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This manual details ten guidelines for the determination of occupational diseases. Prepared for use by Workmen's Compensation adjudicators, State Compensation Boards and Commissions, and medical advisory groups, this publication is intended to assist in establishing the work and substance relatedness of disease in individual workers. This guide expands the previously published A Guide to the Work-Relatedness of Disease (DHEW-NIOSH Publication No. 77-123). A listing of occupations with potential exposure, medical examinations, differential diagnosis, symptoms, clinical analysis, epidemiology, exposure data, and threshold limit values are included for each agent.

17. Key Words and Document Analysis. 17a. Descriptors

Antimony Compounds
Organo-antimony
Benzene
Coke Oven Emissions
Cotton Dust
Lead
Mercury
Nitrogen Dioxide
Crystalline Silica
Sulfur Dioxide

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CHAPTER 1

# ANTIMONY AND ITS COMPOUNDS (EXCEPT STIBINE)

## INTRODUCTION

Antimony is a silvery, lustrous metal or gray lustrous powder; its chief ore is stibnite. Antimony may cause adverse health effects through inhalation, ingestion, and skin absorption. Dust and fumes of antimony and its compounds are sources of the hazard.

Antimony is frequently encountered as a fine dust in industry with inhalation being the usual route of entry. Dust may be ingested by swallowing accumulations which have been deposited in the upper respiratory tract.

Nonoccupational exposure may occur through ingestion (i.e. antimony dissolved from enamel glazed utensils used for acidic foods and fluids such as lemonade) or inhalation (i.e. clothing impregnated with antimony trioxide for flameproofing). However, exposures are low, except in industry.

The symptoms of early antimony poisoning are similar to arsenic (NOTE: See Arsenic Guide), and the two elements are often encountered together in nature. Antimony compounds are irritating to the skin and mucous membranes often resulting in dermatitis, gingivitis, rhinitis, inflammation of the upper and lower respiratory tracts including pneumonitis, gastritis, conjunctivitis, and ulceration of the nasal septum (cartilage separating the nostrils) and larynx. The weakness and fatigue characteristic of the chronic poisoning may be due to anemia caused by antimony. Cardiac injury and cases of sudden death have been reported in persons exposed to antimony.

Antimony has been found to cause pneumoconiosis in workers exposed to the ore stibnite. Antimony may produce changes in the lung detectable by X-ray; lung function may also be affected.

"Antimony spots" is a dermatitis caused by antimony trioxide in which there is intense itching followed by skin eruption. Lesions tend to occur in hot weather due to dust accumulating on moist skin areas.

Chromosome damage in human cells has been induced by antimony (Patton and Allison, 1972).

Antimony can form many compounds, most of which are less toxic than antimony. The following is a listing of common compounds and some common names followed by a listing of occupations with potential exposure to antimony:

| Chemical Name | Common Names |
|---|---|
| antimony | antimony black, antimony regulus, stibium |
| antimony arsenate | |
| antimony arsenite | |
| antimony dioxysulfate | |
| antimony ethoxide | triethyl antimonite |
| antimony α-mercaptoacetamide | antimony thioglycolamide |
| antimony lactate | antimonine, antimony salt of lactic acid |
| antimonyl pyrogallol | |
| antimony oxychloride | algaroth powder, antimonyl chloride, antimony chloride oxide, basic antimony chloride, mercurius vitae |
| antimony oxysulfide | cathusian powder, kermes mineral, kermesite, pyrostibnite antimony blend, sulfurated antimony |
| antimony pentachloride | antimonic chloride, antimony perchloride, butter of antimony |
| antimony pentafluoride | antimony fluoride |
| antimony pentaiodide | antimony iodide |
| antimony pentasulfide | antimonic sulfide, antimonial saffron, antimony red, antimony persulfide, antimony sulfide, golden antimony sulfide |
| antimony pentoxide | antimonic anhydride, antimonic acid, antimonic oxide, antimony pentaoxide, stibic anhydride |
| antimony potassium dimethyl cysteino tartrate | |
| antimony potassium oxalate | potassium-antimony oxalate, potassium oxalatoantimonate |
| antimony potassium tartrate | antimony potassium salt of tartaric acid, potassium antimony tartrate, potassium antimonyl tartrate, tartrated antimony, tartar emetic, tartarized antimony |

| Chemical Name | Common Names |
|---|---|
| antimony sodium dimethylcysteino tartrate | |
| antimony sodium gluconate | antimony gluconate complex sodium salt, antimony gluconate sodium, gluconic acid antimony sodium derivative, sodium antimony gluconate, sodium stibogluconate, triostam, T.S.A.G. |
| antimony sodium tartrate | antimony sodium oxide L-tartrate, Emeto-Na, sodium antimonyl tartrate, stibunal |
| antimony sodium thioglycollate | antimony sodium thioacetate, mercaptoacetic acid antimony derivative sodium salt |
| antimony sulfate | antimony salt of sulfuric acid, antimonous sulfate, antimony trisulfate |
| antimony tetroxide | antimony oxide |
| antimony tribromide | antimonous bromide, antimony bromide |
| antimony trichloride | antimonous chloride, antimony chloride, butter of antimony, caustic antimony, mineral butter |
| antimony trichloride solution | antimony chloride solution, liquid butter of antimony |
| antimony trifluoride | antimonous fluoride, antimony fluoride |
| antimony triiodide | antimonous iodide, antimony iodide |
| antimony trioxide | antimony oxide, antimony white, antimony bloom, diantimony trioxide, Exitelite, flowers of antimony, Senarmontite, sulfuret of antimony, black antimony, Valentinite Weisspiess-glanz |
| antimony triselenide | |
| antimony tritelluride | antimony telluride |
| antimony trisulfide | antimony glance, antimony orange, antimony sulfide, antimonous sulfide, antimonite, crimson antimony, gray antimony, needle antimony, stibnite |

| Chemical Name | Common Names |
|---|---|
| emetine antimony iodide | antimony emetine iodide |
| lead antimonate | antimony yellow, Naples yellow |
| oxo (tartrato) antimonate (1-) aniline | aniline antimonyl tartrate, antimonyl aniline tartrate |
| sodium antimonate | antimony sodiate |
| sodium antimonyl adonitol | |
| sodium antimonyl D-arabitol | |
| sodium antimonyl biscatechol | |
| sodium antimonyl tert-butyl catechol | |
| sodium antimonyl catechol thiosalicylate | sodium antimonous-3-catechol thiosalicylate, stibsol |
| sodium antimonyl citrate | |
| sodium antimonyl erythritol | |
| sodium antimonyl D-funcitol | |
| sodium antimonyl gluco-guloheptitol | |
| sodium antimonyl glycerol | |
| sodium antimonyl 2,5-methylene D-mannitol | |
| sodium antimonyl 2,4-methylene D-sorbitol | |
| sodium antimonyl xylitol | |

## Occupations with Potential Exposures to Antimony

Antimony ore smelters
Antimony workers
Babbitt metal workers
Battery workers, storage
Brass founders
Britannia metal workers
Bronzers
Burnishers
Cable splicers
Ceramic makers
Compositors
Copper refiners
Dye makers
Electroplaters
Explosives makers
Fireworks makers

Occupations with Potential Exposures to Antimony (cont.)

Flameproofers
Foundry workers
Glass makers
Glaze dippers, pottery
Gold refiners
Insecticide makers
Insulators, wire
Lake color makers
Lead burners
Lead hardeners
Lead shot workers
Leather mordanters
Linotypers
Match makers
Metal bronzers
Miners
Monotypers
Mordanters
Organic chemical synthesizers
Paint makers
Painters
Perfume makers
Pewter workers
Pharmaceutical workers
Phosphor makers
Pigment makers
Plaster cast bronzers
Porcelain workers
Pottery workers
Printers
Pyrotechnics workers
Rubber makers
Semiconductor workers
Solder makers
Stereotypers
Stibnite miners
Storage battery workers
Textile dryers
Textile flameproofers
Textile printers
Type metal workers
Typesetters
Vulcanizers
Zinc refiners

## MEDICAL EVALUATION AND DIFFERENTIAL DIAGNOSIS

The following should be considered:

- inflammation of several nerves (polyneuritis) due to other industrial poisons (e.g. lead) or in chronic excessive alcohol intake
- diseases of the heart muscles caused by toxins (toxic cardiomyopathies
- diseases of the stomach
- rashes due to food and drug sensitivity (urticaria)
- jaundice due to phosphorus or arsenic poisoning

### Signs and Symptoms

#### Acute Poisoning

Acute poisoning from antimony seldom occurs as an occupational exposure. Signs and symptoms of acute antimony poisoning are chiefly gastrointestinal and include:

- violent vomiting
- continuous diarrhea with mucus

- hepatitis

- kidney involvement with blood in the urine (hematuria)

- shock may be associated with slow, irregular respiration and a subnormal temperature

- death may occur in several hours

Chronic Poisoning

Chronic antimony poisoning can result from the inhalation of dusts or fumes, by ingestion, or by skin absorption. General complaints are:

- irritability.

- fatigue

- numbness and tingling (neuritis)

- muscular aches

- loss of appetite (anorexia)

- gastrointestinal symptoms such as nausea, constipation

- headache

- dizziness

- chest pain

Respiratory symptoms are:

- inflammation of the larynx (laryngitis)

- inflammation of the trachea (tracheitis)

- cough

- difficulty in breathing (dyspnea)

Antimony and its compounds are generally regarded as primary skin irritants. Lesions usually appear on exposed, moist areas of the body but rarely on the face. Skin disorders include:

- sores resembling chickenpox (pustular, covered with a crust)

- blistering of the lips

- perforation of the nasal septum (cartilage separating the nostrils)

- nodular ulcers on the neck, axilla, or groin (moist areas of the body)

Other symptoms of chronic poisoning are:

- inflammation of the gums (gingivitis)
- inflammation of the mouth (stomatitis)
- inflammation of the membrane that lines the eyelids and the front of the eyeball (conjunctivitis)
- inflammation of the cornea (keratitis)
- constipation
- joint pains (arthralgia)
- possible diseases of the skeletal, voluntary, or cardiac muscles

Either liver or kidney failure or both can occur in the late stages of the disease, and death may result.

Laboratory and Clinical Examinations

Additional tests which will assist in arriving at a correct diagnosis are:

Blood

- antimony level above 6.0 milligrams per deciliter
- white blood count may show a shift to the left (a preponderance of less mature white cells)

Urine

- antimony level above 1.0 milligram per liter

Liver

- liver function studies may reveal hepatic injury

Electrocardiogram

- acute poisoning may induce ST and T wave changes, auricular fibrillation, and possibly ventricular arrhythmias

Chest X-ray

- may indicate pneumonitis or pneumoconiosis (small opacities in all regions of the lung)

## EPIDEMIOLOGY

A great variety of signs and symptoms associated with industrial exposure to antimony has been detailed in the scientific literature. Epidemiological studies have demonstrated the relationship between antimony and myocardial changes, transient pneumonia, chronic dermatitis, irritation of the mucous membrane, and irritation of the digestive tract among workers in trades and occupations such as mining antimony-containing ores, flameproofing, abrasives, and the printing industry. The data demonstrate that worker exposure is dependent on the specific antimony compound present in the work environment. This should be considered when reviewing the following information:

Brieger et al[1] reported a 2 year study of 125 workers in an abrasives industry (using antimony trisulfide) who had been exposed to air concentrations of antimony ranging from 0.58 to 5.5 milligrams per cubic meter, with most values over 3.0 milligrams per cubic meter. During the study, 6 workers died suddenly in addition to 2 other workers who died of chronic heart disease. Four of the deceased were under 45 years of age. Since no autopsies were performed, the cause of death was not determined definitely but in all but 1 case heart disease was suspected. Fourteen had a blood pressure of over 150/90 mm of mercury, and 24 of under 110/70 mm of mercury. Thirty-seven out of 75 showed changes in the electrocardiogram, mostly of the T-waves; 7 out of 111 had ulcers. Irritation of the skin, mucous membranes, or respiratory tract was not found. Urine samples contained 0.8 to 9.6 milligrams of antimony per liter of urine. (Elkins suggested that 1 milligram of antimony per liter of urine is a safe level). When the use of antimony trisulfide was discontinued, no further deaths from heart disease or abnormal increase of cardiovascular disorders occurred. Electrocardiographic changes were reported to persist in 12 of 56 workers who were re-examined. When unattended, evidence showed that injury to the heart may remain undetected during the long latency periods.

Cooper et al[2] reported a study of 28 workers who had been engaged in processing antimony from a crude ore for 1 to 15 years. Workers were exposed to dusts of antimony trioxide and antimony ore; antimony concentrations in air ranged from 0.081 to 138 milligrams per cubic meter with the heaviest concentration being in bagging operations. Of the workers with abnormal pulmonary function, 1 had definite small opacities, 1 had very early changes, and 2 had negative chest X-rays. Three workers with either suspicious or definite chest X-ray abnormalities had normal pulmonary function. Electrocardiograms were obtained from 7 workers (3 had antimony pneumoconiosis); 6 workers had normal tracings, and 1 showed a slight bradycardia. Antimony in the urine samples ranged from 0 to 1.02 milligrams per liter of urine. These low values correspond to the low solubilities of antimony oxides. In contrast, Brieger et al reported that workers exposed to lower air concentrations of the more soluble antimony trisulfide had higher urinary levels of antimony.

Renes[3] published a report on a 5 month study of 69 smelter workers who were exposed to antimony trioxide; antimony levels in air ranged from 4.69 to 11.81 milligrams per cubic meter. (Antimony, arsenic, and caustic soda were present in the air of the smelter but antimony was the predominating aerial contaminant.) Six workers showed definite pneumonitis which cleared after removal from exposure and treatment. The pathological conditions most frequently diagnosed were dermatitis and rhinitis, next in frequency were inflammation of the upper and lower respiratory tract (including pneumonitis), and less than 4% of the cases had conjunctivitis, gastritis, and septal perforations reported.

McCallum[4,5,6] reported a study of 268 process workers. Twenty-three workers (8.5%) exhibited simple pneumoconiosis changes (Categories 1-3, I.L.O. International Classification, Geneva 1958); associated defects in lung function were not present. One furnace worker with antimony pneumoconiosis who had retired at age 65 had 0.055 milligram of antimony per liter of urine 7 months after leaving work and 0.028 milligram per liter 4 years after leaving work.

Karajovic et al[7] reported a study of 160 men employed at an antimony smelter for 5 to 12 years. No symptoms were found which could be related to systemic antimony poisoning but skin changes and pneumoconiosis were present. Thirty-one out of 62 smelter workers had simple pneumoconiosis. No massive lesions were observed. In 20 workers of the total studied, 8 had pneumoconiosis, 13 had emphysema, and 9 had chronic bronchitis.

