Inorganic Lead

Introduction

Lead, a very soft malleable blue-grey metal, has many industrial applications. It is naturally deposited underground and has been mined and spread throughout the environment by emissions of motor vehicle exhausts and airborne emissions from smelters. Lead is one of the most common contaminants of the environment. It accumulates in bone and other tissues with age.

The important routes of absorption of lead in man are the gastrointestinal tract and the lungs. Dermal absorption is relatively insignificant in most cases.

The effects of lead poisoning are cumulative and result in a large variety of health problems beginning with nonspecific symptoms such as fatigue, dizziness, cramps, and headaches and eventually leading to a variety of disorders that can end in paralysis, brain damage, and death.

Serious cases of lead poisoning are rare in industry today because of more efficient material handling methods and biological monitoring. Intestinal colic (colon spasms accompanied by pain throughout the abdomen) preceded by several days of constipation is the most common manifestation of lead poisoning (Hunter, 1975).

Stresses such as an accident, operation, pneumonia or other infection, physical exertion, or alcohol ingestion can cause accumulated body lead to be released into the body and produce symptoms of lead poisoning in workers whose metabolism of lead is in delicate balance (Key et al., eds., 1977, and Plunkett, 1976).

Among female workers exposed to excessive lead levels, an increased number of miscarriages and stillbirths, menstrual disorders, and sterility are recognized risks. Lead poisoning as well as moderate increased absorption of lead decreases the fertility of men.

Blood lead, free erythrocyte protoporphyrin (FEP), and zinc protoporphyrin (ZP or ZPP) have been shown to be indicators of

absorption while urinary coproporphyrin (CP) (nitrogen-containing organic compounds) and delta-aminolevulinic acid (ALA) are reliable indicators of effect (Benson, 1976).

Lead and its compounds have numerous chemical and common names:

Chemical Name	Common Names
lead	C.I. Pigment Metal 4, C.I. 77575, lead flake, plumbum, lead S2, S1, KS-4
lead antimonate	antimony yellow, Naples yellow
lead azide	
lead blue	galena, blue basic lead sulfate
lead m-borate	lead metaborate
lead borosilicate	
lead bromate	
lead bromide	
lead carbonate	cerussete, cerussite, white lead
lead carbonate, basic	BCWL, ceruse, hydrocerussite, leadflake, lead subcarbonate, white lead
lead chlorate	
lead chloride	cotunite
lead chloride, basic	basic lead chloride, mendipite
lead chloride fluoride	matlockite
lead chlorite	
lead, chocolate	

lead chromate	lead salt of chromic acid, chrome yellow, cologne yellow, crocoite, deep chrome, Leipzig yellow, lemon chrome, lemon yellow, midle chrome, pale chrome, Paris yellow, permanent yellow, primrose chrome, primrose yellow, yellow ultramarine
lead chromate, basic	American vermillion, Austrian cinnabar, basic lead chromate, Chinese red, chromate red, chrome orange, chrome red, C.I. Pigment red, derby red, lead chromate oxide, Persian red, red lead chromate, Victoria red
lead cyanate	
lead cyanide	C.I. 77610, C.I. Pigment Yellow 48
lead dicyanoguanidine	
lead diiodide	
lead dioxide	C.I. 77580, lead brown, lead oxide brown, lead peroxide, lead superoxide, Plattnerite, anhydrous plumbic acid
lead di-o-phosphate	
lead dithionate	
lead ferricyanide	
lead ferrite	
lead ferrocyanide	
lead fluoride	lead difluoride, plumbous fluoride, lead silicofluoride
lead glance	galena

lead hydroxide	basic lead hydroxide, hydrated lead oxide, lead hydrate
lead iodate	
lead iodide	
lead mercaptate	
lead molybdolate	wulfenite
lead monoiodide	
lead monoxide	C.I. 77577, C.I. Pigment yellow 46, lead monoxide, lead oxide yellow, lead protoxide, lead oxide, litharge, litharge yellow L-28, massicot, massicotite, plumbous oxide
lead mono-o-phosphate	
lead nitrate	lead salt of nitric acid
lead nitrite	basic lead nitrite, lead sub-nitrite
lead ocher	massicot
lead orange	orange mineral
lead oxalate	
lead oxychloride	Cassel yellow, laurionite, matlockite, Mendipite
lead perchlorate	lead perchlorate hexahydrate, lead salt of perchloric acid hexahydrate
lead p-periodate	
lead-m-phosphate	

lead-o-phosphate	lead orthophosphate, normal lead orthophosphate, lead phosphate, perlex paste 500, perlex paste 600A, plumbous phosphate, lead salt of phosphoric acid, pyromorphite, trilead phosphate
lead-o-phosphite	
lead phosphite, dibasic	
lead potassium thiocyanate	
lead pyrophosphate	
lead selenate	
lead selenide	Clausthalite
lead sesquioxide	lead trioxide, plumbous plumbate
lead silicate	Alamosite, lead-m-silicate, lead metasilicate
lead silicate, dibasic	white lead silicate
lead-sodium thiosulfate	lead-sodium hyposulfite, sodium-lead hyposulfite, sodium-lead thiosulfate
lead stannate	
lead sulfate	Auglisite, C.I. 77630, C.I. Pigment White 3, fash white, Freeman's white lead, lead bottoms, milk white, Mulhouse white, lead salt of sulfuric acid
lead sulfate, basic	sublimed white lead
lead sulfate, blue basic	blue lead, sublimed blue lead

