's medical removal protection (MRP) program. The object of the MRP program is to provide temporary medical removal to workers either with substantially elevated blood lead levels or otherwise at risk of sustaining material health impairment from continued substantial exposure to lead. The following guidelines which are summarized in Table 2 were created under the standard for the temporary removal of an exposed employee and his or her subsequent return to work in an exposure area.

TABLE 2

	Effective date			

	Mar. 1, 1979	Mar. 1, 1980	Mar. 1, 1981	Mar. 1, 1982	Mar. 1, 1983 (final)
A. Blood lead level requiring employee medical removal. (Level must be confirmed with second follow-up blood lead level within two weeks of first report.	>than or = to 80 ug/100 g	>than or = to 70 ug/100 g	>than or = to 60 ug/100 g	>than or = to 60 ug/100 g	>than or = to 60 ug/100 g or average of last three blood samples or all blood samples over previous 6 months (whichever is over a longer time period) is 50 ug/100 g or greater unless last blood sample is 40 ug/100 g or less.
B. Frequency which employees exposed to action level of lead (30 ug/m(3) TWA) must have blood lead level checked (ZPP is also required in each occasion that a blood lead is obtained.):					
1. Last blood lead level less than 40 ug/100 g	Every 6 months	Every 6 months	Every 6 months	Every 6 months	Every 6 months.
2. Last blood lead level between 40 ug/100 g and level requiring	Every 2 months	Every 2 months	Every 2 months	Every 2 months	Every 2 months.

medical					
removal					
(see A					
above)....					
3. Employees	Every 1	Every 1	Every 1	Every 1	Every 1
removed	months	months	months	months	months.
from					
exposure					
to lead					
because					
of an					
elevated					
blood lead					
level.....					
C. Permissible	100	50	30	30	30
airborne	ug/m(3)	ug/m(3)	ug/m(3)	ug/m(3)	ug/m(3)
exposure	8 hr TWA.	8 hr TWA.	8 hr TWA.	8 hr TWA.	8 hr TWA.
limit for					
workers					
removed from					
work due to					
an elevated					
blood lead					
level					
(without					
regard to					
respirator					
protection).					
D. Blood lead	.60	.50	.40	.40	.40
level	ug/100 g	ug/100 g	ug/100 g	ug/100 g	ug/100 g
confirmed					
with a second					
blood					
analysis, at					
which					
employee may					
return to					
work.					
Permissible					
exposure					
without					
regard to					
respirator					
protection is					
listed by					
industry in					
Table 1.					

Under the standard's ultimate worker removal criteria, a worker is to be removed from any work having any eight hour TWA exposure to lead of 30 ug/m(3) or more whenever either of the following circumstances apply: (1) a blood lead level of 60 ug/100 g or greater is obtained and confirmed by a second follow-up blood lead level performed within two weeks after the employer receives the results of the first blood sampling test, or (2) the average of the previous three blood lead determinations or the average of all blood lead determinations conducted during the previous six months, whichever encompasses the longest time period, equals or exceeds 50 ug/100 g, unless the last blood sample indicates a blood lead level at or below 40 ug/100 g in which case the employee need not be removed. Medical removal is to continue until two consecutive blood lead levels are 40 ug/100 g or less.

During the first two years that the ultimate removal criteria are being phased in, the return criteria have been set to assure that a worker's blood lead level has substantially declined during the period of removal. From March 1, 1979 to March 1, 1980, the blood lead level requiring employee medical removal is 80 ug/100 g. Workers found to have a confirmed blood lead at this level or greater need only be removed from work having a daily 8 hour TWA exposure to lead at or above 100 ug/m(3). Workers so removed are to be returned to work when their blood lead levels are at or below 60 ug/100 g of whole blood. From March 1, 1980 to March 1, 1981, the blood lead level requiring medical removal is 70 ug/100 g. During this period workers need only be removed from jobs having a daily 8 hour TWA exposure to lead at or above 50 ug/m(3) and are to be returned to work when a level of 50 ug/100 g is achieved. Beginning March 1, 1981, return depends on a worker's blood lead level declining to 40 ug/100 g of whole blood.

As part of the standard, the employer is required to notify in writing each employee whose blood lead level exceeds 40 ug/100 g. In addition each such employee is to be informed that the standard requires medical removal with MRP benefits, discussed below, when an employee's blood lead level exceeds the above defined limits.