Taylor[8] reported a study of 7 workers who were accidentally exposed to the fume of antimony trichloride. Air contained up to 73 milligrams of antimony per cubic meter when leaks developed during the refining of the ore. After 24 hours, 5 men had symptoms of gastrointestinal disturbance including abdominal pain and persistent anorexia (loss of appetite). All cases reported an absence of abdominal tenderness and a return of normal appetite by the tenth day. The urine antimony content exceeded 1.0 milligram of antimony per liter of urine during the incident and fell rapidly to less than 0.02 milligram 24 hours after exposure. No lung changes or evidence of persistent intoxication were observed after exposure.

Rodier and Souchere[9] report chronic poisoning which resulted from occupational exposure in Moroccan antimony mines. Workers complained of mild symptoms including headaches, sleeplessness, vertigo, appetite loss, and muscular pains. Antimony was detected in the urine and hair. Although the blood-cell picture was altered, antimony was not detected in the blood. Gallina and Luvoni[10] report cases of antimony poisoning among workers exposed to antimony pentasulfide in a Milan glass factory. Among symptoms reported were nausea, vomiting, diarrhea, bitter taste in the mouth, and a characteristic leucocyte count shift.

## EVIDENCE OF EXPOSURE

### Sampling and Analysis

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

1. impingement
2. electrostatic precipitation

The NIOSH approved method for air sample analysis uses atomic absorption spectrophotometry. Two methods previously used are:

1. rhodamine B
2. 9-methyl-2,3,7-trihydroxyfluor-6-one

The above are not intended to be exclusive but alternative methods should be justified.

### Allowable Exposure Limits

The Federal standard for antimony and its compounds is 0.5 milligram per cubic meter of air based on an 8 hour time-weighted average exposure. Occupational exposure to antimony in amounts greater than this is evidence of a possible causal relationship between disease and occupation.

## CONCLUSION

Diagnostic criteria for occupational antimony poisoning are based on the following:

1. confirmed history of occupational exposure to antimony or one of its compounds
2. clinical findings compatible with antimony poisoning
3. urine antimony levels in excess of 1.0 milligram per liter
4. blood antimony levels in excess of 2.0 milligrams per 100 grams

CHAPTER 2

# INORGANIC ARSENIC (EXCEPT ARSINE)

## INTRODUCTION

Arsenic is found in small amounts in soils and waters and in foods, particularly seafoods. For industrial and commercial uses, it is removed from ores during smelting operations as arsenic trioxide which is used in the manufacture of most other arsenic compounds.

Exposure to arsenic can be through ingestion (swallowing accumulations of dust deposited in the upper respiratory tract), inhalation, or percutaneous (absorbed through the skin) as arsenic can be widely distributed throughout body tissues. It is also found in hair, nails, urine, and feces. Nonoccupational exposures to arsenic have resulted in average urinary arsenic levels of 0.014 to 0.25 milligram of arsenic per liter with the highest reported levels being attributed to probable seafood consumption (Dinman, 1960). Therefore, when evaluating occupational exposure to arsenic, nonoccupational exposure of the individual must also be carefully examined.

Arsenic is an irritant to the skin and to mucous membranes and can cause acute and chronic poisoning. Acute arsenic poisoning rarely occurs in industry.

The corrosive action of arsenic may cause perforation of the nasal septum (cartilage separating the nostrils). NOTE: There can be other causes of perforation.

Chronic arsenic poisoning induces numerous skin manifestations which include overgrowth of the horny layer of the epidermis (hyperkeratosis), sensitization, and possibly loss of hair and nails. In addition, skin cancer may be associated with chronic arsenic poisoning. These include squamous cell carcinoma, epithelioma which may arise at sites of keratoses (most common), basal cell carcinoma, and the chronic precancerous dermatitis referred to as Bowen's disease.

Arsenic may also have a depressant effect on bone marrow erythropoiesis and myelopoiesis (the process of blood cell formation).

Epidemiological data (experience with groups of people) suggests that there may be a relationship between the development of cancer in the lung, lymphatic system, and/or skin.

Arsenic can form many compounds. The following is a list of common compounds and some common names followed by a listing of occupations with potential exposure to inorganic arsenic:

| Chemical Name | Common Names |
|---|---|
| ammonium arsenate | ammonium acid arsenate, diammonium salt of arsenic acid, diammonium arsenate, dibasic ammonium arsenate, secondary ammonium arsenate |
| arsenic | gray arsenic, metallic arsenic |
| arsenic acid | orthoarsenic acid, true arsenic acid |
| arsenic acid, magnesium salt | magnesium arsenate |
| arsenic acid, sodium salt | sodium arsenate, sodium metaarsenate |
| arsenical nickel | niccolite, nickel arsenide |
| arsenic acid, trisodium salt, heptahydrate | trisodium arsenate heptahydrate |
| arsenic bisulfide | arsenic sulfide, realgar |
| arsenic diiodide | arsenic iodide |
| arsenic disulfide | arsenic bisulfide, arsenic monosulfide, arsenic sulfide, realger, red arsenic, red arsenic sulfide, red arsenic glass, ruby arsenic |
| arsenic hemiselenide | |
| arsenic oxychloride | |
| arsenic pentachloride | arsenic chloride |
| arsenic pentafluoride | arsenic fluoride |
| arsenic pentaselenide | |
| arsenic pentasulfide | diarsenic pentasulfide |
| arsenic pentoxide | arsenic acid anhydride, arsenic oxide, arsenic anhydride, arsenic pentaoxide, ZOTOX |
| arsenic phosphide | |
| arsenic tribromide | arsenic bromide, arsenous bromide |
| arsenic trichloride | arsenic butter, arsenic chloride, arsenious chloride, arsenous chloride, butter of arsenic, caustic arsenic chloride, fuming liquid arsenic |

| Chemical Name | Common Names |
|---|---|
| arsenic trifluoride | arsenic fluoride, arsenious fluoride, arsenous fluoride |
| arsenic triiodide | arsenic iodide, arsenious iodide, arsenous iodide |
| arsenic trioxide | arsenic sesquioxide, arsenious acid, arsenious oxide, arsenolite, arsenious trioxide, arsenite, arsenous acid, arsenous acid anhydride, arsenous anhydride, arsenous oxide, arsenous oxide anhydride, claudite, claudetite, crude arsenic, white arsenic |
| arsenic triselenide | arsenious selenide, arsenous selenide |
| arsenic trisulfide | arsenic persulfide, arsenic sulfide, arsenic tersulfide, arsenic yellow, arsenious sulfide, arsenous sulfide, auripigment, orpiment, ping's gold, ping's yellow, yellow arsenic sulfide |
| arsenious acid, monosodium salt | sodium arsenite, sodium metaarsenite |
| arsenious acid, sodium salt | sodium salt polymers of arsenious acid, sodium orthoarsenite |
| arsenious acid, potassium salt | Fowler's solution, potassium arsenite, potassium metaarsenite |
| arsenopyrite | aronarsenousulfide, arsenical pyrites, mispickel |
| beta-arsenic | black arsenic |
| calcium arsenate | calcium-o-arsenate, calcium ortho-arsenate, Chip-Cal, Fen Cal, Kalo, Spracal, tricalcium ortho-arsenate |
| copper arsenite | acid copper arsenite, arsenious acid, copper, copper arsenide, cupric arsenite, Scheele's green, Scheele's mineral, Swedish green |
| disodium arsenate | disodium salt of arsenic acid, sodium acid arsenate, sodium arsenate dibasic anhydrous |
| lead arsenate | acid lead arsenate, arsenate of lead, lead salt of arsenic acid, dibasic lead arsenate, lead orthoarsenate, plumbous arsenate |

| Chemical Name | Common Names |
|---|---|
| lead arsenite | lead m-arsenite, lead o-arsenite, lead metarsenite |
| mercuric arsenate | mercury arsenate, mercury arsenite |
| metaarsenic acid | |
| orthoarsenic acid | acid-true arsenic acid, arsenic acid, arsenic acid 75, arsenic acid hemihydrate, liquid arsenic, meta-arsenic acid, orthoarsenic acid hemihydrate |
| nickel arsenate | nickel o-arsenate, nickelous arsenate |
| sodium arsenite | disodium salt of arsenious acid |

Occupations with Potential Exposures to Inorganic Arsenic

Acetylene workers
Acid dippers
Alloy makers
Aniline color makers
Aniline workers
Arsenic workers
Artificial flower makers
Babbitt metal workers
Bleaching powder makers
Boiler operators
Book binders
Brass makers
Bronze makers
Bronzers
Cadmium workers
Candle (colored) makers
Canners
Carpet makers
Carroters, felt hat
Cattle dip workers
Ceramic makers
Ceramic enamel workers
Commercial artists
Copper smelters
Crop dusters
Defoliant applicators
Defoliant makers
Dimethyl sulfate makers
Disinfectant makers
Drug makers
Dye makers
Electroplaters
Electrolytic copper workers
Enamelers
Enamel makers
Etchers
Exterminators
Farmers
Feather workers
Ferrosilicon workers
Fertilizer makers
Fireworks makers
Flypaper makers
Galvanizers
Glass makers
Gold extractors
Gold refiners
Hair remover makers
Herbicide makers
Hide preservers
Ice makers
Illuminating gas workers
Ink makers
Insecticide makers
Japan makers
Japanners
Jewelers
Lead burners
Lead shot makers
Lead smelters
Leather workers
Lime burners

Occupations with Potential Exposures to Inorganic Arsenic (cont.)

Metal cleaners
Metal refiners
Miners
Mordanters
Nitrocellulose makers
Ore smelters
Organic chemical synthesizers
Paint makers
Painters
Paper hangers
Paper makers
Petroleum refinery workers
Pharmaceutical makers
Pigment makers
Plastic workers
Plumbers
Preservative makers
Printing ink workers
Pyrotechnics workers
Rayon makers
Rodenticide makers
Sealing wax makers
Semiconductor compound makers
Sheep dip workers
Sign painters
Silver refiners
Soda makers
Soil sterilizer makers
Solderers
Submarine workers
Sulfuric acid workers
Tanners
Tar workers
Taxidermists
Textile printers
Tinners
Tree sprayers
Type metal workers
Varnish makers
Vine dressers
Wallpaper printers
Warfare gas makers
Water weed controllers
Weed sprayers
Wine makers
Wire drawers
Wood preservative makers
Wood preservers
Zinc chloride makers
Zinc miners
Zinc refiners

## MEDICAL EVALUATION AND DIFFERENTIAL DIAGNOSIS

The following should be carefully evaluated to determine if present symptoms are in fact associated with a previous disease or injury:

- history of Addison's disease
- hemolytic anemia
- viral infections
- upper respiratory tract infections
- gastrointestinal irritants
- gastritis or lower gastrointestinal diseases
- non-obstructive kidney failure (anuria) from lead or mercury poisoning

Nonoccupational Exposure

Arsenic poisoning may occur from a home hobby or other activities such as:

- farming (use of pesticides)
- gardening
- wine making
- diets very heavy in seafood (a high intake of lobster, oysters, and mussels may elevate blood arsenic levels)
- wood preserving
- living near industrial plants which utilize arsenic compounds

Signs and Symptoms

Acute Poisoning

Acute poisoning usually occurs from exposure to arsenic-containing dust. However, it rarely occurs in industry. High exposure may be tolerated without symptoms of systemic poisoning.

In acute poisoning following ingestion of inorganic arsenic, symptoms develop within 1/2 to 4 hours and are characterized by constriction of the throat followed by:

- inability to swallow or difficulty in swallowing (dysphagia)
- epigastric pain
- vomiting and abdominal pain
- watery diarrhea
- shock may occur with severe fluid loss, and death may ensue in 24 hours

If the acute effects are survived, the following may develop:

- inflammation of the skin involving redness and flakiness (exfoliative dermatitis)
- inflammation of the nerves, mainly of the hands and feet (peripheral neuritis)

Acute poisoning due to inhalation is extremely rare in industry. When it occurs, respiratory and central nervous system symptoms predominate. Gastrointestinal symptoms are less frequent and occur later.

Respiratory and central nervous system symptoms occurring initially are:

- cough
- chest pain
- difficult or labored breathing (dyspnea)
- giddiness
- headache
- extreme general weakness

Signs and symptoms which may occur later include:

- nausea
- vomiting
- colic

Chronic Poisoning

The signs and symptoms of chronic arsenic exposure resemble many diseases, including early lead poisoning, and are characterized by:

- insidious onset of malaise
- abdominal complaints
- severe itching (pruritis)
- weakness
- loss of appetite (anorexia)
- weight loss
- inflammation of the gums (gingivitis) and/or mouth (stomatitis)
- inflammation of the mucous membrane of the nose (rhinitis)
- inflammation of the kidney (nephritis)
- decreased pulmonary function

Inorganic arsenical compounds are primary cutaneous (skin) irritants, and signs and symptoms include:

- redness which may be more intense around hair follicles and give the skin a mottled appearance
- brittle nails
- loss of nails and hair
- a broad white transverse line (called Mee's lines) can also be found in association with polyneuritis
- pustular, ulcerative, or gangrenous lesions
- overgrowth of the horny layer of the epidermis (hyperkeratosis) associated with thick, dry, cracking skin, often with excessive sweating of the palms and soles of the feet (hyperhidrosis)
- deposits of black pigments in different body parts (melanosis)
- hyperpigmentation of a "rain-drop" configuration (believed to be a sign of systemic, not local toxicity)
- jaundice, which may be secondary to liver involvement

Signs and symptoms associated with the nervous system are less common and occur in fewer than 5% of all cases:

- inflammation of the peripheral nerves (peripheral neuritis)
- numbness, tingling, "pins and needles", heightened sensation
- symmetrical weakness in feet and legs
- fasciculation and gross tremors, muscular incoordination (ataxia), shuffling gait
- decreased deep tendon reflexes with foot and wrist drop
- mental confusion

In chronic arsenic poisoning, the liver may be involved resulting in:

- enlargement of the liver (hepatomegaly)
- excessive accumulation of serous fluids in the abdominal (peritoneal) cavity
- cirrhosis (liver fibrosis)

Perforation of the nasal septum (the cartilage separating the nostrils) is common in workers chronically exposed to arsenic.

Persons exposed to chronic arsenic absorption have been reported to develop carcinoma of the lung, larynx, and viscera (the abdominal organs) as well as skin. However, the relationship of arsenic to nondermal cancer is much more of an open issue.

Laboratory and Clinical Examinations

Additional tests which will assist in arriving at a correct diagnosis are:

- electrocardiographic abnormalities which indicate a direct toxic effect
- liver function studies may indicate liver cell injury
- decreased white blood cell count
- decreased red blood cell count (anemia)
- basophilic stippling of the red blood cells
- chest X-ray may reveal lung cancer
- pulmonary function tests may be decreased

The normal range of urinary arsenic levels is less than 0.1 milligram of arsenic per liter in 24 hour specimens. Levels greater than 0.2 milligram of arsenic per liter suggest exposure to limits greater than those stated on page 22. However, acquired tolerance may allow levels greater than 0.2 milligram of arsenic per liter without evidence of arsenic poisoning. Conversely, persons with urine arsenic levels less than 0.2 milligram of arsenic per liter may in fact have arsenic poisoning. An additional test that will aid in arriving at a correct diagnosis is a bioassay of nails. For fingernails, maximum arsenic levels are 0.82 to 3.5 parts per million (ppm), and for toenails, 0.52 to 5.6 ppm.