lead sulfide	C.I. 77640, galena, galanite, natural lead sulfide, plumbous sulfide
lead sulfite	
lead telluride	altaite
lead tetrachloride	
lead tetrafluoride	plumbic fluoride
lead tetroxide	C.I. 77578, C.I. Pigment Red 105, gold satinobre, lead ortho-plumbate, lead oxide red, mineral orange, mineral red, minium, minium nonsetting RL-95, orange lead, Paris red, plumbo-plumbic oxide, red lead, sandix, Saturn red
lead thiocyanate	lead sulfocyanate
lead thiosulfate	lead hyposulfite
lead titanate	lead-m-titanate, lead salt of titanic acid
lead tungstate	lead wolframate, raspite, stolzite
lead vanadate	lead-m-vanadate, lead meta-vanadate, lead vanadinate
lead vitriol	anglesite
lead zirconate titanate	LZT

Occupations with Potential Exposures to Inorganic Lead

acid finishers
actors
artists, commercial
auto body shop workers
chemical equipment makers
chippers
chlorinated paraffin makers
cigar makers

babbitters
battery makers and workers
blacksmiths
bookbinders
bottle cap makers
brass founders
brass polishers
braziers
brick layers
brick makers
bronzers
brush makers
cable makers
cable splicers
canners
cartridge makers
ceramic makers
farmers
file cutters
firemen
flower makers, artificial
foundry workers
galvanizers
garage mechanics
glass makers
glass polishers
glost-kiln workers
gold refiners
gun barrel browners
imitation pearl makers
incandescent lamp makers
ink makers
insecticide makers
insecticide users
japan makers
japanners
jewelers
junk metal refiners
labelers, paint can
lacquer makers
lead burners
lead counterweight makers
lead flooring makers
lead foil makers
lead mill workers
crop dusters
cutlery makers
decorators, pottery
demolition workers
dental technicians
diamond polishers
dye makers
dyers
electronic device makers
electroplaters
electrotypers
embroidery workers
emery wheel makers
enamel burners
enamel makers
enamelers
explosives makers
mordanters
musical instrument makers
nitric acid workers
nitroglycerin makers
nuclear reactor workers
nuclear technologists
paint makers
paint pigment makers
painters
paper hangers
patent leather makers
pearl makers, imitation
pharmaceutical makers
photography workers
pipefitters
plastic workers
plumbers
policemen
pottery glaze dippers
pottery glaze mixers
pottery workers
printers
putty makers
pyroxylin-plastics workers
riveters
roofers
rubber buffers
rubber makers

lead miners
lead pipe makers
lead salt makers
lead shield makers
lead smelters
lead stearate makers
lead workers
leather workers
linoleum makers
linotypers
linseed oil boilers
lithographers
lithotransfer workers
lubricant makers
match makers
metal burners
metal cutters
metal grinders
metal miners
metal polishers
metal refiners
metallizers
mirror silverers
type founders
type setters
vanadium compound makers
varnish makers
rubber reclaimers
scrap metal workers
semiconductor workers
service station attendants
sheet metal workers
shellac makers
ship dismantlers
shoe stainers
shot makers
sign painters
silk weighters
slushers, porcelain enameling
solder makers
solderers
steel engravers
stereotypers
tannery workers
television picture tube makers
temperers
textile processors
tile makers
tin foil makers
tinners
vehicle tunnel attendants
wallpaper printers
welders

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

The following should be considered:
—Acute appendicitis,
—chronic gastric or duodenal (first part of the small intestines) ulcer,
—carcinoma of the stomach, and
—pernicious anemia (severe form of blood disease) or secondary anemia due to hemorrhoids, melena, or hematemesis.

Nonoccupational Exposure

Lead is so widely used that a careful inquiry into hobbies and recreation is especially important. Chronic exposure to inorganic lead in hobbies can produce the same signs and symptoms as occupational lead poisoning.

Common nonoccupational lead exposure sources include:

—Foods (bread, meat, canned foods and vegetables),
—hobbies (ceramics, pottery),
—consuming illicit liquors distilled using lead or lead-based tubing, soldered condensers (such as automobile radiators),
—use of lead-glazed earthenware,
—artists using lead paints,
—lead toys
—lead dust in shooting galleries, and
—fumes from burning batteries or painted furniture.

Signs and Symptoms

The early signs and symptoms of lead poisoning are nonspecific and may resemble many diseases including influenza. Early signs and symptoms are:

—Malaise, fatigue,
—sleep disturbance,
—constipation,
—abdominal cramps,
—anemia, hemolytic (red blood cells being destroyed) in type but not usually severe,
—irritability,
—aching muscles and bones,
—headache,
—decreased appetite, and
—nausea and vomiting.

These symptoms are reversible, and complete recovery is possible.

In more advanced cases of lead poisoning, the above signs and symptoms progress and frequently involve the gastrointestinal and neuromuscular systems (both nerves and muscles).

Central nervous system symptoms are:

—Brain dysfunction (encephalopathy) which may mimic bacterial meningitis. However, cerebrospinal fluid glucose level is normal. Symptoms include:
  -Fever,
  -headache,
  -stiff neck,
  -vomiting, and
  -personality changes.
—tremor,
—hallucinations,
—intellectual deterioration,
—rarely, accumulation of cerebrospinal fluid within the brain (hydrocephalus),
—blindness may occur from optical atrophy (wasting away of the optic nerve) secondry to lead exposure, and
—convulsions.