In addition to the above blood lead level criteria, temporary worker removal may also take place as a result of medical determinations and recommendations. Written medical opinions must be prepared after each examination pursuant to the standard. If the examining physician includes a medical finding, determination or opinion that the employee has a medical condition which places the employee at increased risk of material health impairment from exposure to lead, then the employee must be removed from exposure to lead at or above the action level. Alternatively, if the examining physician recommends special protective measures for an employee (e.g., use of a powered air purifying respirator) or recommends limitations on an employee's exposure to lead, then the employer must implement these recommendations. Recommendations may be more stringent than the specific provisions of the standard. The examining physician, therefore, is given broad flexibility to tailor special protective procedures to the needs of individual employees. This flexibility extends to the evaluation and management of pregnant workers and male and female workers who are planning to raise children. Based on the history, physical examination, and laboratory studies, the physician might recommend special protective measures or medical removal for an employee who is pregnant or who is planning to conceive a child when, in the physician's judgment, continued exposure to lead at the current job would pose a significant risk. The return of the employee to his or her former job status, or the removal of special protections or limitations, depends upon the examining physician determining that the employee is no longer at increased risk of material impairment or that special measures are no longer needed.

During the period of any form of special protection or removal, the employer must maintain the worker's earnings, seniority, and other employment rights and benefits (as though the worker had not been removed) for a period of up to 18 months. This economic protection will maximize meaningful worker participation in the medical surveillance program, and is appropriate as part of the employer's overall obligation to provide a safe and healthful workplace. The provisions of MRP benefits during the employee's removal period may, however, be conditioned upon participation in medical surveillance.

On rare occasions, an employee's blood lead level may not acceptably decline within 18 months of removal. This situation will arise only in unusual circumstances, thus the standard relies on an individual medical examination to determine how to protect such an employee. This medical determination is to be based on both laboratory values, including lead levels, zinc protoporphyrin levels, blood counts, and other tests felt to be warranted, as well as the physician's judgment that any symptoms or findings on physical examination are a result of lead toxicity. The medical determination may be that the employee is incapable of ever safely returning to his or her former job status. The medical determination may provide additional removal time past 18 months for some employees or specify special protective measures to be implemented.

The lead standard provides for a multiple physician review in cases where the employee wishes a second opinion concerning potential lead poisoning or toxicity. If an employee wishes a second opinion, he or she can make an appointment with a physician of his or her choice. This second physician will review the findings, recommendations or determinations of the first physician and conduct any examinations, consultations or tests deemed necessary in an attempt to make a final medical determination. If the first and second physicians do not agree in their assessment they must try to resolve their differences. If they cannot reach an agreement then they must designate a third physician to resolve the dispute.

The employer must provide examining and consulting physicians with the following specific information: a copy of the lead regulations and all appendices, a description of the employee's duties as related to exposure, the exposure level to lead and any other toxic substances (if applicable), a description of personal protective equipment used, blood lead levels, and all prior written medical opinions regarding the employee in the employer's possession or control. The employer must also obtain from the physician and provide the employee with a written medical opinion containing blood lead levels, the physician's opinion as to whether the employee is at risk of material impairment to health, any recommended protective measures for the employee if further exposure is permitted, as well as any recommended limitations upon an employee's use of respirators.

Employers must instruct each physician not to reveal to the employer in writing or in any other way his or her findings, laboratory results, or diagnoses which are felt to be unrelated to occupational lead exposure. They must also instruct each physician to advise the employee of any occupationally or non-occupationally related medical condition requiring further treatment or evaluation.

The standard provides for the use of respirators where engineering and other primary controls have not been fully implemented. However, the use of respirator protection shall not be used in lieu of temporary medical removal due to elevated blood lead levels or findings that an employee is at risk of material health impairment. This is based on the numerous inadequacies of respirators including skin rash where the facepiece makes contact with the skin, unacceptable stress to breathing in some workers with underlying cardiopulmonary impairment, difficulty in providing adequate fit, the tendency for respirators to create additional hazards by interfering with vision, hearing, and mobility, and the difficulties of assuring the maximum effectiveness of a complicated work practice program involving respirators. Respirators do, however, serve a useful function where engineering and work practice controls are inadequate by providing supplementary, interim, or short-term protection, provided they are properly selected for the environment in which the employee will be working, properly fitted to the employee, maintained and cleaned periodically, and worn by the employee when required.