EPIDEMIOLOGY

The relationship between occupational exposure to arsenic and chronic poisoning signs and symptoms including malaise, abdominal complaints, anorexia, hyperkeratosis, and pruritis has been well documented in scientific literature.

It has been shown that arsenic may be absorbed through the skin, from the tracheobronchial tree, and from the gastrointestinal tract. For this reason, nonoccupational exposure to arsenic which can be present in the air, water, and food should be reviewed since it can also elicit a toxicological response. Arsenic absorption, however, does not necessarily indicate poisoning. These facts should be kept in mind when considering the epidemiological data.

Pinto et al[11] reported a study of 24 smelter workers who were exposed to an average airborne arsenic concentration range of 0.003 to 0.295 milligram per cubic meter with a mean value of 0.053 milligram per cubic meter. The workers were exposed to arsenic trioxide. Average urinary arsenic values ranged from 0.038 to 0.539 milligram of arsenic per liter of urine with an overall average of 0.174 milligram of arsenic per liter. The Pearson correlation factor between airborne arsenic concentrations and urinary arsenic levels over the range studied was 0.530 ($P < 0.01$) which demonstrates a statistically significant correlation. There was evidence that nonoccupational arsenic absorption from the consumption of seafood resulted in elevated urinary arsenic levels.

The following sections in quotes are from the National Institute for Occupational Safety and Health:[12]

Butzengeiger[13] reported a study of "180 vinedressers and cellarmen who were exposed to arsenical insecticides while tending the vineyards and from consuming homemade wine believed to be contaminated with arsenic. All had symptoms of chronic arsenic intoxication, and in 41, there was evidence of vascular disorders in the extremities. Of 15 cases described in detail, all had varying degrees of hyperpigmentation, and 13 had palmar and plantar keratosis; all 15 had cold hands, feet, or both which seemed to preceed the development of gangrene on toes or fingers in 6 of the 15. Urinary arsenic levels ranged from 0.076 to 0.934 milligram of arsenic per liter with a mean value of 0.324 milligram per liter. Average arsenic content in hair was 0.039 milligram of arsenic per 100 grams of hair."

In a more recent study of the electrocardiograms (ECGs) from 192 vinegrowers suffering chronic arsenic intoxication, Butzengeiger[14] reported that "55 (28.7%) revealed definite changes with 36 cases of these having no possible cause other than arsenic poisoning. ECG abnormalities included Q-T prolongation and flattened T-waves. Further study showed a decline in ECG abnormalities associated with attenuation of other symptoms of arsenic intoxication."

Lee and Fraumeni[15] reported a study of 8047 copper smelter workers who were exposed to arsenic trioxide over a 25 year period. Worker exposure data is summarized as follows:

| | 1965 SMELTER SURVEY ATMOSPHERIC ARSENIC CONCENTRATIONS (milligrams of arsenic per cubic meter of air) | | |
|---|---|---|---|
| | Heavy | Medium | Light |
| Range | 0.10 to 12.66 | 0.03 to 8.20 | 0.001 to 1.20 |
| Mean | 1.47 | 1.54 | 0.206 |
| Median | 0.185 | 0.79 | 0.010 |
| Work Area | Roaster, Kitchen, Cottrell | Reverberatory, Treater Building, Loading | Transfer System, Flue Station, Reactor Building |

(Lee and Fraumeni, 1969)

Overall mortality of the exposed workers was significantly higher than expected when compared to an unexposed population with the cause of excess deaths mainly due to malignant neoplasms of the respiratory system and diseases of the heart. They further reported the excess of respiratory cancer to be as high as eight-fold among workers who were employed for more than 15 years in "heavy" exposure areas.

Perry et al[16] reported a 1 year study of 31 chemical workers who were exposed to sodium arsenite at an English sheep-dip factory. Air concentrations of arsenic ranged from 0.110 to 4.038 milligrams of arsenic per cubic meter with a mean value of 0.562 milligram per cubic meter. Twenty-eight workers exhibited hyperpigmentation, and 9 had wart-like lesions. The average urinary arsenic concentration was 0.23 milligram of arsenic per liter; average arsenic concentration in hair was 108 parts per million.

Birmingham et al[17] emphasized that the cutaneous effect of exposure to arsenic occurs more frequently than the rare systemic toxicities. Milham and Strong[18] reported a study of smelter workers in which "80% of the workers excreted 1.0 to 3.0 milligrams of arsenic per liter of urine and had dermatitis. All workers excreting over 3.0 milligrams of arsenic per liter had dermatitis."

## EVIDENCE OF EXPOSURE

### Sampling and Analysis

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

- impingement
- electrostatic precipitation

The NIOSH approved method for air sample analysis uses atomic absorption spectrophotometry. Four methods previously used are:

- Gutziet method
- silver diethyldithiocarbamate method
- iodine microtitration
- molybdenum blue method

The above methods are not intended to be exclusive but other methods used should be justified.

Allowable Exposure Limits

The standards adopted by the Occupational Safety and Health Act (OSHA) limit arsenic and its compounds (except arsine) to 0.5 milligram per cubic meter of air (as arsenic) based on an 8 hour time-weighted average exposure. (Note: Based on arsenic's carcinogenic effects, NIOSH has recommended a reduction in the standard to 0.002 milligram per cubic meter of air as a ceiling value determined during a 15 minute sampling period.)

Arsenic is a confirmed occupational carcinogen with the target organ/tissue being the lung and skin; it is a suspect lymphatic tissue carcinogen (Ket et al eds., 1977).

The OSHA allowable limit for calcium arsenate is 1.0 milligram per cubic meter of air based on an 8 hour time-weighted average exposure.

The OSHA allowable limit for lead arsenate is 0.15 milligram per cubic meter of air based on an 8 hour time-weighted average exposure.

Occupational exposure to arsenic or its compounds in amounts greater than the OSHA limits is evidence of a possible causal relationship between disease and occupation.

CONCLUSION

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

1. confirmed history of occupational exposure to arsenic or one or more of its compounds
2. clinical findings compatible with arsenic poisoning
3. analysis of urine (or nails) for arsenic is of value in confirming exposure but is not diagnostic in itself

NOTE: See Appendix V for a complete listing of the mandatory physical examinations and medical and biological monitoring required by OSHA.

# CHAPTER 3

# BENZENE

## INTRODUCTION

Benzene, an aromatic organic compound, is a colorless liquid that is a good solvent for many other organic compounds. It is commonly called benzol and in high doses exerts an acute narcotic action on the body. Because of benzene's extreme toxicity, less toxic substances are now being substituted for it in industries such as artificial leather manufacture, rubber products manufacture, and printing.

Absorption of benzene through inhalation is the most important route of entry in industrial exposures. Benzene interferes with the body's blood forming system and can cause such disturbances as leukopenia (reduction in the number of white blood cells), aplastic anemia (the number of white blood cells predominate), thrombocytopenia (reduction in the number of platelets), and leukemia. Prolonged or repeated skin exposure can result in the development of blisters, erythema (localized redness), and a dry, scaly dermatis.

Long latency periods of 10 to 15 years from the time of exposure to the development of disease are possible. In addition, signs and symptoms of toxicity may persist after treatment.

The following is a listing of common names for benzene followed by a listing of occupations with potential exposures to benzene:

### Common Names

benzin
benzine
benzol
benzole
benzolene
bicarburet of hydrogen
carbon oil
coal naphtha
cyclohexatriene
motor benzol
mineral naphtha
phene
phenyl hydride
pyrobenzol
pyrobenzole

### Occupations with Potential Exposures to Benzene

Adhesive makers
Airplane dope makers
Alcohol workers
Aniline makers
Art glass workers
Artificial leather makers
Asbestos product impregnators
Asphalt mixers
Automotive workers
Battery makers, dry
Belt scourers
Benzene hexachloride makers
Benzene workers
Brakelining makers
Bronzers
Burnishers
Can makers
Carbolic acid makers

Occupations with Potential Exposures to Benzene (cont.)

Cast scrubbers, electroplating
Chemical synthesis
Chlorobenzene makers
Chlorodiphenyl makers
Clutch disc impregnators
Coal tar refiners
Coal tar workers
Cobblers
Coke oven workers
Cyclohexane makers
DDT makers
Degreasers
Detergent makers
Dichlorobenzene makers
Diphenyl makers
Disinfectant makers
Drug makers
Dry cleaners
Dye makers
Electroplaters
Enamelers
Engravers
Ethylbenzene makers
Explosive makers
Feather workers
Fuel oil handlers
Fumigant makers
Fungicide makers
Furniture finishers
Gas workers, illumination
Glue makers
Hairdressers
Herbicide makers
Histology technicians
Hydrochloric acid workers
Ink makers
Insecticide makers
Lacquer makers
Leather makers
Linoleum makers
Lithographers
Maleic acid makers
Millinery workers
Mirror silverers
Mordanters
Nitrobenzene makers
Nitrocellulose workers
Oil processors
Oilcloth makers
Organic chemical synthesizers
Paint makers
Painters
Paraffin processors
Pencil makers
Perfume makers
Petrochemical workers
Petroleum refinery workers
Pharmaceutical workers
Phenol makers
Photographic chemical makers
Picric acid makers
Polish makers
Pottery decorators
Printers
Putty makers
Reclaimers, rubber
Resin makers
Respirator makers
Rotogravure printers
Rubber cementers
Rubber gasket makers
Rubber makers
Shellac makers
Shoe factory workers
Shoe finishers
Soap makers
Solvent makers
Stainers
Stain makers
Styrene makers
Synthetic fiber makers
Tobacco seedling treaters
Trinitrotoluol makers
Type cleaners
Varnish makers
Vulcanizers
Wax makers
Welders
Window shade makers
Wire insulators

MEDICAL EVALUATION AND DIFFERENTIAL DIAGNOSIS

The following should be carefully evaluated to determine if present findings may be associated with a previous disease:

- history of blood disease
- bleeding abnormalities
- replacement of bone marrow by fibrous tissue (myelofibrosis)
- kidney disease
- liver disease
- serious bacterial, viral, or protozoan infection of the colon
- inflammation of several nerves (polyneuritis) due to other industrial poisons (e.g. lead) or in chronic excessive alcohol intake

Nonoccupational Exposure

Consider also that exposure to benzene may be from a hobby or home activity. Included are the following:

- woodworking
- furniture refinishing
- paint stripping
- use of dry cleaners and spot removers
- use of gasoline, paint, wax, lacquer, or leather preservatives

In addition, the following should be considered:

- any known exposure to benzene or any other poisonous substances affecting the blood or blood-forming tissues (hematologic toxins)
- ionizing radiation exposures (which also affect the blood-forming organs)
- radiomimetic substances (those which imitate radiation effects)

Signs and Symptoms

Acute Poisoning

Acute benzene intoxication occurs following exposure to high levels of benzene vapor. Signs and symptoms include:

- feeling of exaggerated well-being (euphoria)
- excitement
- exhilaration followed by drowsiness, fatigue, vertigo, nausea, and headache

- respiratory irritation and pulmonary edema
- gastrointestinal irritation, with vomiting and colic
- localized redness (erythema), blistering of the skin, petechial hemorrhage (small hemorrhagic spots on skin)
- insomnia
- giddiness
- nervousness
- paresthesia of hands and feet (numbness, prickling, and tingling)
- staggering gait
- incoherent speech
- flushed face

Continued exposure can result in unconsciousness and death from respiratory paralysis. The course of the intoxication may be enhanced by muscular exertion, emotions, and fear.

The clinical effects of accidental ingestion of benzene include:

- local irritation of the mucous membranes of the mouth, throat, esophagus, and stomach
- bronchitis, pneumonia, and collapse may ensue

Ingestion of benzene results in blood absorption and may proceed to systemic intoxication.

Chronic Poisoning

Chronic benzene poisoning can occur from daily inhalation of benzene. The clinical manifestations of chronic benzene poisoning tend to be insidious in onset and are nonspecific. They resemble those of many other diseases:

- loss of appetite (anorexia)
- headache
- weight loss
- dizziness
- irritability and nervousness
- nausea
- tiredness, lassitude, weariness, fatigue and weakness

Other symptoms may include:

- pallor
- tendency to bruise easily
- bleeding gums
- nose bleeds
- retinal hemorrhages
- excessive bleeding at the time of menstruation (menorrhagia)

Benzene exposure predisposes to leukemia of the following types: chronic myeloid, chronic lymphatic, and acute myeloblastic.

Laboratory and Clinical Examinations

Additional tests which will assist in arriving at a correct diagnosis are:

Blood

- arterial blood benzene
- red blood cells destroyed at a rapid rate (hemolysis)
- elevated young red blood cell count (reticulocytosis)
- reduced platelet count (thrombocytopenia)
- reduced white blood cell count (leucopenia), an early sign
- white blood cells predominate (aplastic anemia)
- increased mean corpuscular volume (average volume of red blood cells)
- reduced hemoglobin (that part of the red blood cell that carries oxygen and carbon dioxide)
- increased plasma bilirubin (orange-colored or yellowish pigment in bile)
- hemosiderosis of the liver, spleen, kidneys, or bone marrow (a condition characterized by the depositing of an iron-containing pigment in these sites)
- the bone marrow may have reduced or defective production of blood cells (hypoplasia); this may be seen in persons with short or long term exposure to benzene and is more common in females

- the bone marrow may have increased or defective production of red blood cells (hyperplasia); this may be seen in persons with long term exposure and is more common in males

- chromosome aberrations in peripheral and bone marrow cells occur and may persist after exposure

Urine

- increased urobilinogen

- increased phenol - less than 75 milligrams per liter is normal

- ratio of inorganic to total sulfates - normal limits are 0.05 to 0.1 milligram per liter. NOTE: Sample must be collected and test begun within one hour after workday exposure

In workers exposed to benzene, hematologic tests should be conducted monthly, while urine sulfate tests should be conducted every week. Liver and kidney function studies should also be evaluated.

EPIDEMIOLOGY

The relationship between benzene and the blood-forming tissues of the body has been demonstrated in many epidemiological studies. However, the National Institute for Occupational Safety and Health has reported that "symptomatic effects associated with benzene poisoning often do not correlate with objective findings. Even in serious cases of chronic benzene poisoning, symptomatic effects may be completely absent."[19] For this reason, a dose-response relationship has not been established. These facts should be considered when referring to the following material. Sections in quote are from NIOSH:[19]

Aksoy et al[20] reported a study of 217 workers exposed to benzene for periods of 3 months to 17 years in small shops manufacturing shoes under poorly ventilated conditions. Benzene exposures ranged between 30 and 210 parts per million (ppm). Fifty-one (23.5%) of the workers showed hemotological abnormalities, consisting of leucopenia, thrombocytopenia, or pancytopenia. No cases of leukemia were observed.