Gastrointestinal symptoms are:

—Colon spasms (colic),
—nausea, vomiting,
—loss of appetite, and
—constipation.

Signs and symptoms associated with the blood and blood-forming tissues are:

—Anemia, in which the red blood cells have a reduced hemoglobin content (hypochromic normocytic type) and
—increased serum iron.

The marrow also reveals increased production, and specific structural (morphological) changes in nucleated red corpuscles (erythroblasts) such as:

—Basophilic stippling and
—deformed nuclei.

The iron content of the marrow is increased, and increased siderocytes, sideroblasts and reticuloendthelial cells are noted. Some investigators believe the basic effect lead has on the bone marrow is first hyperstimulation, followed by delayed maturation.

Kidney (renal) symptoms are:

—An abnormal amount of uric acid in the blood (hyperuricemia,
—inflammation (nephritis),
—the presence of glucose in the urine (glycosuria),
—an abnormal amount of amino acids in the urine (hyperaminoaciduria), and
—progressive increase in blood urea.

Additional signs and symptoms which may be present are:

—Gum lead line (black or purplish line on gum margin),
—skin pallor (ashen gray),
—loss of weight, and
—weakness of extensor muscles (such as wrist or foot drop).

Cortical atrophy (reduction in size of brain tissue) has also been described but this is not a common finding.

Laboratory and Clinical Examinations

Signs pertaining to lead's effect on the blood-forming organs (hemopoietic system) are determined by laboratory analysis. These signs occur early with excess lead absorption — usually before the outward symptoms of poisoning appear. These tests are useful in the routine biological monitoring of persons exposed to lead.

NOTE: In studies of lead excretion, satisfactory figures cannot be obtained unless specimens of stools and urine are collected for at least 3 days. Normal persons excrete lead in feces and urine because lead is present in soil and therefore in vegetation and animal food sources.

Results of blood and urine laboratory analyses for lead are subject to a 10 to 15% error factor. The normal values for the laboratory performing the tests should be ascertained. Blood lead determinations must be corrected for the mass of circulating red cells (hematocrit), and urinary lead determinations, for the specific gravity of the urine.

Parameters which will be useful in the laboratory diagnosis of lead poisoning are presented along with abnormal laboratory values that may be found in lead poisoning:

Blood
—decreased hemoglobin — less than 13 gram %
—increased blood lead (PbB) — greater than 60 to 80 micrograms per deciliter
—decreased red blood count
—stippled basophilia and reticulocytosis
—increased free erythocyte protoporphyrin (FEP) and zinc protoporphyrin (ZP or ZPP)

Urine
—increased urinary lead — greater than 0.15 milligram per liter
—increased urinary lead after Ca-EDTA treatment — greater than 2 milligrams in 24 hours
—increased urinary coproporphyrin (CP) — greater than 80 micrograms per 100 milligrams creatinine
—increased urinary delta-aminolevulinic acid (ALA) — greater than 2.0 milligrams per 100 milligrams creatiine
—increased urinary porphobilinogen — greater than 0.15 milligram per 100 milligrams creatinine

Central Nervous System
—decreased peroneal nerve conduction velocity

Epidemiology

Extensive studies have been conducted around smelters, battery factories, soap reclaiming facilities, chemical plants producing lead salts, and gasoline refineries. Neuropathies, nephropathy, and blood changes are well documented. However, lead absorption does not necessarily indicate poisoning.

Feldman et al.[59] reported a study of subacute low level exposure to lead which occurred when a demolition company dismantled an elevated train network. The old steel structure was heavily coated with several coats of lead paint. Respirators were in use. Data from the report are presented in the following table.

LABORATORY AND CLINICAL DATA FROM 44 DEMOLITION WORKERS, BY EXPOSURE							
Group	Number of Workers	Average Lead Exposure (milligram per cubic meter of air)	Blood Lead (microgram per 100 grams)		Mean Hemato-crit (%)	Mean F.E.P.[a] (microgram per deciliter red blood cells)	Mean M.N.C.V.[b] (m/s)
			range	mean			
Burners	32	4.36	44-100	79.5	41.4	1134[c]	43.2[c]
Nonburners	12	0.23	24-75	48.8	44.0	714[d]	49.0[e]
Normal Value						46.9 ± 14.9	54.09 ± 5.96

(Feldman et al., 1977)

a. Free erythrocyte protoporphyrin
b. Peroneal motor-nerve conduction velocity
c. Measured in 13 burners
d. Measured in 5 nonburners
e. Measured in 6 nonburners

Nonburners included laborers and supervisory personnel who had been on the job for 4 to 10 months and offered no complaints. Burners with as little as 1 month on the job before symptoms and other signs of increased body burden of lead appeared reported experiencing nausea, abdominal discomfort, mood change and irritability, sleeplessness, fatigue, headache, and numbness and tingling of the extremities. Four of the burners had no complaints. Workers having abnormalities in 2 of 3 variables (blood lead, F.E.P., or M.N.C.V.) and complaining of some symptoms were considered to have intoxication and were referred for chelation therapy.

(Blood lead levels greater than 60 micrograms of lead per 100 grams whole blood are indicative of unacceptable lead absorption; urine lead levels of 0.20 milligram lead per liter of urine or greater are indicative of unacceptable lead absorption.[60])

Elkins[61] assembled data available on lead in air and lead in urine and reported that a urinary lead level of 0.2 milligrams lead per liter of urine would correspond to an air concentration of 0.2 milligrams lead per cubic meter of air.