In its final standard on occupational exposure to inorganic lead, OSHA has prohibited prophylactic chelation. Diagnostic and therapeutic chelation are permitted only under the supervision of a licensed physician with appropriate medical monitoring in an acceptable clinical setting. The decision to initiate chelation therapy must be made on an individual basis and take into account the severity of symptoms felt to be a result of lead toxicity along with blood lead levels, ZPP levels, and other laboratory tests as appropriate. EDTA and penicillamine which are the primary chelating agents used in the therapy of occupational lead poisoning

have significant potential side effects and their use must be justified on the basis of expected benefits to the worker. Unless frank and severe symptoms are present, therapeutic chelation is not recommended given the opportunity to remove a worker from exposure and allow the body to naturally excrete accumulated lead. As a diagnostic aid, the chelation mobilization test using CA-EDTA has limited applicability. According to some investigators, the test can differentiate between lead-induced and other nephropathies. The test may also provide an estimation of the mobile fraction of the total body lead burden.

Employers are required to assure that accurate records are maintained on exposure monitoring, medical surveillance, and medical removal for each employee. Exposure monitoring and medical surveillance records must be kept for 40 years or the duration of employment plus 20 years, whichever is longer, while medical removal records must be maintained for the duration of employment. All records required under the standard must be made available upon request to the Assistant Secretary of Labor for Occupational Safety and Health and the Director of the National Institute for Occupational Safety and Health. Employers must also make environmental and biological monitoring and medical removal records available to affected employees and to former employees or their authorized employee representatives. Employees or their specifically designated representatives have access to their entire medical surveillance records.

In addition, the standard requires that the employer inform all workers exposed to lead at or above the action level of the provisions of the standard and all its appendices, the purpose and description of medical surveillance and provisions for medical removal protection if temporary removal is required. An understanding of the potential health effects of lead exposure by all exposed employees along with full understanding of their rights under the lead standard is essential for an effective monitoring program.

II. ADVERSE HEALTH EFFECTS OF INORGANIC LEAD

Although the toxicity of lead has been known for 2,000 years, the knowledge of the complex relationship between lead exposure and human response is still being refined. Significant research into the toxic properties of lead continues throughout the world, and it should be anticipated that our understanding of thresholds of effects and margins of safety will be improved in future years. The provisions of the lead standard are founded on two prime medical judgments: first, the prevention of adverse health effects from exposure to lead throughout a working lifetime requires that worker blood lead levels be maintained at or below 40 g/100 g and second, the blood lead levels of workers, male or female, who intend to parent in the near future should be maintained below 30 ug/100 g to minimize adverse reproductive health effects to the parents and developing fetus. The adverse effects of lead on reproduction are being actively researched and OSHA encourages the physician to remain abreast of recent developments in the area to best advise pregnant workers or workers planning to conceive children.

The spectrum of health effects caused by lead exposure can be subdivided into five developmental stages: normal, physiological changes of uncertain significance, pathophysiological changes, overt symptoms (morbidity), and mortality. Within this process there are no sharp distinctions, but rather a continuum of effects. Boundaries between categories overlap due to the wide variation of individual responses and exposures in the working population. OSHA's development of the lead standard focused on pathophysiological changes as well as later stages of disease.

1. Heme Synthesis Inhibition. The earliest demonstrated effect of lead involves its ability to inhibit at least two enzymes of the heme synthesis pathway at very low blood levels. Inhibition of delta aminolevulinic acid dehydrase (ALA-D) which catalyzes the conversion of delta-aminolevulinic acid (ALA) to protoporphyrin is observed at a blood lead level below 20 ug/100 g whole blood. At a blood lead level of 40 ug/100 g, more than 20% of the population would have 70% inhibition of ALA-D. There is an exponential increase in ALA excretion at blood lead levels greater than 40 ug/100 g.