In a 7 year study of 28,500 shoeworkers who were chronically exposed to a range of 150-650 ppm benzene for from 4 months to 20 years, Aksoy et al[21] reported on 34 workers who had various types of leukemia. Acute myeloblastic leukemia was the most frequent type (14 workers, 41.1%), followed by preleukemic (6 workers, 17.6%), acute erythroleukemia (6 workers, 17.6%), and acute lymphoblastic leukemia (4 workers, 11.7%). Among 31 shoeworkers, the incidence of leukemia of 13.5 per 100,000 was significantly greater ($P < 0.001$) than the overall incidence of leukemia in the general population which was 6 per 100,000. It was reported that there was a decline in the number of cases in the last year of the study which may be attributed to the prohibition of the use of benzene.

Forni et al[22] and Hartwick and Schwanitz[23] reported benzene-induced chromosome changes in peripheral blood lymphocytes or bone marrow. In followup studies, Forni observed significantly increased rates of "unstable" and "stable" chromosome aberrations which persisted several years after exposure to benzene had ceased and clinical recovery from the poisoning had occurred.

From the data Greenberg[24] collected, he concluded that cases showing less than 5,500 white blood cells per cubic millimeter should be considered positive; 7,500 to 9,000 was considered the normal count.

Hardy and Elkins[25] reported "that levels of benzene exposure ranging from 40-80 ppm with an estimated average of 60 ppm in the artificial leather industry had produced deviations in more than 1 blood element in 16 out of 52 workers exposed."

Juzwiak[26] reported a study of "585 workers in 13 shoe plants who had been exposed to benzene in a glue mixture. Mean concentrations of benzene fluctuated from 31-156 ppm. Reduced red blood cell counts, white blood cell counts, and hemoglobin levels were reported. Ninety-one percent (91%) of the workers had reduced hemoglobin levels but only 8.5% had reduced white blood cell counts." Environmental data were inadequately documented, so it is difficult to correlate medical findings with the airborne exposures in this study.

In a report of an 11 plant study of 162 workers in the rubber coating industry, Pagnotto et al[27] concluded that the urinary phenol determination test provides a "good index of benzene exposure." The following table from NIOSH presents air-urinary correlation data:

| URINARY PHENOL LEVELS WITH CORRESPONDING EQUIVALENT ENVIRONMENTAL BENZENE EXPOSURE LEVELS | |
|---|---|
| Urine Phenol (milligrams per liter) | Approx. Av. Equiv. Benzene Air Level (ppm) |
| 100 | 10 |
| 120 | 13 |
| 140 | 16 |
| 160 | 19 |
| 180 | 22 |
| 200 | 25 |
| 220 | 27 |
| 240 | 29 |
| 260 | 31 |
| 280 | 33 |
| 300 | 35 |
| 320 | 38 |
| 340 | 41 |
| 360 | 44 |
| 380 | 47 |
| 400 | 50 |
| 420 | 53 |
| 440 | 56 |
| 460 | 59 |
| 480 | 62 |
| 500 | 65 |
| 520 | 68 |
| 540 | 71 |
| 560 | 74 |
| 580 | 77 |
| 600 | 80 |

(NIOSH, 1974)

Sherwood[28] cites the following guidelines for monitoring benzene exposure using routine assay of phenol in urine:

1. values over 100 milligrams per liter - significant risk indicated

2. values over 30 milligrams per liter - probability of benzene exposure indicated

3. values less than 10 milligrams per liter - no benzene exposure indicated

## EVIDENCE OF EXPOSURE

### Sampling and Analysis

The NIOSH approved air sampling method uses adsorption on activated charcoal. Four methods previously used are:

1. absorption in anhydrous methanol
2. absorption in nitrating solutions
3. direct collection of whole-air samples
4. absorption on silica gel

The NIOSH approved method for air sample analysis uses gas chromatography. Two methods previously used are:

1. colorimetric evaluation
2. ultraviolet spectrophotometry

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

Various types of direct reading field instruments are also available to measure benzene concentrations in air. They include detector tubes, combustible gas meters, flame ionization meters, portable gas chromatographs, and portable infrared analyzers.

### Allowable Exposure Limit

The Federal standard for benzene is 1 ppm based on an 8 hour time-weighted average exposure, with 5 ppm as a maximum peak above the acceptable ceiling for a maximum duration of 15 minutes. If initial exposure measurements show that concentrations are below 1/2 of the permissible level, or 0.5 ppm, periodic monitoring will not be required. Above that level, however, monitoring and routine medical surveillance as well as other practices will be triggered. Benzene has been classed as a suspect leukemogen by OSHA. NIOSH has identified benzene as a confirmed occupational carcinogen for blood-forming tissue and a suspect carcinogenic agent for lymphatic tissue (Key et al eds., 1977).

## CONCLUSION

Diagnostic criteria for chronic occupational benzene poisoning is based on meeting the following:

1. confirmed history of occupational benzene exposure
2. clinical signs and symptoms consistent with benzene poisoning
3. findings from blood studies consistent with aplastic anemia and/or leukopenia and/or thrombocytopenia and/or leukemia

It is possible to have the signs and symptoms of chronic benzene poisoning with a normal blood picture.

Post-mortem findings in acute benzene poisoning via inhalation include extensive petechial hemorrhages in the brain, pleurae (lining of the chest cage and lungs), pericardium, urinary tract, mucous membranes, and skin.

Urine phenol and sulfate levels are not diagnostic by themselves but are indicative of excessive exposure to benzene.

NOTE: See Appendix V for a complete listing of the mandatory physical examinations and medical and biological monitoring required by OSHA.

# CHAPTER 4

# COKE OVEN EMISSIONS

## INTRODUCTION

Coke oven emissions are a complex mixture of particulates, vapors, and gases that result from the destructive distillation of bituminous coal in the production of coke. (Coke finds its major application in the production of steel.)

Coke oven workers have an increased risk of developing cancer of the lung, urinary tract, and skin. This risk has been shown to be related to the area of employment (i.e. workers employed at the top of the oven have the greatest risk followed by part-time topside and side oven jobs) and the length of employment. Epidemiological studies have also shown that exposure to coke oven emissions increases the risk of nonmalignant respiratory diseases such as bronchitis and emphysema. It should be noted that smoking habits, previous exposure in a dusty industry or environment, and oven work area have been identified as significant factors in the development of these diseases. These factors should be considered when determining whether nonmalignant diseases are caused wholly or in part by occupational exposure to coke oven emissions.

Long latency periods of 15 to 25 years from the time of exposure to the development of carcinoma have been observed.

The following is a listing of occupations with potential exposures to coke oven emissions:

### Occupations with Potential Exposures to Coke Oven Emissions

Coke oven door cleaners - luterman
Coke oven door machine operators
Coke oven heater
Coke oven larry car operators
Coke oven lidmen-larrymen
Coke oven maintenance men
Coke oven patcher
Coke oven pusher operators
Coke oven quench car operators
Coke oven tar chaser

## MEDICAL EVALUATION AND DIFFERENTIAL DIAGNOSIS

The following should be considered:

- any history of skin, genitourinary or pulmonary disease should be carefully evaluated to determine the relationship between the previous disease and the claimant's present condition

- a respiratory questionnaire, a sample of which is shown on page 91 of A Guide to the Work-Relatedness of Disease (NIOSH Publication No. 77-123), can be useful in evaluating respiratory symptoms

Signs and Symptoms of Cancerous Conditions

Lungs

The early signs and symptoms of lung (bronchogenic) cancer are nonspecific and include:

- cough
- coughing up mucus or phlegm (expectoration)
- coughing up blood (hemoptysis)
- weight loss which may not be associated with symptoms until late in the course of the disease
- collapsed or airless condition of a section of the lung (atelectasis)
- wheezing respiration
- segmental emphysema (trapping of air in a part of the lung) or fibrosis (scar tissue formation)
- pneumonitis
- abscess formation
- signs of metastasis (spreading of the cancer from one organ to another)

Genitourinary

The signs and symptoms associated with the kidney, bladder, and urinary tract include:

- blood in the urine which may be intermittent (hematuria)
- pains between the rib and pelvis area (loin pains)
- an abdominal mass
- weight loss
- fatigue

Skin

- an ulcer that does not heal
- a small mass on the skin
- a lesion that bleeds easily or may ooze fluid and form a scab
- pain over the area exposed (the infiltration site)

Signs and Symptoms of Noncancerous Conditions

Respiratory - Bronchitis, Pulmonary Fibrosis, Chronic Obstructive Pulmonary Disease

Signs and symptoms can include:

- cough
- coughing up mucus or phlegm
- frequent upper respiratory infections
- shortness of breath
- the use of extra-respiratory muscles to assist breathing

The following grading system has been devised to classify the degree of bronchitis according to symptomatology:

| BRONCHITIS GRADING SYSTEM | | |
|---|---|---|
| GRADE | LABEL | SYMPTOMS |
| 0 | Asymptomatic | No positive responses or only rare respiratory symptoms |
| 1 | Probable acute bronchitis | Cough OR sputum production present occasionally, but for less than 3 months of the year and for less than 2 years |
| 2 | Acute bronchitis | Cough AND sputum with the same frequency and duration as in Grade 1 |
| 3 | Severe acute bronchitis | Symptoms as in Grade 2 plus dyspnea on exertion |
| 4 | Probable chronic bronchitis | Cough OR sputum present for 3 months each year and for at least 2 years |
| 5 | Chronic bronchitis | Cough AND sputum with some frequency and duration as in Grade 4 |
| 6 | Moderately severe chronic bronchitis | Symptoms as in Grade 5 plus dyspnea on heavy exertion (i.e. hill climbing) |
| 7 | Severe chronic bronchitis | Symptoms as in Grade 5 plus dyspnea on slight exertion (i.e. slow pace on level) |

(Mittman et al, 1974)

NOTE: Cigarette smoking has been associated with increasing the severity of symptoms and should be considered when reviewing each case.

Skin

Signs and symptoms which may be present include:

- the skin reacts abnormally to light (photosensitization) with resultant:
  - -- diffused redness (erythema)
  - -- swelling of body tissues (edema)
  - -- burning of the skin with hyperpigmentation developing later
- acne and blackheads (comedones)
- thickening of the skin (keratosis)
- contact dermatitis
- formation of benign tumors or warts (papillomas)
- inflammation of follicles

Systemic

- loss of appetite (anorexia)
- nausea
- vomiting

Eye

- inflammation of the membrane that lines the eyelids and the front of the eyeball (conjunctivitis)

Laboratory and Clinical Examinations

Additional tests which will assist in arriving at a correct diagnosis when cancerous conditions are being evaluated are:

Lungs

- chest X-ray
- examination of sputum for cancer cells (sputum cytology)
- visual examination of the bronchi (bronchoscopy)
- microscopic examination of a scalene node (scalene node biopsy)

- percutaneous (effected through the skin) needle biopsy

- lung biopsy

Genitourinary

- urinalysis

- X-ray examination of the kidney and ureters (intravenous pyelogram)

- visual examination of the bladder (cystoscopy)

- kidney biopsy

- abdominal X-ray

- examination of the urine for cancer cells

Skin

- total removal of lesion, and microscopic (histological) examination

Additional tests which will assist in arriving at a correct diagnosis when noncancerous conditions are being evaluated are:

Respiratory

- chest X-ray

- pulmonary function test

Skin

- examination under Wood's light for fluorescence of residual tar

EPIDEMIOLOGY

The disease response to coke oven emissions has been shown to be related to length of employment and exposure level. Various studies have indicated that coal carbonization workers have a high risk of developing cancer of the skin, lungs, and urinary tract. These workers have also demonstrated an increase in mortality from cancer of the lungs and kidneys.

A causal but unproven relationship has been reported for carcinoma of the larynx, the nasal sinuses, pancreas, stomach, and blood-forming organs (leukemia). Evidence of an elevated risk of nonmalignant diseases such as bronchitis or emphysema has also been presented.

It has been shown that topside coke oven workers experience a higher rate for carcinoma of the lung than other coke oven workers, and non-topside workers have a higher rate for carcinoma of the kidney.

The reports of disease response in parenthesis are from NIOSH:[29]

Doll[30] reported an "81% excess of lung cancer deaths among gas works pensioners (gas retort workers) in comparison with the general population." Lloyd[31] reported that "coke oven workers had an average lung cancer mortality rate 2-1/2 times that predicted by the experience of all steelworkers."

Lloyd[31] and Redmond et al[32] reported that "men employed at the Allegheny County coke ovens for 5 or more years exhibited a lung cancer rate that was 3.5 times the expected rate." Also, men employed full time topside of the coke ovens had a lung cancer mortality rate which was 9 times the expected rate, for partial topside it is almost 2-1/2 times the expected rate, and for side oven only it is more than 1-1/2 times the expected mortality. (All of these rates are based on 5 or more years exposure in the job category).

Henry et al[33] reported an "excess risk of bladder cancer among men employed at coal carbonization processes." Redmond et al[32] did not observe an excessive incidence of bladder cancer in 4661 coke oven workers; however, when it is considered that this is a comparatively rare cancer site with a long latent period and the study population has an extremely high risk for cancer of several other sites, the possibility of excess mortality cannot be ruled out.

Over a 43 year period, Henry also reported 84 cases of epitheliomatous ulceration (cancer of the skin) including 40 scrotal cancers. Among men with prior coke oven employment, 11 fatal scrotal cancers were reported.

The United Steelworkers of America[34] reported a study of 112 coke oven employees in which over 50% were diagnosed as having some lung impairment (i.e. pneumoconiosis, emphysema, fibrosis and chronic bronchitis).

## EVIDENCE OF EXPOSURE

### Air Sampling and Analysis

The NIOSH approved air sampling method uses mechanical filtration.

The NIOSH approved analytic method uses gravimetric techniques. Three methods previously used are:

1. chromotography
2. fluorometry
3. spectrophotometric techniques

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

Allowable Exposure Limits

The standards adopted by the Occupational Safety and Health Act (OSHA) have established a limit of 0.2 milligram per cubic meter of air for coal tar pitch volatiles (benzene-soluble fraction of total particulate), based on an 8 hour time-weighted average exposure. Coke oven emissions are confirmed occupational carcinogens with the target organ/tissue being the kidney, lung, and skin. (NOTE: A reduction in the standard to 0.15 milligram per cubic meter of air has been proposed by NIOSH.)

CONCLUSION

Diagnostic criteria for occupational carcinoma due to exposure to coke oven emissions are:

1. confirmed history of occupational exposure to coke oven emissions

2. diagnosis of carcinoma as determined by laboratory evaluation and clinical findings

NOTE: As carcinoma also occurs in the population which is not occupationally exposed to coke oven emissions, the decision whether a claimant's carcinoma is work related is most difficult.

Criteria for diagnosing occupational noncancerous conditions due to exposure to coke oven emissions include the following:

1. confirmed history of occupational exposure to coke oven emissions

2. clinical findings of respiratory, genitourinary, or skin examination as outlined above and medical history

NOTE: See Appendix V for a complete listing of the mandatory physical examinations and medical and biological monitoring required by OSHA.