The data in the table on the next page relating average and median blood lead content with exposure and duration of employment have been adapted from Dreesen et al.[62], the Committee on Biological Effects of Atmospheric Pollutants,[63] and the National Institute for Occupational Safety and Health[60].

Tola and Nordman[64] reported a study of 335 men representing the general population and 2,209 men occupationally exposed to lead. No association between blood lead concentrations and smoking was demonstrated in the men from the general population. A dose-response relationship was found between the amount of smoking and the blood lead concentrations of workers occupationally exposed to lead with smokers having statistically significant higher blood lead levels than nonsmokers.

Sakurai et al.[65] reported a study of 218 male workers in a rubber hose and automobile tire factory who had an overall mean duration of occupational lead exposure of 5.0 years (the range was 6 months to 21 years). Lead exposure had been so low that in the past the plant physician had diagnosed no cases of clinical lead poisoning; average 8-hour lead in air concen-

trations were below 60 micrograms per cubic meter. Sakurai et al. concluded that subjective symptoms are not likely to be induced by lead when the blood level is 50 micrograms per 100 grams and less.

AVERAGE AND MEDIAN BLOOD LEAD CONTENT IN MILLIGRAMS PER 100 GRAMS OF BLOOD IN STORAGE BATTERY WORKERS, BY EXPOSURE AND DURATION OF EMPLOYMENT

Duration of Lead Exposure	Air Lead Content (milligrams per cubic meter)			
	0-0.074	0.075-0.14	0.15-0.29	0.3 or greater
Years: 0-4				
Number	17	16	32	20
Average	0.0187	0.0316	0.0378	0.0463
Median	0.021	0.030	0.038	0.050
Years: 5-9				
Number	10	13	40	24
Average	0.0278	0.0405	0.0501	0.0505
Median	0.033	0.040	0.043	0.050
Years: 10-14				
Number	23	24	30	32
Average	0.0198	0.0375	0.0502	0.0481
Median	0.018	0.038	0.046	0.048
Years: 15+				
Number	44	30	59	45
Average	0.0293	0.0407	0.0457	0.0493
Median	0.023	0.036	0.045	0.045

(Dreesen et al., 1941, National Academy of Sciences, 1971, and NIOSH, 1972)

Lancranjan et al.[66] reported a study of the reproductive ability of 150 men occupationally exposed to lead in a storage battery plant. One hundred men (Group A) had an average exposure of 8.5 years (the range was 1 to 23 years) in the plant; 50 technicians and office workers (Group B) worked in annex workrooms in a lead-polluted environment for 1 to 27 (mean 6) years. Environmental measurements were not given. Workers displaying moderately increased absorption of lead or lead poisoning showed a highly significant fertility decrease. Laboratory values from the report are given in the following table.

MEAN VALUES OF LEAD IN BLOOD AND URINE OF COPROPORPHYRIN AND DELTA-AMINOLEVULINIC ACID

Group	Lead in Blood (microgram per 100 milliliter)	Lead in Urine (microgram per liter)	Coproporphyrine (microgram per liter)	delta-Amino-levulinic Acid (milligram per liter)
A. (a) Lead-poisoned workmen, 23	74.50 ± 26	385 ± 71	394 ± 116	56.52 ± 20
(b) Lead workmen with moderately increased absorption, 42	52.80 ± 21	251 ± 106	295 ± 132	22.44 ± 8.8
(c) Lead workmen with slightly increased absorption, 35	41 ± 12	100.6 ± 41	80 ± 44	7.7 ± 4.2
B. Men with physiologic absorption of lead working in a polluted environment, 23	23 ± 14	92 ± 34	35 ± 16	4.4 ± 2.2

(Lancranjan et al., 1975)

Cooper and Gaffey[67] reported a mortality study of 7,032 men who had been employed in 6 lead production facilities and 10 battery plants for 1 or more years over a 23 year period. Lead absorption in many of the men was greatly in excess of currently acceptable standards based upon urinary and blood lead concentrations available for a portion of the group. Although the workers had high levels of exposure, only small deviations from expected mortality were reported. Cooper and Gaffey predicted no detectable effect on the mortality of male adults from occupational exposure to lead controlled in conformity to currently recommended environmental and biological standards[60].

Evidence of Exposure

Air Sampling and Analysis

The NIOSH approved air sampling method uses mechanical filtration. Two methods previously used are:

1. Electrostatic precipitator and
2. impingement.

The NIOSH approved method of analysis is atomic absorption spectrophotometry. Four methods previously used are:

1. Polargraphic,
2. spectrographic,
3. dethizone procedure, and
4. titrimetric-extraction.

These methods are not intended to be exclusive but other methods should be justified.

Allowable Exposure Limits

The Federal standard for lead and its inorganic compounds (except lead arsenate) is 0.2 milligram per cubic meter of air based on an 8-hour time-weighted average exposure. Lead is a suspected occupational carcinogen with the lung and kidney being the target organs (Key et al., eds., 1977) (NOTE: A reduction in the standard to less than 50 micrograms of lead per cubic meter of air has been proposed by NIOSH.)

Conclusion

Diagnostic criteria for occupational lead poisoning are based on meeting the following:

1. Confirmed history of occupational exposure to lead,
2. findings compatible with lead poisoning, and
3. increased lead in blood and/or urine.

NOTE: A diagnosis of lead poisoning does not necessarily mean that it is occupational in origin. Further, lead intoxication with symptoms can exist with normal laboratory test findings.