Another enzyme, ferrochelatase, is also inhibited at low blood lead levels. Inhibition of ferrochelatase leads to increased free erythrocyte protoporphyrin (FEP) in the blood which can then bind to zinc to yield zinc protoporphyrin. At a blood lead level of 50 ug/100 g or greater, nearly 100% of the population will have an increase in FEP. There is also an exponential relationship between blood lead levels greater than 40 ug/100 g and the associated ZPP level, which has led to the development of the ZPP screening test for lead exposure.

While the significance of these effects is subject to debate, it is OSHA's position that these enzyme disturbances are early stages of a disease process which may eventually result in the clinical symptoms of lead poisoning. Whether or not the effects do progress to the later stages of clinical disease, disruption of these enzyme processes over a working lifetime is considered to be a material impairment of health.

One of the eventual results of lead-induced inhibition of enzymes in the heme synthesis pathway is anemia which can be asymptomatic if mild but associated with a wide array of symptoms including dizziness, fatigue, and tachycardia when more severe. Studies have indicated that lead levels as low as 50 ug/100 g can be associated with a definite decreased hemoglobin, although most cases of lead-induced anemia, as well as shortened red-cell survival times, occur at lead levels exceeding 80 ug/100 g. Inhibited hemoglobin synthesis is more common in chronic cases whereas shortened erythrocyte life span is more common in acute cases.

In lead-induced anemias, there is usually a reticulocytosis along with the presence of basophilic stippling, and ringed sideroblasts, although none of the above are pathognomonic for lead-induced anemia.

2. Neurological Effects. Inorganic lead has been found to have toxic effects on both the central and peripheral nervous systems. The earliest stages of lead-induced central nervous system effects first manifest themselves in the form of behavioral disturbances and central nervous system symptoms including irritability, restlessness, insomnia and other sleep disturbances, fatigue, vertigo, headache, poor memory, tremor, depression, and apathy. With more severe exposure, symptoms can progress to drowsiness, stupor, hallucinations, delirium, convulsions and coma.

The most severe and acute form of lead poisoning which usually follows ingestion or inhalation of large amounts of lead is acute encephalopathy which may arise precipitously with the onset of intractable seizures, coma, cardiorespiratory arrest, and death within 48 hours.

While there is disagreement about what exposure levels are needed to produce the earliest symptoms, most experts agree that symptoms definitely can occur at blood lead levels of 60 ug/100 g whole blood and therefore recommend a 40 ug/100 g maximum. The central nervous system effects frequently are not reversible following discontinued exposure or chelation therapy and when improvement does occur, it is almost always only partial.

The peripheral neuropathy resulting from lead exposure characteristically involves only motor function with minimal sensory damage and has a marked predilection for the extensor muscles of the most active extremity. The peripheral neuropathy can occur with varying degrees of severity. The earliest and mildest form which can be detected in workers with blood lead levels as low as 50 ug/100 g is manifested by slowing of motor nerve conduction velocity often without clinical symptoms. With progression of the neuropathy there is development of painless extensor muscle weakness usually involving the extensor muscles of the fingers and hand in the most active upper extremity, followed in severe cases by wrist drop or, much less commonly, foot drop.

In addition to slowing of nerve conduction, electromyographical studies in patients with blood lead levels greater than 50 ug/100 g have demonstrated a decrease in the number of acting motor unit potentials, an increase in the duration of motor unit potentials, and spontaneous pathological activity including fibrillations and fasciculations. Whether these effects occur at levels of 40 ug/100 g is undetermined.

While the peripheral neuropathies can occasionally be reversed with therapy, again such recovery is not assured particularly in the more severe neuropathies and often improvement is only partial. The lack of reversibility is felt to be due in part to segmental demyelination.

3. Gastrointestinal. Lead may also affect the gastrointestinal system producing abdominal colic or diffuse abdominal pain, constipation, obstipation, diarrhea, anorexia, nausea and vomiting. Lead colic rarely develops at blood lead levels below 80 ug/100 g.

4. Renal. Renal toxicity represents one of the most serious health effects of lead poisoning. In the early stages of disease nuclear inclusion bodies can frequently be identified in proximal renal tubular cells. Renal function remains normal and the changes in this stage are probably reversible. With more advanced disease there is progressive interstitial fibrosis and impaired renal function. Eventually extensive interstitial fibrosis ensues with sclerotic glomeruli and dilated and atrophied proximal tubules; all represent end stage kidney disease. Azotemia can be progressive, eventually resulting in frank uremia necessitating dialysis. There is occasionally associated hypertension and hyperuricemia with or without gout.