# CHAPTER 5

# COTTON DUST

## INTRODUCTION

Among workers in cotton mills particularly where there exist high levels of dust exposure, for example in breakdown, opening, and card rooms, the pneumoconiosis, byssinosis, has been found to be highly prevalent. The term "pneumoconiosis" applies to conditions caused by accumulation of a variety of dusts capable of inducing a tissue reaction in the lung. Inhalation of cotton dust results in a type of pneumoconiosis which is known to cause decreases of the ventilatory capacity of the lungs as well as symptoms of chest tightness and dyspnea (labored or difficult breathing). Symptoms become progressively more severe during the work week, and workers experience relief over the weekend. However, there is a critical point when irreversible pulmonary (lung) changes occur.

No specific cause has been found for byssinosis. There is sufficient evidence to suggest that cotton dust itself, as well as an agent in the bracts (a major component of cotton trash), can lead to the liberation of excess histamine (a naturally occurring broncho-constrictor) when either or both come into contact with the bronchial mucosa (mucous membrane). Airway constriction may be induced by the deposition of cotton dust in the airways in the absence of an immunological reaction.

It has been proposed that a polyphenol extracted from the cotton plant causes a Type III or Arthus reaction (a severe local inflammatory response) which in turn causes the disease. Though an antibody to cotton antigen was found in workers with byssinosis, this may represent a nonspecific immune (allergic) reaction.

The lungs of byssinotic workers contain an excessive amount of reticulin and collagen (connective tissue). Rounded yellow dust bodies with a central black core are visible. Emphysematous changes and pathological evidence of chronic bronchitis are also seen.

The severity of reaction depends on a number of factors: duration of exposure to cotton dust, individual susceptibility, and the composition of the cotton dust fiber or particle size, and concentration. Smoking has been found to be significantly associated with byssinosis for workers in opening, picking, and carding operations.

Byssinosis is similar in many respects to nonoccupational bronchitis and emphysema and is often confused with it, especially in advanced stages when symptoms of shortness of breath and tightness of the chest are severe every day.

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

Occupations with Potential Exposure to Cotton Dust

Beaming operators (cotton mill)
Carders (cotton mill)
Carding machine operators (cotton mill)
Cleaner operators (cotton mill)
Cleaners (cotton mill)
Combining machine operators (cotton mill)
Drawing frame operators (cotton mill)
Dryer operators (cotton mill)
Gin stand operators (cotton gin)
Ginners
Grinders (cotton mill)
Handpickers (cotton)
Lint cleaner operators (cotton mill)
Openers (cotton mill)
Pickers (cotton mill)
Press box operators (cotton mill)
Roving frame operators (cotton mill)
Slashing operators (cotton mill)
Spindle pickers (cotton)
Spinners (cotton mill)
Spooling operators (cotton mill)
Stripper operators (cotton)
Stripper operators (cotton mill)
Twisters (cotton mill)
Weavers (cotton mill)

MEDICAL EVALUATION AND DIFFERENTIAL DIAGNOSIS

The following should be considered in the Medical Evaluation:

- respiratory allergy
- chronic lung disease
- other diseases of the cardiopulmonary system
- smoking

A worker having a positive history for any or all of the above is at increased risk from occupational exposure to cotton dust.

A respiratory questionnaire, such as that in the NIOSH Criteria Document on cotton dust can be useful in evaluating the extent and importance of the following respiratory symptoms:

- breathlessness
- sputum production
- chest pain
- cough
- wheezing

Signs and Symptoms

These may be shown as soon as after a few hours of exposure or may even first appear as long as after 10 years of exposure to cotton, hemp, or flax dust.

The following clinical grading (or staging) system has been devised to classify the degree of byssinosis according to symptomatology:

Grade 1/2: occasional chest tightness on the first day of the working week

Grade I: chest tightness and/or difficulty in breathing on every first day of the working week

Grade II: chest tightness and difficulty in breathing on the first and on other days of the working week

Grade III: Grade II symptoms, accompanied by permanent (irreversible) pulmonary incapacity (i.e. chronic respiratory symptoms and decreased ventilatory capacity not relieved by appropriate drugs)

Although early symptoms of byssinosis are reversible if exposure to cotton dust ceases, a point is reached where permanent, irreversible airway obstruction persists.

When the disease is classed as Grade II, continued exposure to cotton dust can induce episodes of bronchitis and/or asthma.

In Grade III, chronic lung disease can be accompanied by the following symptoms:

- chronic bronchitis and progression to emphysema

- cough with mucopurulent (consisting of mucus and pus) sputum

The chest X-ray may be normal in Grade III.

Though byssinosis was originally thought to be related to bronchial asthma, there are several important differences between them. The onset of the symptoms of byssinosis occur gradually, while asthma develops soon after exposure to an antigen.

Other conditions which may result from cotton dust exposure are as follows: "weaver's cough," "mill fever," "mattress maker's fever," "stripper's asthma," "grinder's asthma," and "cotton card room asthma."

Other acute illnesses resulting in fatigue, loss of appetite (anorexia), headache, nausea, and vomiting, have occurred from the use of low grade or stained cotton. The aerobacter cloacae bacteria may be a cause.

Laboratory and Clinical Examinations

Pulmonary function tests are not conclusive but are generally necessary in making a correct diagnosis:

- a significant decline in one second forced expiratory volume ($FEV_1$) from the morning of the first day of the working week to the afternoon of the same day. The decrement is greater on the first day of the working week than later in the week.

- decreased forced vital capacity (FVC). This measurement is less sensitive than $FEV_1$ and more dependent upon subject cooperation.

NOTE: The findings of these tests (i.e. $FEV_1$ and FVC) have a greater validity when performed together rather than separately. However, these values are usually obtained from the same test record.

The following grading system which uses one second forced expiratory volume ($FEV_1$) has been devised to classify ventilatory impairment. The mean of the two highest values of $FEV_1$ is compared to standard normal values. The acute effect of dust exposure is measured before and after the first full workday after a weekend. The difference between the values before and after cotton dust exposure is utilized with the following guides:

Function (F) 0: no demonstrable acute effect of the dust on ventilatory capacity; no evidence of chronic ventilatory impairment; $FEV_1$ is greater than 80% of the predicted value

Function (F) 1/2: slight acute effect of dust on ventilatory capacity; no evidence of chronic ventilatory impairment; $FEV_1$ is greater than 80% of the predicted value

Function (F) 1: definite acute effect of dust on ventilatory capacity; no evidence of chronic ventilatory impairment; $FEV_1$ is greater than 80% of the predicted value

Function (F) 2: evidence of a slight to moderate irreversible impairment of ventilatory capacity; $FEV_1$ is 60 to 79% of the predicted value

Function (F) 3: evidence of a moderate to severe irreversible impairment of ventilatory capacity; $FEV_1$ is less than 60% of the predicted value

F 0 are normal workers without evidence of permanent ventilatory impairment or acute response to the dust. F 1/2 and F 1 are workers who are showing an acute response to the dust but have at present no evidence of permanent impairment of ventilatory capacity. The more severe F 1 grade should be accepted as indicating that further exposure to textile dust is likely to cause permanent ventilatory impairment in the worker. Grades F 2 and F 3 include those workers who have some permanent impairment of ventilatory capacity (Bouhoys et al, 1970).

NOTE: There are no characteristic changes in chest X-ray.

EPIDEMIOLOGY

Population studies in the cotton spinning industry have shown that the occurrence of the symptom of chest tightness on the first day of the working week (a specific symptom of byssinosis) depends upon work place,

type of exposure, length of exposure, and the quality of the raw cotton being processed. The symptom may be accompanied by detectable loss of ventilatory capacity and increased breathlessness.

In a 14 plant study of the records of 6631 employees, Martin and Higgins[35] reported a significant association between byssinosis and bronchitis, and between smoking and byssinosis for employees in opening, picking, and carding. Three percent (3%) had subjective symptoms (by history) of byssinosis; 0.8% indicated both symptoms and objective signs by a 10% or greater drop during the working day of the one-second forced expiratory volume ($FEV_1$). Martin and Higgins also reported the anatomy of the mouth to be an important factor related to pulmonary function testing. Due to an obstructive phenomenon unrelated to the lower pulmonary system, ill-fitting or loose dentures were reported to cause a considerable drop in the forced expiratory volume in one second at the end of eight hours of work ($FEV_{1a}$).

Shilling et al[36] reported a study of 190 cardroom and blowroom workers. Thirty-nine percent (39%) of the workers were normal, 35% had Grade I byssinosis, and 25% had Grade II byssinosis. It was further reported that 45% of the carders and 65% of the strippers and grinders and blowroom workers had byssinosis.

Shilling[37] reported a survey of 28 mills spinning the coarser grades of raw cotton that demonstrated a "geography" of disease. The highest prevalence of disease was found in groups working the nearest to carding engines. This finding could not be explained by age differences or in years of exposure to dust.

Zuskin et al[38] reported a study of 120 men and 38 women workers in two air-conditioned cotton mills. The average length of employment in these mills was 16 years, and the average age was 43. The following table from the report summarizes chronic respiratory symptoms and illnesses:

| BYSSINOSIS GRADES | | | | |
|---|---|---|---|---|
| Number of Workers | Grade 1/2[a] | Grade I[b] | Grades II[c] and III[d] | Total Number of Workers |
| Carders M (59) | 4 | 6 | 5 | 15 (25%) |
| Spinners M (61) | 2 | 5 | 2 | 9 (15%) |
| Spinners F (38) | 3 | 0 | 0 | 3 ( 8%) |
| Total (158) | 9 | 11 | 7 | 27 (17%) |

(Zuskin et al, 1969)

a - Cough on the first day of the working week or chest tightness sometimes on the first day of the working week, or both.
b - Chest tightness every first day of the working week.
c - Chest tightness on the first day of the working week and other work days.
d - Chest tightness on all days.

Eight carders from Mill A with a history of byssinosis had an average $FEV_1$ decrease of 0.82 liter. In 6 men in both mills who had worked for less than 1 year, the reduction of $FEV_1$ on the first working day of the week ranged from 0.09 to 0.43 liter. $FEV_1$ decreased significantly during the work shift on the first working day of the week for all workers in both mills. The following table from the report summarizes total dust concentrations in all work areas:

DUST CONCENTRATIONS AT DIFFERENT SITES OF WORK IN MILLS A AND B (in milligrams per cubic meter)

| Work Place | N* | "Respirable" Range | "Respirable" Mean | Total Range | Total Mean |
|---|---|---|---|---|---|
| Mill A Carders | 3 | 0.76 to 1.05 | 0.87 | 1.47 to 1.92 | 1.63 |
| Mill A Spinners | 3 | 0.80 to 1.07 | 0.92 | 1.79 to 2.15 | 1.91 |
| Mill B Carders | 4 | 0.43 to 0.54 | 0.50 | 1.23 to 1.70 | 1.55 |
| Mill B Spinners | 4 | 0.50 to 0.60 | 0.55 | 1.25 to 1.75 | 1.54 |

(Zuskin et al, 1969)

*Number of samples in each location.

In a study of 509 cotton textile workers, Schrag and Gullett[39] classified 63 (12%) as having byssinosis. Twenty-nine percent (29%) of cardroom workers, 10% of weavers, and 9% of spinners had byssinosis. Dust concentrations ranged from 0.3 milligram per cubic meter to 5.8 milligram per cubic meter. The following table taken from the report summarizes respiratory symptoms among the workers studied.

RESPIRATORY SYMPTOMS IN MALE CARDERS, SPINNERS,
AND WEAVERS WITH BYSSINOSIS AND IN THOSE WITHOUT BYSSINOSIS

| | Number of Workers | Persistent Cough (%) | Persistent Phlegm (%) | Persistent Wheezing (%) | Chest Illness in the Last Three Years (Absence from Work) (%) | Believed They Had Bronchitis (%) | Believed They Had Asthma (%) |
|---|---|---|---|---|---|---|---|
| Carders | | | | | | | |
| With byssinosis | 28 | 71 | 57 | 50 | 17 | 25 | 10 |
| Without byssinosis | 67 | 37 | 32 | 24 | 9 | 4 | 1 |
| Total | 95 | 47 | 40 | 32 | 12 | 10 | 4 |
| Weavers | | | | | | | |
| With byssinosis | 11 | 91 | 82 | 64 | 36 | 27 | 18 |
| Without byssinosis | 76 | 36 | 26 | 15 | 12 | 8 | 7 |
| Total | 87 | 44 | 33 | 21 | 15 | 11 | 8 |
| Spinners | | | | | | | |
| With byssinosis | 7 | 43 | 71 | 14 | 0 | 0 | 14 |
| Without byssinosis | 52 | 25 | 23 | 15 | 14 | 10 | 6 |
| Total | 59 | 27 | 29 | 15 | 12 | 8 | 7 |

(Schrag and Gullett, 1970)

Workers with byssinosis had a significantly lower average $FEV_1$ than did workers without byssinosis when this measurement was made in the middle of the week. Schrag and Gullett concluded that a single measurement of $FEV_1$ would not identify all symptomatic workers.

Molyneux and Tombleson[40] report a 3-year study of 1359 cotton workers and 227 man-made fiber workers in 14 cotton spinning and 2 man-made fiber spinning mills. Total dust levels averaged 3.1 milligram per cubic meter in cotton fiber coarse mills and 1.2 milligram per cubic meter in cotton fiber medium mills. The total prevalence of byssinosis, 26.9%, was higher in coarse fiber than in medium fiber cotton mills. [The count of yarn spun in the medium mills ranged from 10 to 50 (60 to 12 Tex) and 1 to 24 (600 to 25 Tex) in the coarse mills.] In the coarse mills, symptoms developed in some men and women within the first 4 years of exposure; in medium mills, symptoms developed between 5 and 10 years' exposure. Symptoms similar to those of byssinosis occurred in 10 (4.4%) of the total population of the man-made fiber mills; however, all 10 had a previous history of exposure to cotton dust.

In a study of 10,133 workers employed in 19 plants that process raw cotton in the manufacture of yarn, Imbus and Suh[41] found a marked relationship between the incidence of byssinosis and bronchitis and lowered pulmonary function. Cigarette smoking appeared to further increase the incidence of bronchitis and lower pulmonary function. A drop in $FEV_1$ during the working day, though associated with, was often present without byssinosis symptoms.

Merchant et al[42] reported a study of 441 workers employed in a modern cotton-synthetic blend mill in which 20% of those working in preparation areas, 2% of those in yarn processing areas, and 6% of all employees were diagnosed as byssinotic. Among men, the byssinosis index increased with smoking, and the bronchitis index increased with smoking plus dust exposure. Byssinotic workers were found to have more chronic bronchitis and dyspnea than matched control workers.

## EVIDENCE OF EXPOSURE

### Sampling and Analysis

The NIOSH approved air sampling method uses mechanical filtration.

The NIOSH approved method for air sample analysis requires the reweighing of a preweighed filter after collection of the sample. An additional analysis method is based on beta-radiation counting of a size selective sample.

The above methods are not intended to be exclusive, but other methods should be justified.

## Allowable Exposure Limits

The standards adopted by the Occupational Safety and Health Act (OSHA) have established a limit for exposure to raw cotton dust (total dust sample) of one milligram per cubic meter of air based on an 8 hour time-weighted average exposure. (NOTE: A reduction in the standard to 0.2 milligram per cubic meter as lint-free cotton dust has been proposed by NIOSH.)