The medical literature has extensive references to the treatment of lead toxicity. Basically, there are 3 types of drugs currently known to be effective in treating lead toxicity: calcium-ethylenediaminetetra-acetic acid (Ca-EDTA), British anti-lewisite (BAL), and Penicillamine (PCA). Treatment usually depends upon the severity of symptoms and available laboratory data. Because the chemicals used in the treatment of lead poisoning are not without their own toxicities, their use in exposed workers should be followed closely with repeat blood levels and urinalysis.

Inorganic Mercury

Introduction

Mercury, a chemically stable element which is liquid at room temperature, is found everywhere—in rocks, soils, plants, animals, water, air. It is found in food in the range of 0.005 milligram to 0.02 milligram daily and is not considered toxic at this level. However, it is becoming increasingly significant as a potential hazard in the environment. For industrial and commercial uses, it is removed from the ore, cinnabar, in reduction plants.

Exposure to mercury can be through percutaneous (skin) absorption, ingestion, or inhalation with the principal source of poisoning being mercury vapor. Nonoccupational exposure to mercury has resulted in urinary excretions of 0.5 milligram per day in urine and 10 milligrams in feces.

Mercurialism may be the result of chronic excessive exposure to inorganic mercury, and is characterized by 1 or more of the 4 classical signs of poisoning: Gingivitis (inflammation of the gums), sialorrhea (excessive flow of saliva), tremors (affecting fingers, eyelids, lips or tongue), and erethism (emotional instability). Mercury vapor has been reported to cause fibrotic lesions in the lung. Many of the symptoms associated with exposure are very general and have no connection whatever with such exposure: tiredness and drowsiness at work, with insomnia, a feeling of weakness, and exhaustion. Because of the many nonspecific signs and symptoms which can be associated with mercury, occupational exposure levels at which no effects are observed have not been established.

Acute intoxication from inhaling mercury vapor in high concentrations was common in the past among those who extracted mercury from its ores; now, it is relatively infrequent. Acute severe exposures are characterized by metallic taste, nausea, abdominal pain, vomiting, diarrhea, headache, and sometimes albuminurea (usually a sign of renal impairment).

The latency period for signs of toxicity to be produced can vary from 1 to 30 years.

Mercury and its compounds have numerous chemical and common names:

Chemical Name	Common Names
mercuric ammonium chloride	mercury amide chloride, mercury ammonium chloride
mercuric arsenate	mercury arsenate, mercury-o-arsenate
mercuric-barium bromide	barium-mercury bromide, mercury-barium bromide
mercuric-barium iodide	barium mercury iodide, mercury-barium iodide
mercuric bromate	
mercuric bromide	mercury bromide
mercuric bromide ammobasic	
mercuric bromide diammine	
mercuric bromide iodide	
mercuric chlorate	
mercuric chloride	bichloride of mercury, calochlor, corrosive mercury chloride, corrosive sublimate, MC, mercuric bichloride, mercury bichloride, mercury perchloride, perchloride of mercury
mercuric chloride diammine	
mercuric chloride iodide	
mercuric chloroiodide	
mercuric chromate	

mercuric-cuprous iodide	copper-mercury iodide, mercury-copper iodide
mercuric cyanate	fulminate of mercury, mercuric fulminate, mercury fulminate
mercuric cyanide	mercury cyanide
mercuric dichromate	mercuric dichromate, mercury bichromate
mercuric fluoride	mercury fluoride
mercuric fluorsilicate	
mercuric iodate	mercury iodate
mercuric iodide	mercuric biniodide, mercury biniodide, yellow mercury iodide, red mercuric iodide, red mercury iodide
mercuric iodide ammonobasic	
mercuric iodide, aquobasic-ammonobasic	
mercuric iodide diammine	
mercuric nitrate	mercury nitrate, mercury pernitrate, mercury salt of nitric acid
mercuric oxalate	
mercuric oxide, red	mercuric oxide, red mercury oxide, red oxide of mercury, red precipitate
mercuric oxide, yellow	yellow mercury oxide, yellow mercuric oxide, yellow oxide of mercury, yellow precipitate
mercuric oxybromide	
mercuric oxychloride	

mercuric oxycyanide	mercury cyanide oxide, mercury oxycyanide
mercuric oxyfluoride	
mercuric oxyiodide	
mercuric perchlorate	
mercuric phosphate	mercuric-o-phosphate, mercury phosphate, neutral mercuric phosphate, normal mercuric phosphate, tertiary mercuric phosphate, trimercuric orthophosphate
mercuric-potassium cyanide	mercury-potassium cyanide
mercuric-potassium iodide	Channing's solution, Mayer's reagent, mercury-potassium iodide, Nessler's reagent, potassium mercuric iodide, potassium tetraiodomercurate, potassium triiodomercurate, solution potassium iodohydragyrate
mercuric potassium thiosulfate	
mercuric salicylate	mercury subsalicylate, salicylated mercury
mercuric selenide	
mercuric sesquiiodide	
mercuric silver iodide	mercury-silver iodide, silver-mercury iodide
mercuric subsulfate	basic mercuric sulfate, mercuric dioxysulfate, turbith mineral, turpeth mineral
mercuric sulfate	mercury bisulfate, mercury persulfate, mercury sulfate, mercury salt of sulfuric acid