Early kidney disease is difficult to detect. The urinalysis is normal in early lead nephropathy and the blood urea nitrogen and serum creatinine increase only when two-thirds of kidney function is lost. Measurement of creatinine clearance can often detect earlier disease as can other methods of measurement of glomerular filtration rate. An abnormal Ca-EDTA mobilization test has been used to differentiate between lead-induced and other nephropathies, but this procedure is not widely accepted. A form of Fanconi syndrome with aminoaciduria, glycosuria, and hyperphosphaturia indicating severe injury to the proximal renal tubules is occasionally seen in children.

5. Reproductive effects. Exposure to lead can have serious effects on reproductive function in both males and females. In male workers exposed to lead there can be a decrease in sexual drive, impotence, decreased ability to produce healthy sperm, and sterility. Malformed sperm (teratospermia), decreased number of sperm (hypospermia), and sperm with decreased motility (asthenospermia) can all occur. Teratospermia has been noted at mean blood lead levels of 53 ug/100 g and hypospermia and asthenospermia at 41 ug/100 g. Furthermore, there appears to be a dose-response relationship for teratospermia in lead exposed workers.

Women exposed to lead may experience menstrual disturbances including dysmenorrhea, menorrhagia and amenorrhea. Following exposure to lead, women have a higher frequency of sterility, premature births, spontaneous miscarriages, and stillbirths.

Germ cells can be affected by lead and cause genetic damage in the egg or sperm cells before conception and result in failure to implant, miscarriage, stillbirth, or birth defects.

Infants of mothers with lead poisoning have a higher mortality during the first year and suffer from lowered birth weights, slower growth, and nervous system disorders.

Lead can pass through the placental barrier and lead levels in the mother's blood are comparable to concentrations of lead in the umbilical cord at birth. Transplacental passage becomes detectable at 12-14 weeks of gestation and increases until birth.

There is little direct data on damage to the fetus from exposure to lead but it is generally assumed that the fetus and newborn would be at least as susceptible to neurological damage as young children. Blood lead levels of 50-60 ug/100 g in children can cause significant neurobehavioral impairments and there is evidence of hyperactivity at blood levels as low as 25 ug/100 g. Given the overall body of literature concerning the adverse health effects of lead in children, OSHA feels that the blood lead level in children should be maintained below 30 ug/100 g with a population mean of 15 ug/100 g. Blood lead levels in the fetus and newborn likewise should not exceed 30 ug/100 g.

Because of lead's ability to pass through the placental barrier and also because of the demonstrated adverse effects of lead on reproductive function in both the male and female as well as the risk of genetic damage of lead on both the ovum and sperm, OSHA recommends a 30 ug/100 g maximum permissible blood lead level in both males and females who wish to bear children.

6. Other toxic effects. Debate and research continue on the effects of lead on the human body. Hypertension has frequently been noted in occupationally exposed individuals although it is difficult to assess whether this is due to lead's adverse effects on the kidney or if some other mechanism is involved. Vascular and electrocardiographic changes have been detected but have not been well characterized. Lead is thought to impair thyroid function and interfere with the pituitary-adrenal axis, but again these effects have not been well defined.

III. MEDICAL EVALUATION

The most important principle in evaluating a worker for any occupational disease including lead poisoning is a high index of suspicion on the part of the examining physician. As discussed in Section 2, lead can affect numerous organ systems and produce a wide array of signs and symptoms, most of which are non-specific and subtle in nature at least in the early stages of disease. Unless serious concern for lead toxicity is present, many of the early clues to diagnosis may easily be overlooked.

The crucial initial step in the medical evaluation is recognizing that a worker's employment can result in exposure to lead. The worker will frequently be able to define exposures to lead and lead containing materials but often will not volunteer this information unless specifically asked. In other situations the worker may not know of any exposures to lead but the suspicion might be raised on the part of the physician because of the industry or occupation of the worker. Potential occupational exposure to lead and its compounds occur in at least 120 occupations, including lead smelting, the manufacture of lead storage batteries, the manufacture of lead pigments and products containing pigments, solder manufacture, shipbuilding and ship repair, auto manufacturing, construction, and painting.