## CONCLUSION

Diagnosis of occupational byssinosis due to exposure to cotton dust is based on the following:

1. confirmed history of occupational exposure to cotton dust over a period of years.

2. chest tightness and dyspnea which appear on the first work day following absences from exposure to cotton dust.

3. a reduction of ventilatory capacity following return to work on the first work day and during the work day as demonstrated by lung function tests.

NOTE: Although most persons with Grade I, II, or III byssinosis have a moderate to marked decrease in $FEV_1$, absence of this decrement does not rule out the diagnosis of byssinosis in persons with symptoms.

Chronic bronchitis may or may not be associated with byssinosis. The person with chronic bronchitis will usually experience chest tightness when exposed to any dusty atmosphere, whereas, the early byssinotic is affected only by cotton dust and is worse on returning to work on the first working day or after several days absence.

Older females without byssinosis employed in mills for many years while rearing families and performing usual household duties will show a significant drop in the difference in before and after-shift ($FEV_1$) tests. It has been concluded that physical work causes extreme fatigue in these women, and already lowered pulmonary function should be interpreted as muscular fatigue rather than a significant increase in bronchial resistance (Martin and Higgins, 1976).

See Appendix V for a complete listing of the mandatory physical examinations and medical and biological monitoring required by OSHA.

# CHAPTER 6

# 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 (Ket 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 decrease 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 |

| Chemical Name | Common Names |
|---|---|
| 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 | |

| Chemical Name | Common Names |
|---|---|
| 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 subnitrite |
| lead ocher | massicot |
| lead orange | orange mineral |
| lead oxalate | |
| lead oxychloride | Cassel yellow, laurionite, matlockite, Mendipite |

| Chemical Name | Common Names |
|---|---|
| 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 | |

| Chemical Name | Common Names |
|---|---|
| lead telluride | altaite |
| lead tetrachloride | |
| lead tetrafluoride | plumbic fluoride |
| lead tetroxide | C.I. 77578, C.I. Pigment Red 105, gold satinobre, lead orthoplumbate, 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 wulframate, raspite, stolzite |
| lead vanadate | lead-m-vanadate, lead metavanadate, 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
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
Chemical equipment makers
Chippers
Chlorinated paraffin makers
Cigar makers
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

Occupations with Potential Exposures to Inorganic Lead (cont.)

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
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
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
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

Occupations with Potential Exposures to Inorganic Lead (cont.)

Type founders
Type setters
Vanadium compound makers
Varnish makers
Vehicle tunnel attendants
Wallpaper printers
Welders

MEDICAL EVALUATION AND DIFFERENTIAL DIAGNOSIS

The following should be considered:

- acute appendicitis
- chronic gastric or duodenal (first part of the small intestines) ulcer
- carcinoma of the stomach
- 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
- 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
- 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
  - -- 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) secondary to lead exposure
- convulsions

Gastrointestinal symptoms are:

- colon spasms (colic)
- nausea, vomiting
- loss of appetite
- 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)
- increased serum iron

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

- basophilic stippling
- 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)
- 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
- 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

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 erythrocyte 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 creatinine
- 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[43] 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 is 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.[44])

Elkins[45] 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, averaging, correspond to an air concentration of 0.2 milligrams lead per cubic meter of air.

The data in the following table relating average and median blood lead content with exposure and duration of employment has been adapted from Dreesen et al,[46] the Committee on Biologic Effects of Atmospheric Pollutants,[47] and the National Institute for Occupational Safety and Health.[44]

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 (milligram 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)

Tola and Nordman[48] reported a study of 335 men representing the general population and 2209 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[49] 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 concentrations 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.

Lancranjan et al[50] 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-Aminolevulinic 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[51] reported a mortality study of 7032 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.[44]

## EVIDENCE OF EXPOSURE

### Air Sampling and Analysis

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

1. electrostatic precipitator
2. impingement

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

1. polargraphic
2. spectrographic
3. dethizone procedure
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
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.

See Appendix V for a complete listing of the mandatory physical examinations and medical and biological monitoring required by OSHA.

# CHAPTER 7

# INORGANIC MERCURY

## INTRODUCTION

Mercury, a chemically stable element which is liquid at room temperature, is found everywhere - in rocks, soils, plants, animal, 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, cinnebar, 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 occurs in a chronic and an acute form. Chronic excessive exposure to inorganic mercury may result in 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 |

| Chemical Name | Common Names |
|---|---|
| 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 |

| Chemical Name | Common Names |
|---|---|
| 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 | |

| Chemical Name | Common Names |
|---|---|
| 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 |

| Chemical Name | Common Names |
|---|---|
| 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 proto-iodide, 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 |

| Chemical Name | Common Names |
|---|---|
| mercury ammoniated | aminomercuric chloride, ammonobasic mercuric chloride, ammoniated mercuric chloride, ammoniated mercury chloride, ammoniated mercury, fusible white precipitate, Lamery's white precipitate, mercuric 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
Cinnebar 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
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, 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

Occupations with Potential Exposure to Mercury (cont.)

Pharmaceutical workers
Photoengravers
Photographers
Pressure gage makers
Refiners, mercury
Seed handlers
Sign painters
Silver extractors
Switch makers, mercury
Tanners
Taxidermists
Textile printers
Thermometer makers
Vacuum pump makers
Vapor tube makers
Vinyl chloride makers
Wood preservative workers

## MEDICAL EVALUATION AND DIFFERENTIAL DIAGNOSIS

The following should be considered:

- history of blood disease
- hemorrhages into the skin or mucous membrane
- syphilis
- reddening of the skin
- scurvy
- 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 mercurial compounds
- broken home thermometers, barometers using mercury colomns
- 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
- 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
- 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

- 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

- 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)

  -- 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)
- blind spots or areas in the field of vision (scotomas)

Ear symptoms are:

- possible loss of hearing
- vertigo
- hypo-excitability of vestibular function (middle ear insensitivity)

Skin symptoms are:

- a pallor that is unassociated with anemia
- allergic hypersensitivity may occur

Laboratory and Clinical Examinations

Additional tests 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 quantities 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 (renal) 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 to 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[52] 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[53] 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[54] 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[55] and NIOSH:[56]

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[57] 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[58] 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[58] and are taken in table form from the NIOSH.[56]

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

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 |
| <0.01 | 29 | 6.9 | 86.2 | 6.9 | 0 | 0 | 0 |
| 0.01-0.05 | 188 | 6.9 | 66.0 | 24.5 | 2.7 | 0 | 0 |
| 0.06-0.10 | 91 | 0 | 62.6 | 30.8 | 6.6 | 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 | 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 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.

The following sections in quotes are from the National Institute for Occupational Safety and Health:[56]

In a study of the records of 1173 hatters, Baldi et al[59] 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
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
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 Act (OSHA) limit mercury (inorganic) exposure to 0.1 milligram of mercury per cubic meter of air as a ceiling value. The 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)
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.

See Appendix V for a complete listing of the mandatory physical examinations and medical and biological monitoring required by OSHA.

# CHAPTER 8

# NITROGEN DIOXIDE

## INTRODUCTION

Nitrogen oxides include nitrous oxide, nitric oxide, nitrogen dioxide, nitrogen trioxide, nitrogen tetroxide, nitrogen pentoxide, nitric acid, and nitrous acid. A thorough discussion of the chemical and toxic properties of each oxide can be found in the NIOSH Criteria Document on the oxides of nitrogen.[60] This chapter is concerned with the chemical agent nitrogen dioxide ($NO_2$) which exists in equilibrium with nitrogen tetroxide ($N_2O_4$) at body temperature.

Exposure to nitrogen dioxide is through inhalation. There is often only mild irritation of the upper respiratory tract, apparently because little of the inhaled gas enters into solution until it reaches the moist alveolar (air cell) spaces of the lungs. Therefore, at the time of exposure, there may be little pain or shortness of breath, and a seriously damaging dose can be delivered to the lungs while a worker is not immediately aware of the danger.

Depending upon the concentration and duration of exposure, toxic reactions to nitrogen dioxide can range from mere mucosal irritation to chemical pneumonitis, acute pulmonary edema (excess fluid in lung tissue), or death. There is a latent period of from 3 to 30 hours from the time of initial exposure to the onset of potentially fatal pulmonary (lung) symptoms.

Chronic exposure to low levels of nitrogen dioxide (5 to 20 ppm) may result in mild irritation to the eyes, nose, and throat, continued pulmonary irritation, coughing, and possible lung damage, especially to the alveolar tissue. Chronic bronchitis and a clinical condition in which the blood's ability to transport oxygen is reduced (methemoglobinemia) may result.

The following is a listing of common names for nitrogen dioxide followed by a listing of occupations with potential exposure to nitrogen dioxide:

### Common Names

dinitrogen tetroxide
liquid dioxide
nitrito
nitro
nitrogen oxide
nitrogen peroxide
nitrogen tetroxide

### Occupations with Potential Exposures to Nitrogen Dioxide

Acid dippers
Aniline makers
Arsenic acid makers
Artificial leather makers
Auto garage workers
Auto painters
Blueprinters
Brass cleaners

Occupations with Potential Exposures to Nitrogen Dioxide (cont.)

Braziers
Bright dip workers
Bronze cleaners
Celluloid makers
Copper cleaners
Cotton bleachers
Dental workers
Diesel equipment operators
Dye makers
Electroplaters
Electric arc welders
Etchers
Explosive makers
Explosive users
Farm workers
Fertilizer makers
Firemen
Flour bleachers
Food bleachers
Gas shrinking operators
Gas welders
Glass blowers
Heat treaters
Jet fuel makers
Jewelrymakers
Lacquer makers
Lithographers
Medical technicians
Metal cleaners
Mine workers
Nitrate workers
Nitric acid workers
Nitrite workers
Nitrogen dioxide workers
Nitrous acid workers
Nurses
Organic chemical synthesizers
Oxalic acid makers
Oxidized cellulose compound makers
Pharmaceutical makers
Phosphoric acid makers
Photoengravers
Phthalic acid makers
Physicians
Picklers
Pipe fitters
Plasma torch operators
Raw silk bleachers
Rocket fuel makers
Silo fillers
Sulfuric acid makers
Textile (rayon) bleachers
Tunnel workers
Welders

MEDICAL EVALUATION AND DIFFERENTIAL DIAGNOSIS

The following should be considered:

- smoking history
- periodic chest X-rays
- periodic pulmonary function tests such as 1 second forced expiratory volume ($FEV_1$) and forced vital capacity (FVC)
- methemoglobin studies

The differential diagnosis includes:

- pneumonia
- acute bronchitis
- X-ray findings may mimic tuberculosis

A respiratory questionnaire, such as that in A Guide to the Work-Relatedness of Disease (NIOSH Publication No. 77-123), Appendix C, can be useful in evaluating the extent and importance of respiratory symptoms such as:

- breathlessness
- sputum production
- chest pain
- cough
- wheezing

Nonoccupational Exposure

It should also be considered that exposure to nitrogen dioxide can be from a home hobby or activity such as:

- automotive and related hobbies
- welding, especially arc
- use of a gas heater or stove
- air pollution (especially in cities with heavy vehicle use)

NOTE: Nitrogen dioxide is contained in both tobacco smoke and smog. Smokers in smoggy areas are likely to be at enhanced risk of developing chronic obstructive pulmonary disease.

Signs and Symptoms

Local

- irritation of eyes
- irritation of mucus membranes of upper respiratory tract
- yellowish or brownish staining of skin and teeth (may indicate nitric acid exposure)

Systemic

Nitrogen dioxide fumes can cause 2 types of upper respiratory tract injuries:

- severe pulmonary irritation, progressing to pulmonary edema, which may occur within 24 hours of prolonged and/or concentrated exposure
- insidious bronchiolar damage, causing life-threatening respiratory tract obstruction in 1 to 4 weeks after only mild to moderate exposure to the toxic gas.

Methemoglobinemia may also occur.

Acute Exposure

- discomfort, uneasiness, or indisposition, often indicative of infection (malaise)
- bluish or grayish discoloration of the skin (cyanosis)
- cough
- expectoration of blood (hemoptysis)
- rapid breathing (tachypnea)
- labored or difficult breathing (dyspnea)
- chills
- fever
- headache
- nausea
- vomiting
- unconsciousness
- collapse and death from respiratory failure
- bronchial irritation, a 5 to 12 hour symptom-free period, followed by sudden onset of acute pulmonary edema may also occur

Nitrogen oxides formed from green silage may produce "silo-filler's disease" and/or bronchiolitis fibrosa obliterans. It may develop within a few days or 6 weeks and is accompanied by:

- fever
- severe and progressive dyspnea
- cyanosis

Silo filler's disease may also progress to chronic pulmonary obstruction.

Chronic Exposure

- pulmonary dysfunction
  - -- decreased vital and breathing capacities
  - -- decreased lung compliance
  - -- increased residual volume

- low arterial oxygen saturation

- dyspnea on exertion

- moist rales and wheezes

- sporadic cough with mucopurulent expectoration (consisting of mucus and pus)

- methemoglobinemia, usually mild and transient. Persons with genetic susceptibility may develop toxic levels of methemoglobin

Laboratory and Clinical Examinations

Additional tests which may assist in the diagnosis are:

- chest X-ray of acute exposure shows diffuse, reticular fine, nodular infiltration, or numerous scattered nodular densities (1 to 5 millimeters in diameter)

- decreased blood pH

- decreased serum proteins

- increased urinary hydroxyproline and acid mucopolysaccharides

EPIDEMIOLOGY

When considering exposure to nitrogen dioxide, both concentration and exposure time must be evaluated. There is sufficient data to conclude that the primary irritant effects of nitrogen dioxide are dose-related.

It should be noted that the acute and usually delayed effects of higher concentrations of nitrogen dioxide are well established but the critical concentration needed to produce either acute pulmonary edema or bronchiolitis fibrosa obliterans is not known. In addition, subacute and chronic responses to low levels of exposure to nitrogen dioxide are not well-established or defined in the human.

Müller[61] reported a study of 7 workers or guests who were exposed to nitrogen dioxide during the final blast which would connect 2 parts of a tunnel under construction. No environmental measurements are available. Two of them were hospitalized shortly after the explosion, and the third was hospitalized 12 days later after exposure during a second explosion. After the acute disease, the most common complaints were bronchitis, cough, sputum, and exertional dyspnea. Seven months after the accident, 4 of the exposed group had a 1 second forced expiratory volume ($FEV_1$) of less than 70%. Within 7 to 14 months after the accident, 5 recovered completely; 2 who suffered from a mild bronchitis previous to the accident had a worsening of symptoms afterwards and were unable to carry out normal duties. The following table from the report presents latency periods and bridging symptoms.