mercuric sulfide, black	black mercury sulfide, ethiops mineral
mercuric sulfide, red	artificial cinnabar, chinese vermilion, cinnabar, quicksilver vermilion, red mercury sulfide, red mercury sulfurel, vermilion
mercuric sulfocyanate	mercuric sulfocyanide, mercury rhodanide, mercury sulfocyanate, mercuric thiocyanate, mercury thiocyanate
mercuric tellurate	
mercuric thallium iodide	
mercuric tungstate	
mercuric cyanamid	
mercurous arsenite	mercury arsenite
mercurous azide	
mercurous bromate	
mercurous bromide	mercury bromide
mercurous carbonate	
mercurous chlorate	mercury chlorate
mercurous chloride	calogreen, calomel, calosan, cyclosan, mercury monochloride, mercury protochloride, mild mercury chloride, precitite blanc, subchloride of mercury
mercurous chromate	mercury chromate
mercurous fluoride	
mercurous fluosilicate	

mercurous iodate	
mercurous iodide	mercury iodide, mercury protoiodide, yellow mercury iodide
mercurous monohydrogen-o-arsenate	
mercurous nitrate	hydrated mercurous nitrate, mercury nitrate, mercury salt of nitric acid
mercurous nitrate, ammoniated	ammoniated mercury nitrate, black precipitate, Hahnemann's soluble mercury
mercurous nitrite	
mercurous oxalate	
mercurous oxide	black mercurous oxide
mercurous phosphate	neutral mercurous phosphate, normal mercurous phosphate, mercury phosphate, tertiary mercurous phosphate, trimercurous orthophosphate
mercurous sulfate	mercury sulfate
mercurous sulfide	
mercury	hydrargyrum, quick silver
mercury ammoniated	aminomercuric chloride, ammono-basic mercuric chloride, ammoniated mercuric chloride, ammoniated mercury chloride, ammoniated mercury, fusible white precipitate, Lamery's white precipitate, mecuric ammonium chloride, mercury amine chloride, mercury ammoniated, mercury cosmetic, white precipitate

mercury antimony sulfide

mercury nitride

The following is a listing of occupations with potential exposure to mercury:

Occupations with Potential Exposure to Mercury

amalgam makers
bactericide makers
barometer makers
battery makers, mercury
boiler makers
bronzers
calibration instrument makers
cap loaders, percussion
carbon brush makers
caustic soda makers
ceramic workers
chlorine makers
cinnabar ore processors
commercial artists
dental amalgam makers
dentists
direct current meter workers
disinfectant makers
disinfectors
drug makers
dye makers
electric apparatus makers
electroplaters
embalmers
explosive makers
pharmaceutical workers
photoengravers
photographers
pressure gage makers
refiners, mercury
seed handlers
sign painters
silver extractors
switch makers, mercury
farmers
feltmakers
fingerprint detectors
fireworks makers
fungicide makers
fur preservers
fur processors
gold extractors
hatters
histology technicians
ink makers
insecticide makers
investment casting workers
jewelers
laboratory workers, blood
laboratory workers, chemical
lamp makers, fluorescent
lamp makers, mercury arc
manometer makers
mercury workers
miners, mercury
neon light makers
paint makers
paper makers
percussion cap makers
pesticide workers
tanners
taxidermists
textile printers
thermometer makers
vacuum pump makers
vapor tube makers
vinyl chloride makers
wood preservative workers

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

The following should be considered:
- —History of blood disease,
- —hemorrhages into the skin or mucous membrane,
- —syphilis,
- —reddening of the skin,
- —scurvy, and
- —inflammation of several nerves (polyneuritis) due to other industrial poisons (e.g., lead) or in chronic excessive alcohol intake.

Nonoccupational Exposure

Potential nonoccupational sources of mercury include:
- —Air pollution (particularly in industrialized areas burning fossil fuels),
- —use of mildew proofing and antifouling paint,
- —use of medicinals of mercurical compounds,
- —broken home thermometers, barometers using mercury columns, and
- —consumption of fish (ocean fish, swordfish, tuna, cod, halibut, mackerel) NOTE: Local Environmental Protection Agency (EPA) standards should be checked to determine the applicability of this potential source.

Signs and Symptoms

Acute Poisoning

Acute industrial toxicity is rare, and associated signs and symptoms are:
- —Inflammation of the gums (gingivitis),
- —salivation,
- —loss of teeth,
- —gastrointestinal upset (diarrhea),
- —kidney failure (toxic nephrosis),
- —cardiac function abnormalities, and
- —tremor may exist as an isolated finding.

Chronic Poisoning

In chronic poisoning, all acute symptoms may occur but the onset may be slower and insidious. Additional oral symptoms associated with ingestion or inhalation which may occur are:

—Characteristic "blue line" on the gums, similar to lead poisoning,
—upper respiratory tract inflammation, and
—inflammation of the membrane that lines the eyelid and the front of the eyeball (conjunctivitis).

"Erethism", a form of anxiety neurosis and personality changes first described in the felt hat industry, may occur. The triad of gingivitis, tremor, and emotional instability described may be practically specific. Erethism may be accompanied by:

—Self-consciousness,
—timidity,
—inappropriate embarrassment,
—anxiety indecision,
—inability to concentrate,
—dependency,
—depression,
—resentment of criticism, and
—irritability on excitement.

Headache, fatigue, weakness, drowsiness, or insomnia may follow. In advanced cases, hallucination, memory loss, and intellectual deterioration may occur.