Once the possibility for lead exposure is raised, the focus can then be directed toward eliciting information from the medical history, physical exam, and finally from laboratory data to evaluate the worker for potential lead toxicity.

A complete and detailed work history is important in the initial evaluation. A listing of all previous employment with information on work processes, exposure to fumes or dust, known exposures to lead or other toxic substances, respiratory protection used, and previous medical surveillance should all be included in the worker's record. Where exposure to lead is suspected, information concerning on-the-job personal hygiene, smoking or eating habits in work areas, laundry procedures, and use of any protective clothing or respiratory protection equipment should be noted. A complete work history is essential in the medical evaluation of a worker with suspected lead toxicity, especially when long term effects such as neurotoxicity and nephrotoxicity are considered.

The medical history is also of fundamental importance and should include a listing of all past and current medical conditions, current medications including proprietary drug intake, previous surgeries and hospitalizations, allergies, smoking history, alcohol consumption, and also non-occupational lead exposures such as hobbies (hunting, riflery). Also known childhood exposures should be elicited. Any previous history of hematological, neurological, gastrointestinal, renal, psychological, gynecological, genetic, or reproductive problems should be specifically noted.

A careful and complete review of systems must be performed to assess both recognized complaints and subtle or slowly acquired symptoms which the worker might not appreciate as being significant. The review of symptoms should include the following:

General-weight loss, fatigue, decreased appetite.

Head, Eyes, Ears, Nose, Throat (HEENT)-headaches, visual disturbances or decreased visual acuity, hearing deficits or tinnitus, pigmentation of the oral mucosa, or metallic taste in mouth.

Cardio-pulmonary-shortness of breath, cough, chest pains, palpitations, or orthopnea.

Gastrointestinal-nausea, vomiting, heartburn, abdominal pain, constipation or diarrhea.

Neurologic-irritability, insomnia, weakness (fatigue), dizziness, loss of memory, confusion, hallucinations, incoordination, ataxia, decreased strength in hands or feet, disturbances in gait, difficulty in climbing stairs, or seizures.

Hematologic-pallor, easy fatigability, abnormal blood loss, melena.

Reproductive (male and female and spouse where relevant)-history of infertility, impotence, loss of libido, abnormal menstrual periods, history of miscarriages, stillbirths, or children with birth defects.

Musculo-skeletal-muscle and joint pains.

The physical examination should emphasize the neurological, gastrointestinal, and cardiovascular systems. The worker's weight and blood pressure should be recorded and the oral mucosa checked for pigmentation characteristic of a possible Burtonian or lead line on the gingiva. It should be noted, however, that the lead line may not be present even in severe lead poisoning if good oral hygiene is practiced.

The presence of pallor on skin examination may indicate an anemia, which if severe might also be associated with a tachycardia. If an anemia is suspected, an active search for blood loss should be undertaken including potential blood loss through the gastrointestinal tract.

A complete neurological examination should include an adequate mental status evaluation including a search for behavioral and psychological disturbances, memory testing, evaluation for irritability, insomnia, hallucinations, and mental clouding. Gait and coordination should be examined along with close observation for tremor. A detailed evaluation of peripheral nerve function including careful sensory and motor function testing is warranted. Strength testing particularly of extensor muscle groups of all extremities is of fundamental importance.

Cranial nerve evaluation should also be included in the routine examination.

The abdominal examination should include auscultation for bowel sounds and abdominal bruits and palpation for organomegaly, masses, and diffuse abdominal tenderness.

Cardiovascular examination should evaluate possible early signs of congestive heart failure. Pulmonary status should be addressed particularly if respirator protection is contemplated.

As part of the medical evaluation, the lead standard requires the following laboratory studies:

1. Blood lead level
2. Hemoglobin and hematocrit determinations, red cell indices, and examination of the peripheral blood smear to evaluate red blood cell morphology
3. Blood urea nitrogen
4. Serum creatinine
5. Routine urinalysis with microscopic examination.
6. A zinc protoporphyrin level.

In addition to the above, the physician is authorized to order any further laboratory or other tests which he or she deems necessary in accordance with sound medical practice. The evaluation must also include pregnancy testing or laboratory evaluation of male fertility if requested by the employee.