LATENCY PERIODS AND BRIDGING SYMPTOMS FROM
7 CASE STUDIES OF ACCIDENTAL NITROGEN DIOXIDE EXPOSURE

| CASE | LATENT PERIOD | BRIDGING SYMPTOMS |
|---|---|---|
| 1 | 4 weeks | Dull pressure in chest<br>Fatigue, moderate dyspnea<br>Irritating cough<br>Temperature<br>Severe dyspnea<br>24 days hospitalization |
| 2 | none | none<br>(gradual worsening of respiration) |
| 3 | 18 days | Fatigue, dyspnea<br>Irritating cough<br>Temperature<br>Suffocation<br>18 days hospitalization |
| 4 | 4 days | Temperature<br>Bronchitis and cough<br>Moderate dyspnea<br>a few days sick at home |
| 5 | 2-1/2 days | Sanguinolent sputum<br>Feeling of suffocation<br>Acute dyspnea<br>39 days hospitalization |
| 6 | none | none |
| 7 | 14 hrs | Pressure in chest<br>Vomiting<br>Dyspnea<br>1 week sick at home |

(Müller, 1969)

NOTE: Case 2 became gradually short of breath but never was seriously sick. Case 6 had neither a latent period nor bridging symptoms, although he had respiratory symptoms for about 6 months.

The following report of a dose-respone relationship in quotes has been taken from the National Institute for Occupational Safety and Health:[60]

In a study of 70 workers, "aged 26 to 48, exposed for 6 to 8 hours daily for 4 to 6 years in a chemical plant to what was described as oxides of nitrogen, Kosider et al[62] reported concentrations between 0.4 and 2.7 ppm as nitrogen dioxide. Sampling and analysis methods were not reported. A control group of 80 workers of similar ages who were not exposed to nitrogen oxides was selected, and workers smoking more than 10 cigarettes daily were excluded from both groups. Workers exposed to nitrogen dioxide complained of sporadic cough with mucopurulent expectoration and dyspnea on exertion. Fine bubbling rales and 'whistling' sounds were heard in some men, primarily over the lower lungs. There were no chest X-ray abnormalities noted."

Over a period of 18 months, Tse and Bockman[63] observed 4 firemen with acute toxic reactions due to accidental inhalation of nitrogen dioxide which originated from a leak in a chemical plant. Environmental measurements were not made but reports indicated the presence of dense, reddish brown fumes. Pulmonary function data for the 1 fireman who experienced every phase of the illness including the eventual development of chronic pulmonary insufficiency follows:

| PULMONARY FUNCTION DATA | | | | |
|---|---|---|---|---|
| | | Date | | |
| | Predicted Normal | 11/2/66 | 5/9/67 | 2/28/68 |
| Vital Capacity (ml) | 3,943 | 3,000 | 2,750 | 2,650 |
| RV/TLC[a] (3%) | 23 | 45 | 46 | 53 |
| $FEV_1$[b] (% of vital capacity) | 79 | 51 | 61 | 70 |
| $FEV_3$[c] (% of vital capacity) | 95 | 73 | 87 | 100 |
| Maximum Mid-Expiratory Flow (liters/min) | 130-395 | 45 | 60 | 65 |
| Maximal Breathing Capacity (liters/min) | 133 | 62 | 86 | 71 |
| Lung Compliance (liters/cm/$H_2O$) | 0.2 | 0.112 | ..... | ..... |
| Arterial pH | 7.35-7.45 | 7.40 | 7.44 | 7.48 |
| Oxygen Saturation (%) | 99.5 | 94.3 | 94.3 | 86.8 |
| $Pao_2$[d] (mm Hg) | | | | |
| air | 90-100 | 75 | 75 | 55 |
| 100% oxygen | 550 | 560 | 550 | 550 |
| $Paco_2$[e] | | | | |
| air | 40 | 29 | 35 | 35 |
| Diffusing Capacity (DLCO)[f] ml/min/mm Hg | 23.2-39.2 | 30.7 | 32.3 | 24.0 |

(Tse and Bockman, 1970)

a - Residual volume/total lung capacity
b - Forced expiratory volume in 1 second
c - Forced expiratory volume in 3 seconds
d - Arterial oxygen pressure
e - Arterial carbon dioxide pressure
f - Diffusing capacity of the lung for carbon monoxide

All 4 firemen experienced respiratory discomfort of varying degrees with or without abnormal X-ray findings about 4 to 6 weeks after exposure. No correlation between individual response and smoking was made; however, it was noted that the 1 fireman most severely affected had stopped smoking 14 years prior to exposure. The others who smoked about 1/2 package of cigarettes per day at the time of the accident eventually became asymptomatic.

The probable results of excessive single exposures to nitrogen dioxide as determined by the American Industrial Hygiene Association are as follows:[64]

PROBABLE RESULT OF SINGLE EXPOSURE TO NITROGEN DIOXIDE

| Exposure Time (min) | Concentration in Air (ppm) | Expected Effect in Humans |
|---|---|---|
| 5<br>15<br>30<br>60 | 400<br>200<br>150<br>100 | Pulmonary edema and death |
| 5<br>15<br>30<br>60 | 200<br>100<br>75<br>50 | Pulmonary edema with possible subacute or chronic lesions in the lungs |
| 5<br>15<br>30<br>60 | 100<br>50<br>40<br>25 | Respiratory irritation, chest pain |

(Emergency Exposure Limits, AIHA J, 1964)

Lowry and Schuman[65] reported a study of 4 workers who were exposed to nitrogen dioxide after entering a silo or silo chute. Exposure occurred within 48 hours of filling of the silo. Although actual environmental measurements were not available, observed concentrations of nitrogen dioxide obtained during research experiments in agricultural science have ranged from 200 to 4000 ppm. Irritating fumes were noted by all 4 workers who also experienced respiratory symptoms of varying degrees of severity and developed bronchiolitis fibrosa obliterans. Two of the workers died, 1 on the 27th and the other on the 30th day. One of the surviving 2 was hospitalized for about 1 week, the other for 3 weeks. Both were able to resume normal duties but tiny nodular densities throughout both lung fields were still detectable by X-ray 2 months after exposure.

## EVIDENCE OF EXPOSURE

### Sampling and Analysis

The NIOSH approved air sampling method uses a solid sorbent tube (packed column). Two previous methods used are:

1. a commercially available field kit
2. impingement

Direct reading indicator tubes are still in use for spot sampling and analysis.

The NIOSH approved method for air sample analysis uses gas chromatography. Three methods previously used are:

1. alpha-naphthylamine-nitrate spectrophotometry analysis for the field kit method
2. colorimetric intensity measurement
3. phenol-disulfonic method

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

### Allowable Exposure Limits

The Occupational Safety and Health Act (OSHA) has adopted standards that limit exposure to nitrogen dioxide to 5 ppm (or 9 milligrams per cubic meter) of air by volume, based on an 8 hour time-weighted average exposure. (NOTE: A reduction in the standard to 1 ppm as a ceiling value has been proposed by NIOSH to prevent acute irritant effects in the lungs of workers exposed to nitrogen dioxide. In addition, the prevention of repeated acute episodes of irritancy should lessen the risk of developing chronic obstructive lung disease.)

The American Conference of Governmental Industrial Hygienists recommends an exposure limit to nitrogen dioxide of 5 ppm (or 9 milligrams per cubic meter) expressed as a Ceiling Limit which should never be exceeded.

## CONCLUSION

There are no specific tests for diagnosing nitrogen dioxide poisoning. Diagnostic criteria for occupational nitrogen dioxide poisoning are based on meeting the following:

1. confirmed history of occupational exposure to nitrogen dioxide
2. clinical findings as outlined in this guide
3. blood platelet may increase 10 to 100% above normal

4. methemoglobin determination may be helpful

5. carbon dioxide in the blood may be increased

6. X-rays may show chemical pneumonitis or pulmonary edema

7. pulmonary function tests

X-ray findings and lung function test results are of diagnostic value but diagnosis cannot be based on these findings alone.

The clinical findings or effects of inhalation exposure to nitrogen dioxide may simulate other diseases such as pneumonia, acute bronchitis or even cerebral hemorrhage. These entities can usually be excluded by an accurate medical history.

The history of acute nitrogen dioxide exposure is characteristic: initial symptoms subside upon termination of exposure, followed by a sudden onset of pulmonary edema (excess fluid in lung tissue) after a latent period of 3 to 30 hours.

# CHAPTER 9

# CRYSTALLINE SILICA

## INTRODUCTION

The crystalline form of silica, silicon dioxide, is widely distributed in nature and constitutes a major portion of most rocks and their products such as soils and sands. Silica occurs in three principal crystalline forms: quartz, tridymite, and cristobalite. During many industrial operations such as drilling blast holes and grinding stone objects, a dust of silica particles can be formed. Inhalation of these very sharp, insoluble particles into the lungs can produce the disease silicosis which is a form of pneumoconiosis characterized by the formation of small discrete fibrous nodules in the lungs.

The silicosis nodule is composed of circular bundles of collagen (a fibrous insoluble protein) resulting in fibrous nodules measuring 1 to 10 millimeters in diameter. The nodules are found in lymphatics around blood vessels, beneath the pleura (the membrane covering the thoracic cage and lungs), and in groups of lymph nodes within the chest cavity. The upper lobes and hilar lymph nodes are more severely affected than the lung bases. The nodules may fuse to become "progressive massive fibrosis."

Silicosis may be of an acute or chronic nature. Acute silicosis refers to a rapidly-developing lung disease which may occur in workers exposed to high levels of respirable free silica over a relatively short period ranging from a few weeks to 4 or 5 years. Eight to 18 months may lapse from the time of first exposure to the onset of symptoms which include progressive dyspnea (labored or difficult breathing), fever, cough, and weight loss. After development of symptoms, survival time is likely to be short. This disease has been most often reported in manufacturers and packers of abrasive soap powders, in sand-blasters working in enclosed tanks, and in high-power drillers of tunnel rock.

Chronic pulmonary silicosis, the type commonly encountered in industry, is similar to acute silicosis but usually develops after many years of exposure to silica dust and may take many more years to progress. This disease occurs most frequently in the mining industry but is also seen in other industries such as potteries, foundries, stone cutting and finishing, tile and clay producing, and glass manufacturing.

Both acute and chronic silicosis have a definite tendency to progress whether or not the worker remains exposed to dust. Tuberculosis is a common complication of silicosis. Silicosis is also associated with pulmonary hypertension and cor pulmonale (hypertrophy or failure of the right ventricle).

Lung function tests and chest X-rays classed according to the ILO U/C system (international classification of radiographs of the pneumoconiosis) are useful in diagnosing and following the progression of silicosis.

Exposure to crystalline silica can result in the occupational dermatosis, silica granuloma (a granular tumor or growth usually of lymphoid and epitheloid cells).

The common names of some minerals that contain varying amounts of crystalline silica follow:

Common Names

Agate
Amethyst
Beach sand
Chalcedony
Chert
Chrysoprase
Citrine quartz
Cristobalite
Diatomaceous earth
Feldspar
Flint
Free silica
Ganister
Granite
Gritstone
Jasper
Muscovite
Pegmatite
Quartz
Quartzite
Rock crystal
Rose quartz
Sand
Sandstone
Sardonyx
Silica flour
Silican hydride
Tridymite
Tripoli

The following is a list of trade names of products that either consist of or contain silica:

Trade Names

A 175
Acticel
Aerogel 200
Aerogel
Aerosil
Aerosil 175
Aerosil 200
Aerosil 300
Aerosil 380
Aerosil A 175
Aerosil A 300
Aerosil BS-50
Aerosil E 300
Aerosil K 7
Aerosil M-300
Aerosil TT 600
Aerosil-Degussa
Amorphous silica dust
Aquafil
C.I. 77811
C.I. Pigment White 27
Cab-o-Sil
Cab-o-Sil H-5
Cab-o-Sil L-5
Cab-o-Sil MS-7
Cab-o-Sil M-5
Cabosil N 5
Cabosil ST-1
Carplex
Carplex 30
Carplex 80
Celite
Celite Superfloss
Coloidal silica
Colloidal silicon dioxide
Corasil II
Crystallite A 1
Diatomaceous silica
Dicalite
Dri-die
Extrusil
Fossil flour
Gasil
HK 125

Trade Names (cont.)

HK 400
HI-Sil-C
Iatrobeads 6RS8060
KS 160
KS 300
KS 404
Ludox
Ludox HS 40
Manosil VN 3
Milowhite
Min-U-Sil
Minusil 5
Minusil 30
Nalcast P1W
Nalco 1050
Nalfloc
Nalfloc N 1050
Neosil
Neosyl
Nipsil VN 3
OK 412
Porasil
Positive Sol 130M
Positive Sol 232
Pregel
Protek-Sorb
Quso G 30
Quso 51
RD 8
Santocel CS
Santocel 62
Santocel Z
Si-O-Lite
Siflox
Silanox
Silanox 101
Silica ($SiO_2$)
Siliceous earth
Silicic anhydride
Silicon dioxide
Silicon oxide ($SiO_2$)
Silikil
Silikolloid
Siloxid
Sipur 1500
Snowtex
Snowtex 30
Snowtex N
Snowtex O
Snowtex OL
Snowtex C
Super-Cel
Superfloss
Suprasil
Syton 2X
Syton WL
TK 900
Tokusil Gu-N
Tokusil TPLM
U 333
Ultrasil VN 3
Ultrasil VH 3
Ultrasil VN 2
Verticurine
Vitasil 220
Vulcasil S
Wessalon S
White carbon
Zeofree 80
Zipax
Zorbax SIL

Occupations with Potential Exposures to Crystalline Silica

Abrasive blasters
Abrasives makers
Agriculture
Auto garage workers
Bisque-kiln workers
Brick layers
Brickmakers
Buffers
Buhrstone workers
Carborundum makers
Casting cleaners, foundry
Cement makers
Cement mixers
Ceramic workers
Chemical glass makers
Chippers
Coal miners
Construction workers
Cosmetics makers
Cutlery makers
Diatomaceous earth calciners
Electronic equipment makers
Enamellers
Farming

Occupations with Potential Exposures to Crystalline Silica (cont.)

Fertilizer makers
Fettlers
Flint workers
Foundry workers
Furnace liners
Fused quartz workers
Glass makers
Glaze mixers, pottery
Granite cutters
Granite workers
Grinding wheel makers
Grindstone workers
Hard rock miners
Insecticide makers
Insulators
Jewelers
Jute workers
Kiln liners
Lithographers
Masons
Metal buffers
Metal burnishers
Metal polishers
Miners
Mortar makers
Motormen
Oil purifiers
Oilstone workers
Optical equipment makers
Paint mixers
Polishing soap makers
Porcelain workers
Pottery workers
Pouncers, felt hat
Pulpstone workers
Quarry workers
Quartz workers
Refractory makers
Road constructors
Rock crushers
Rock cutters
Rock drillers
Rock grinders
Rock screeners
Rubber compound mixers
Sand cutters
Sand pulverizers
Sandblasters
Sandpaper makers
Sandstone grinders
Sawyers
Scouring soap workers
Silica brick workers
Silicon alloy makers
Silver polishers
Slate workers
Smelters
Sodium silicate makers
Spacecraft workers
Stone bedrubbers
Stone cutters
Stone planers
Street sweepers
Subway construction workers
Tile makers
Tooth paste makers
Tube mill liners
Tumbling barrel workers
Tunnel construction workers
Whetstone workers
Wood filler workers

MEDICAL EVALUATION AND DIFFERENTIAL DIAGNOSIS

The following should be considered:

- other pneumoconioses
- sarcoidosis (a chronic granulomatous disease)
- tuberculosis
- fibrosing alveolitis (hardening of lung tissue)
- carcinomatous lymphangitis (spread of cancer via lymph channels)
- pulmonary hemosiderosis (iron deposits in lung tissue)

A respiratory questionnaire, such as that in A Guide to the Work-Relatedness of Disease (NIOSH Publication No. 77-123) Appendix C, can be useful in evaluating the extent and importance of respiratory symptoms.