Circulatory disturbances may be linked to emotional disturbances and result in:

—Blushing,
—excessive perspiration, and
—dermographia (ability to sketch figures on skin).

Central nervous system symptoms are:

—Personality disorders as described above,
—tremor, one of the most prominent signs. It is the fine intention type and can be seen in the face and arms but rarely in the legs; it may progress to the coarse type and convulsions. Tremor also affects handwriting.
—speech disorders such as "scanning speech" (hesitancy, slurring of words, and difficulty in pronunciation). This may be more severe in organic mercury poisoning.

—motor and sensory deficits such as:
--Unsteady walk, may be spastic,
--ataxia in severe cases, may affect both arms and legs,
-hyperactive tendon reflexes,
-toes extend when foot is stimulated (plantar extensor response),
--numbness, prickling, and tingling sensations (paresthesias),
-severe nerve pain (neuralgia),
-decreased sensitivity to taste and smell,
-postural sensation loss (loss of position sense), and
--muscle pain and cramps.

Signs and symptoms associated with the eye are:
—Constriction of visual fields, seen in severe cases,
—fine punctate opacities in lens (mercuria lentis),
—defects in accommodation and eye muscular balance,
—lens reflex; slit-lamp examination reveals brownish colored lusterless reflex from the anterior capsule of the lens. This may be due to mercury deposits in the anterior capsule and may depend on the duration of exposure; visual clarity is not affected.
—continuous involuntary movement of the eyeball in any direction (nystagmus),
—eye muscle paralysis (paralysis of external rectus),
—dimness of vision (amblyopia), and
—blind spots or areas in the field of vision (scotomas).

Ear symptoms are:
—Possible loss of hearing,
—vertigo, and
—hypo-excitability of vestibular function (middle ear insensitivity).

Skin symptoms are:
—A pallor that is unassociated with anemia and
—allergic hypersensitivity may occur.

Laboratory and Clinical Exposures

Additonal data that will assist in arriving at a correct diagnosis are:

Blood

—excess lymph cells (lymphocytosis)
—increase in the number of cells that stain readily with the acid stain eosin (eosinophil count increase)
—electrophoretic pattern of serum proteins are consistent with nephrosis
—increased blood urea in nitrogen (BUN)
—increased quantites of creatinine
—increased uric acid (with or without gout)

Urinalysis

—urine mercury levels above 300 milligrams per day are likely to be associated with symptoms
—proteinuria
—changes associated with nephrosis (hyaline casts)

Feces

—mercury levels above 10 milligrams per day

Kidney

—evidence of nephritis

There may be no correlation between urinary mercury excretion and clinical evidence of mercury poisoning, since prolonged exposure may induce kidney (rena) injury and decreased urinary excretion. Also, urinary mercury may be increased in workers exposed to mercury, but who may or may not exhibit symptomatology. However, urinary values are useful guides to early exposure.

Epidemiology

Scientific literature has well documented the fact that chronic exposure to mercury can result in complex alterations to a worker's physiological state. The primary effects are on the central nervous system. These effects manifest themselves in varied signs and symptoms, as well as altering the worker's performance capabilities.

In the studies that follow, oropharyngeal changes, other than those of the teeth and gums, showed some dose-response relation; abnormalities of the teeth and gums were shown not to be dose-related however.

In an 18-month study of 142 workers from 4 plant groups (3 plants were engaged in chlor-alkali manufacturing, and 1 plant manufactured magnetic materials), Miller et al.[68] reported changes in neuromuscular indices of tremor and electromyography (EMG) in a significant number of workers when blood concentrations of mercury exceeded approximately 0.1 milligram of mercury per liter. The duration of chronic exposure to metallic mercury vapor ranged from 6 months to 20 years with a mean of approximately 9 years.

Shandar and Simson[69] reported a study of 334 workers in a variety of occupations or industries with mercury in air levels ranging from approximately 0 to 2.0 milligrams per cubic meter and exposure periods ranging from 1 month to 38 years. Symptoms including bleeding gums, tremor, metallic taste, and insomnia were associated with urine mercury values greater than 0.3 milligram of mercury per liter. Symptoms including headache, nervousness, tiredness, and abdominal upset were associated with a urine mercury content of 0.1 to 0.3 milligram per liter.

Rentos and Seligman[70] reported a study of 9 mercury mine and mill locations. Average work area air concentrations between 0.08 and 0.73 milligrams of mercury per cubic meter were associated with clinical evidence of mercury poisoning found in 18 out of 83 workers examined. Symptoms included loss of teeth, sore gums, loose teeth, salivation, headaches, personality changes, and tremor. Workers exposed to average air concentrations less than 0.03 milligram of mercury per cubic meter displayed no symptoms.

The following table relating symptoms and exposure data has been adopted from Turrian et al.[71] and NIOSH[72]:

SYMPTOMS OBSERVED IN 58 MERCURY WORKERS

	Air Concentration (milligrams of mercury per cubic meter)		
	0.01-0.06	0.05-0.23	0.3-0.6
Number of workers	26	15	17
Average exposure, years	9.1	16.7	7.4
Tremor	19%	20%	29%
Erethism	8%	33%	29%
Impaired memory	0%	13%	18%
Demographia	8%	27%	18%
Gingivitis	42%	40%	35%
Bad teeth or dentures	46%	67%	41%

(Turrian et al., 1956, and NIOSH, 1973)

McGill et al.[73] reported a study of 58 workers in a mercury-cell chlorine plant where mercury vapor levels ranged between 0.08 and 0.10 milligram of mercury per cubic meter. No signs or symptoms of poisoning were detected. Urine mercury samples were usually between 0 and 0.157 milligram of mercury per liter; blood mercury levels were between 0 and 0.003 milligram of mercury per liter.