Additional tests which are probably not warranted on a routine basis but may be appropriate when blood lead and ZPP levels are equivocal include delta aminolevulinic acid and coproporphyrin concentrations in the urine, and dark-field illumination for detection of basophilic stippling in red blood cells.

If an anemia is detected further studies including a careful examination of the peripheral smear, reticulocyte count, stool for occult blood, serum iron, total iron binding capacity, bilirubin, and, if appropriate, vitamin B12 and folate may be of value in attempting to identify the cause of the anemia.

If a peripheral neuropathy is suspected, nerve conduction studies are warranted both for diagnosis and as a basis to monitor any therapy.

If renal disease is questioned, a 24 hour urine collection for creatinine clearance, protein, and electrolytes may be indicated. Elevated uric acid levels may result from lead-induced renal disease and a serum uric acid level might be performed.

An electrocardiogram and chest x-ray may be obtained as deemed appropriate.

Sophisticated and highly specialized testing should not be done routinely and where indicated should be under the direction of a specialist.

IV. LABORATORY EVALUATION

The blood lead level at present remains the single most important test to monitor lead exposure and is the test used in the medical surveillance program under the lead standard to guide employee medical removal. The ZPP has several advantages over the blood lead level. Because of its relatively recent development and the lack of extensive data concerning its interpretation, the ZPP currently remains an ancillary test.

This section will discuss the blood lead level and ZPP in detail and will outline their relative advantages and disadvantages. Other blood tests currently available to evaluate lead exposure will also be reviewed.

The blood lead level is a good index of current or recent lead absorption when there is no anemia present and when the worker has not taken any chelating agents. However, blood lead levels along with urinary lead levels do not necessarily indicate the total body burden of lead and are not adequate measures of past exposure. One reason for this is that lead has a high affinity for bone and up to 90% of the body's total lead is deposited there. A very important component of the total lead body burden is lead in soft tissue (liver, kidney, and brain). This fraction of the lead body burden, the biologically active lead, is not entirely reflected by blood lead levels since it is a function of the dynamics of lead absorption, distribution, deposition in bone and excretion. Following discontinuation of exposure to lead, the excess body burden is only slowly mobilized from bone and other relatively stable body stores and excreted. Consequently, a high blood lead level may only represent recent heavy exposure to lead without a significant total body excess and likewise a low blood lead level does not exclude an elevated total body burden of lead.

Also due to its correlation with recent exposures, the blood lead level may vary considerably over short time intervals.

To minimize laboratory error and erroneous results due to contamination, blood specimens must be carefully collected after thorough cleaning of the skin with appropriate methods using lead-free blood containers and analyzed by a reliable laboratory. Under the standard, samples must be analyzed in laboratories which are approved by the Center for Disease Control (CDC) or which have received satisfactory grades in proficiency testing by the CDC in the previous year. Analysis is to be made using atomic absorption spectrophotometry, anodic stripping voltammetry or any method which meets the accuracy requirements set forth by the standard.

The determination of lead in urine is generally considered a less reliable monitoring technique than analysis of whole blood primarily due to individual variability in urinary excretion capacity as well as the technical difficulty of obtaining accurate 24 hour urine collections. In addition, workers with renal insufficiency, whether due to lead or some other cause, may have decreased lead clearance and consequently urine lead levels may underestimate the true lead burden. Therefore, urine lead levels should not be used as a routine test.

The zinc protoporphyrin test, unlike the blood lead determination, measures an adverse metabolic effect of lead and as such is a better indicator of lead toxicity than the level of blood lead itself. The level of ZPP reflects lead absorption over the preceding 3 to 4 months, and therefore is a better indicator of lead body burden. The ZPP requires more time than the blood lead to read significantly elevated levels; the return to normal after discontinuing lead exposure is also slower. Furthermore, the ZPP test is simpler, faster, and less expensive to perform and no contamination is possible. Many investigators believe it is the most reliable means of monitoring chronic lead absorption.

Zinc protoporphyrin results from the inhibition of the enzyme ferrochelatase which catalyzes the insertion of an iron molecule into the protoporphyrin molecule, which then becomes heme. If iron is not inserted into the molecule then zinc, having a greater affinity for protoporphyrin, takes the place of the iron, forming ZPP.

An elevation in the level of circulating ZPP may occur at blood lead levels as low as 20-30 ug/100 g in some workers. Once the blood lead level has reached 40 ug/100 g there is more marked rise in the ZPP value from its normal range of less than 100 ug/100 ml. Increases in blood lead levels beyond 40 ug/100 g are associated with exponential increases in ZPP.

Whereas blood lead levels fluctuate over short time spans, ZPP levels remain relatively stable. ZPP is measured directly in red blood cells and is present for the cell's entire 120 day life-span. Therefore, the ZPP level in blood reflects the average ZPP production over the previous 3-4 months and consequently the average lead exposure during that time interval.

It is recommended that a hematocrit be determined whenever a confirmed ZPP of 50 ug/100 ml whole blood is obtained to rule out a significant underlying anemia. If the ZPP is in excess of 100 ug/100 ml and not associated with abnormal elevations in blood lead levels, the laboratory should be checked to be sure that blood leads were determined using atomic absorption spectrophotometry anodic stripping voltammetry, or any method which meets the accuracy requirements set forth by the standard by a CDC approved laboratory which is experienced in lead level determinations. Repeat periodic blood lead studies should be obtained in all individuals with elevated ZPP levels to be certain that an associated elevated blood lead level has not been missed due to transient fluctuations in blood leads.

ZPP has a characteristic fluorescence spectrum with a peak at 594 nm which is detectable with a hematofluorimeter. The hematofluorimeter is accurate and portable and can provide on-site, instantaneous results for workers who can be frequently tested via a finger prick.

However, careful attention must be given to calibration and quality control procedures. Limited data on blood lead-ZPP correlations and the ZPP levels which are associated with the adverse health effects discussed in Section 2 are the major limitations of the test. Also it is difficult to correlate ZPP levels with environmental exposure and there is some variation of response with age and sex. Nevertheless, the ZPP promises to be an important diagnostic test for the early detection of lead toxicity and its value will increase as more data is collected regarding its relationship to other manifestations of lead poisoning.

Levels of delta-aminolevulinic acid (ALA) in the urine are also used as a measure of lead exposure. Increasing concentrations of ALA are believed to result from the inhibition of the enzyme delta-aminolevulinic acid dehydrase (ALA-D). Although the test is relatively easy to perform, inexpensive, and rapid, the disadvantages include variability in results, the necessity to collect a complete 24 hour urine sample which has a specific gravity greater than 1.010, and also the fact that ALA decomposes in the presence of light.

The pattern of porphyrin excretion in the urine can also be helpful in identifying lead intoxication. With lead poisoning, the urine concentrations of coproporphyrins I and II, porphobilinogen and uroporphyrin I rise. The most important increase, however, is that of coproporphyrin III; levels may exceed 5,000 ug/1 in the urine in lead poisoned individuals, but its correlation with blood lead levels and ZPP are not as good as those of ALA. Increases in urinary porphyrins are not diagnostic of lead toxicity and may be seen in porphyria, some liver diseases, and in patients with high reticulocyte counts.

Summary. The Occupational Safety and Health Administration's standard for inorganic lead places significant emphasis on the medical surveillance of all workers exposed to levels of inorganic lead above the action level of 30 ug/m(3) TWA. The physician has a fundamental role in this surveillance program, and in the operation of the medical removal protection program.

Even with adequate worker education on the adverse health effects of lead and appropriate training in work practices, personal hygiene and other control measures, the physician has a primary responsibility for evaluating potential lead toxicity in the worker. It is only through a careful and detailed medical and work history, a complete physical examination and appropriate laboratory testing that an accurate assessment can be made. Many of the adverse health effects of lead toxicity are either irreversible or only partially reversible and therefore early detection of disease is very important.

This document outlines the medical monitoring program as defined by the occupational safety and health standard for inorganic lead. It reviews the adverse health effects of lead poisoning and describes the important elements of the history and physical examinations as they relate to these adverse effects. Finally, the appropriate laboratory testing for evaluating lead exposure and toxicity is presented.

It is hoped that this review and discussion will give the physician a better understanding of the OSHA standard with the ultimate goal of protecting the health and well-being of the worker exposed to lead under his or her care.

[56 FR 24686, May 31, 1991; 60 FR 52856, Oct. 11, 1995]

[➡ Next Standard \(1910.1025 App D\)](#)

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