Smith et al.[74] reported a 1-year study of 567 workers exposed to mercury in 21 chlor-alkali plants where air concentrations of vapor ranged from less than 0.01 to 0.27 milligram of mercury per cubic meter with a mean of 0.065 milligram of mercury per

cubic meter. Smith concluded that loss of appetite, weight loss, and objective tremors were dose-related. Also, when exposure was greater than 0.10 milligram of mercury per cubic meter, there was an appreciably higher incidence of abnormal reflexes.

The following data relating mercury exposure to blood levels and urine levels have been reported by Smith et al.[74] and are taken in table form from NIOSH.[72]

RELATIONSHIP OF MERCURY EXPOSURE TO BLOOD MERCURY LEVELS*

TWA** Exposure Level Groups (milligrams per cubic meter)	Number of Workers	Percentage of Group within Blood Level Range (micrograms per 100 milliliters)			
		<1	1-5	6-10	>10
Controls 0.00	117	69.3	30.7	0.0	0.0
<0.01	27	33.3	63.0	3.7	0.0
0.01-0.05	175	20.6	74.9	4.0	0.6
0.06-0.10	77	10.4	81.8	6.5	1.3
0.11-0.14	53	3.8	22.6	26.4	47.2
0.24-0.27	26	0.0	19.2	26.9	53.9

(Smith et al., 1970 and NIOSH, 1973)

*Expressed as percentage of each exposure level group with designated ranges of blood mercury levels.

**Time-weighted averages

The data support Elkins' suggestion of a "biological threshold value," a urine level of 0.25 milligram of mercury per liter. Smith reports a corresponding blood level of about 6 micrograms of mercury per 100 milliliters.

RELATIONSHIP OF MERCURY EXPOSURE TO MERCURY LEVELS IN URINE, UNCORRECTED FOR SPECIFIC GRAVITY*

TWA** Exposure Level Groups (milligrams per cubic meter)	Number of Workers	Percentage of Group within Urine Level Range (milligram per liter)					
		<0.01	.01-.10	.11-.30	.31-.60	.61-1.0	>1.00
Controls 0.00	142	35.2	62.7	2.1	0.0	0.0	0.0
<0.01	29	6.9	86.2	6.9	0.0	0.0	0.0
0.01-0.05	188	6.9	66.0	24.5	2.7	0.0	0.0
0.06-0.10	91	0.0	62.6	30.8	6.6	0.0	0.0
0.11-0.14	60	3.3	18.3	31.7	16.7	23.3	6.7
0.24-0.27	27	0.0	14.3	29.6	44.5	7.4	3.7

(Smith et al., 1970, and NIOSH, 1973)

*Expressed as percentage of each exposure level group within designated ranges of urine mercury levels

**Time-weighted averages

The following sections in quotes are from the National Institute for Occupational Safety and Health:[72]

In a study of the records of 1,173 hatters, Baldi et al.[75] reported "300 cases of mercury poisoning resulting from exposure to concentrations ranging from 0.5 to greater than 2.0 milligrams of mercury per cubic meter. One hundred of these cases resulted in permanent disability. Although some cases were reported at exposure levels below 0.5 milligram of mercury per cubic meter, there were no cases reported for workers exposed to less than 0.1 milligram of mercury per cubic meter."

NIOSH concludes that the results of the epidemiological surveys on mercury exposure "demonstrate that the higher the concentrations of mercury in air the greater the likelihood that an exposed worker will develop signs or symptoms of mercury intoxication although it cannot be assured that toxicity will develop at high exposure levels."

Evidence of Exposure

Air Sampling and Analysis

The NIOSH approved air sampling method uses adsorption on a two section tube containing silvered Chromosorb P in one section and carbosieve B in the other section. In addition, the suspect air is screened with a portable mercury meter or detector to determine the airborne mercury concentration. The direct-reading instrument determines the length of time that the air is sampled with the adsorption tube. Five methods previously used are:

1. Mercury vapor meters or detectors,
2. gold chloride on silica gel,
3. selenium sulfide coated paper,
4. impingement, and
5. length of stain detector tubes.

The NIOSH approved analysis method uses cold vapor atomic absorption. Six analysis methods previously used are:

1. Visual reading of length of stain detector tubes,
2. direct-reading meters or detection instruments,
3. visual color determination of gold chloride on silica gel,
4. visual color determination of selenium sulfide coated paper,
5. dithizone colorimetric method, and
6. Barnes method.

These methods are not intended to be exclusive but other methods should be justified.

Allowable Exposure Limits

Standards adopted by the Occupational Safety and Health Administration (OSHA) limit mercury (inorganic) exposure to 0.1 milligram of mercury per cubic meter of air as a ceiling

value. The NIOSH recommended standard is 0.05 milligram of mercury per cubic meter of air as an 8-hour time-weighted average.

Conclusion

Diagnosis of occupational mercury poisoning is based on the following:

1. Confirmed history of occupational exposure to mercury,
2. mercury in the blood and urine (level may not correlate well with the severity of disease), and
3. clinical findings compatible with mercury poisoning.

NOTE: Hunter states that a high urinary excretion of mercury is of diagnostic significance only when the signs and symptoms of mercury poisoning can be demonstrated.