Nonoccupational Exposure

Potential nonoccupational sources of silica dust include:

- ceramics, pottery and related hobbies
- work with plasters, mortars, or cements having a high silica content
- rock working hobbies (carving, cutting, chiseling)

Signs and Symptoms - Simple Silicosis

Simple silicosis may be nonspecific early in the course of the illness and have little effect on ventilatory capacity. Generally, the only finding is nodulation of the lungs as seen on a chest X-ray. Approximately 20 to 30% of the persons with simple silicosis go on to develop complicated silicosis despite removal from a silica environment.

Signs and Symptoms - Complicated Silicosis

- cough and sputum (productive cough)
- labored or difficult breathing (dyspnea)
- wheezes (rhonchi)
- crackling sound (crepitations), on examination of the lungs
- chest pain
- bluish or grayish discoloration of the skin (cyanosis)
- decreased pulmonary function
- chest X-ray shows nodulation of the lungs

Progression of involvement is related to continued exposure, increasing age, smoking, and pulmonary infections. Severe pulmonary fibrosis may occur in 20 to 30% of workers who develop silicosis. In advanced cases, the following may occur:

- chronic bronchitis
- obstructive pulmonary disease (emphysema)
- cardiac failure may occur
- death may occur from respiratory failure

Silicosis may also co-exist with tuberculosis. It should also be noted that silicosis favors the growth of the tubercle bacillii. However, silicosis may suppress the usual features of epitheloid cell proliferation, giant cell formation, and lymphocytic reaction.

Laboratory and Clinical Examinations

Additional tests that will assist in arriving at a correct diagnosis are:

Pulmonary Function

- reduced forced vital capacity (FVC)
- decreased 1 second forced expiratory volume ($FEV_1$)
- reduced diffusing capacity
- reduced maximal breathing capacity

NOTE: These test results indicate impairment of lung function; there are no lung function tests which specifically assay for silica.

Pulmonary impairment including oxygen desaturation on exercise progresses rapidly in complicated silicosis. Associated chronic bronchitis may be a key factor in the decreased pulmonary function.

Chest X-Ray

The following classifications are used in charting the possible progression of silicosis:

- simple silicosis: multiple opacities of various sizes and densities (from less than 1.5 to 10 millimeters) may be diffused over the entire lung field; the opacities may be calcified. Hilar nodes may develop "egg shell" calcification.
- complicated silicosis: conglomerate masses are greater than 1 centimeter in diameter and are usually found in upper and middle zone. Large sausage-shaped masses which may be surrounded by emphysematous bullae may appear in advanced cases.
- Caplan's syndrome: occurs when larger nodules appear against the background of simple silicosis; rheumatoid disease may be associated.

Radiographs should be classified by the ILO U/C scheme.

NOTE: Because silicosis shares the X-ray appearance of at least 20 other chest diseases, X-ray findings alone cannot be the basis of diagnosis. However, most worker's compensation insurance acts use X-ray criteria for compensation purposes.

Acute, high exposure can result in death without any X-ray evidence of silicosis. It should also be noted that nodular densities can be induced by silica, iron, tin, and barium without associated fibrosis.

EPIDEMIOLOGY

The relationship between crystalline silica and respiratory impairment, including silicosis, has been demonstrated in various epidemiological studies. The available information indicates that 1 or more of the following factors may have important etiologic significance in the development of lung disease: the particle size of the crystalline silica dust, the concentration of the free crystalline silica, possible synergistic action of other ions present, differences in individual susceptibility, and the presence of a concomitant infection (especially tuberculosis). This should be taken into consideration when interpreting the following studies:

Musk et al[66] reported a 4 year study of 688 granite shed workers who were exposed to mean silica dust concentrations less than the threshold limit value of 100 micrograms per cubic meter for respirable free silica. Excessive average yearly decrements in pulmonary function were observed: 75 to 84 milliliters per year for forced vital capacity (FVC) and 53 to 67 milliliters per year for 1 second forced expiratory volume ($FEV_1$). Observed decrements were independent of exposure group (i.e. cutter, sculptor, polisher, sandblast area worker, etc.) and could not be accounted for by cigarette smoking. In 528 additional granite shed workers, decrements in ventilatory capacity were measured for 1, 2, or 3 years and were of the same order of magnitude.

Prospective studies of lung function in working populations and in the general population have shown that $FEV_1$ and FVC in healthy mean decrease at a rate of less than 40 milliliters per year after the age of 25 years with a greater rate of decline for cigarette smokers than for those who have never smoked. Subjects with chronic obstructive pulmonary disease exhibit a rate of decline of $FEV_1$ and FVC of approximately 80 milliliters per year.[66]

In a study of 727 mine workers from a representative group of metal mines, Dreessen et al[67] reported an incidence of silicosis in 25% of the workers exposed for more than 6 years to silica dust concentrations of 10 to 23 million particles per cubic foot (mppcf) having a free silica content of 20 to 40%. No cases of silicosis were observed in workers whose exposures did not exceed an average of 18 mppcf and whose employment exposure did not exceed 10 years. The severity of pulmonary fibrosis among cases of silicosis increased greatly with increasing length of employment.

Flinn et al[68] reported a study of 2516 workers who manufactured pottery products in 9 potteries. Workers were exposed to dust containing from 1 to 39% quartz and having an average particle diameter of 1.2 micrometers. The following table summarizing data from the report is taken from the NIOSH Criteria Document on crystalline silica:[69]

RELATION OF DUST CONCENTRATION AND LENGTH OF EMPLOYMENT IN THE POTTERY INDUSTRY TO SILICOSIS*

| Dust Concentration (million particles per cubic foot) | Years in Pottery Industry | | | | |
|---|---|---|---|---|---|
| | 0-9 | 10-19 | 20-29 | 30-39 | Over 40 |
| 0-3.9: | | | | | |
| Cases of silicosis | - | 1 | 1 | - | - |
| Workers exposed | 481 | 223 | 65 | 21 | 8 |
| Percentage | 0 | 0.4 | 1.5 | 0 | 0 |
| 4-7.9: | | | | | |
| Cases of silicosis | 1 | 6 | 26 | 27 | 29 |
| Workers exposed | 321 | 198 | 110 | 53 | 34 |
| Percentage | 0.3 | 3 | 24 | 51 | 85 |
| 8-15.9: | | | | | |
| Cases of silicosis | - | 8 | 5 | 10 | 10 |
| Workers exposed | 176 | 119 | 25 | 17 | 14 |
| Percentage | 0 | 7 | 20 | 59 | 71 |
| Over 16: | | | | | |
| Cases of silicosis | 13 | 33 | 10 | 5 | 4 |
| Workers exposed | 363 | 174 | 21 | 7 | 5 |
| Percentage | 4 | 19 | 48 | 71 | 80 |

(Flinn et al, 1939 and NIOSH, 1974)

*Includes 1st, 2nd, and 3rd stage cases.

Flinn et al suggested that new cases of silicosis would not develop if the dust concentration in potteries could be brought below 4 mppcf.

Rajhans and Budlovsky[70] reported a study of 1166 production workers in 10 brick and tile plants in Ontario in which no cases of silicosis were found. Workers had been exposed for 1 to 30 years to mean work place dust concentrations ranging from 12 to 1026 mppcf. Average respirable dust concentrations ranged from 1.05 to 4.26 milligrams per cubic meter and had a free silica content of approximately 13%. Rajhan and Budlovsky suggested that progression of the silicotic process was inhibited by the 14% alumina content in the clays and tiles used to manufacture brick and tile. In an earlier study in 3 British brick plants, Keatinge and Potter[71] reported similar findings and concluded that excessive occupational hazards were not associated with brick making.

Theriault et al[72,73,74] reported a comprehensive study of approximately 800 workers from 13 occupational groups in 49 granite sheds. Granite dust and quartz were reported to cause significant decreases in FVC, $FEV_1$, and total lung volume but not in residual volume. Of 784 workers, 233 had X-rays classed as abnormal which showed opacities compatible with pneumoconiosis.[75] Workers with abnormal X-rays had been exposed to an average of 2.3 times more dust than workers having normal X-rays. Theriault et al concluded that pulmonary function measurements are more sensitive indicators of the effect of granite dust than chest roentgenograms.

In a 1 year study of 869 workers in 5 diatomite plants, Cooper and Cralley[76] suggested that nearly all presumptive abnormal chest roentgenograms found in 156 workers were associated with exposure to calcined diatomite containing 15 to 61% cristobalite. The extent and severity of pneumoconiosis also appeared to correlate with length of exposure. For all plant operations, airborne dust concentrations ranged from 1 to 66 mppcf, and the median particle size was 1.1 micrometers.

## EVIDENCE OF EXPOSURE

### Sampling and Analysis

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

1. impingement
2. cascade impactor
3. electrostatic precipitator

The NIOSH approved methods for samples analysis are:

1. gravimetric plus X-ray diffraction
2. gravimetric plus colorimetric analysis
3. gravimetric plus infrared spectrophotometry

Three methods previously used are:

1. electron microscopic
2. exo-electron emission
3. differential thermoanalysis

The above methods are not intended to be exclusive, but other methods should be justified.

Allowable Limits

Standards adapted by the Occupational Safety and Health Administration (OSHA) have recommended that limits for dusts containing greater than one percent of silicon dioxide ($SiO_2$) are to be calculated from the following formula:

quartz (respirable mass fraction)

(microscopic counting)

$$\frac{250^*}{\%\ SiO_2 + 5}$$ million particles per cubic foot

quartz (respirable mass fraction)

(gravimetric analysis)

$$\frac{10^{**}}{\%\ SiO_2 + 2}$$ milligrams per cubic meter

quartz (total dust)

(gravimetric analysis)

$$\frac{30}{\%\ SiO_2 + 2}$$ milligrams per cubic meter

cristobalite

1/2 the value calculated from the mass or count formula for quartz

tridymite

1/2 the value calculated from the mass or count formula for quartz

*The percent of crystalline silica in the formula is the amount determined from airborne samples except in those instances in which other methods have been shown to be applicable.

**Both concentration and percent quartz for the application of this limit are to be determined from the fraction passing a size selector with the following characteristics.

| AERODYNAMIC DIAMETER (um) (unit density sphere) | Percent Passing Selector |
|---|---|
| 2.0 | 90 |
| 2.5 | 75 |
| 3.5 | 50 |
| 5.0 | 25 |
| 10.0 | 0 |

(NOTE: NIOSH has recommended a reduction in the standard to 50 micrograms per cubic meter as respirable free silica based on an 8 hour time-weighted average exposure.)

CONCLUSION

Diagnostic criteria for occupational silicosis are based on meeting the following:

1. confirmed history of occupational exposure to free silica of:
   a. a particle size capable of producing the disease (pathologic)
   b. sufficient intensity of exposure
   c. sufficient duration of exposures
2. X-ray findings as outlined above (in accordance with ILO U/C International Classification of Radiographs of Pneumoconiosis 1971)
3. clinical findings compatible with silicosis as outlined above
4. lung function test results that are indicative of respiratory dysfunction associated with the formation of fibrous tissue within the tissue spaces (interstitial fibrosis)

Lung biopsy and lung sections collected after death remain the only unequivocal methods of making a definitive diagnosis.

NOTE: A complete listing of the mandatory physical examinations and medical and biological monitoring required by OSHA is in Appendix V.

# CHAPTER 10

# SULFUR DIOXIDE

## INTRODUCTION

Sulfur dioxide, a colorless gas at room temperature with a distinctive, irritating odor, can also exist as a liquid and is soluble in water and organic solvents. It is produced in the smelting of sulfide ores and in the processing of sulfur-containing fuels. In large cities and areas surrounding smelters and oil refineries, sulfur dioxide is a major contributor to atmospheric pollution.

Because sulfur dioxide is very soluble, it mainly affects the upper respiratory tract: nose, throat, trachea (windpipe), and bronchi. These tissues may swell and block the passage of air. After acute exposure, the alveoli (air sacs) are also injured, and pulmonary edema (filling of the lungs with fluid) can result, which may be fatal.

The average individual is able to detect 0.3 to 1 part per million (ppm) mainly by taste, 3 ppm by odor, and 6 ppm by immediate sharp irritation of the nose and throat. Concentrations of 20 ppm can cause an immediate irritation to the eyes (Daum and Stellman, 1973).

Severe acute gassing accidents are rare because sulfur dioxide is so intensely irritating that workers run for their lives to escape from its effects. Workers in atmospheres fairly heavily contaminated by sulfur dioxide do acquire a degree of tolerance.

The long-term effects of low concentrations of sulfur dioxide are not known though nasopharyngitis (chronic irritation of the nose and throat), changes in the senses of taste and smell, and increased fatigue have been documented. Chronic irritation of the trachea due to exposure to sulfur dioxide may cause chronic bronchitis and emphysema.

Eye injury varies according to whether the gaseous or liquid form of sulfur dioxide is involved. When only the gas is employed, as in magnesium foundries, ocular reactions are mild probably due to the warning characteristics of the gas which enable the worker to avoid excessive exposure. Even in acute gaseous exposures, severe enough to almost be fatal to the worker, the severe conjunctivitis (inflammation of the membrane that lines the eyelids and the front of the eyeball) that occurs recovers completely and leaves no ocular damage.

The accidental spraying of liquefied sulfur dioxide into the eyes of workers on refrigeration machines may cause permanent reduction of visual acuity (sharpness of vision) from its clouding effect on the cornea. Blindness can result.

Inhaled sulfur dioxide may cause thiamine deficiency-like symptoms. In women, menstrual disorders may be observed.

The following is a listing of common names for sulfur dioxide followed by a listing of occupations with potential exposure to sulfur dioxide:

Common Names

fermenticide liquid
sulfer oxide
sulfur oxide
sulfurous acid anhydride
sulfurous anhydride
sulfurous oxide
sulphur dioxide

Occupations with Potential Exposures to Sulfur Dioxide

Alkali-salt makers
Automotive workers
Beet sugar bleachers
Blast furnace workers
Boiler water treaters
Bone extractors
Brewery workers
Brickmakers
Broommakers
Carbolic acid makers
Cellulose makers
Coke oven workers
Copper smelters
Diesel engine operators
Diesel engine repairmen
Disinfectant makers
Disinfectors
Dye makers
Exterminators
Feather workers
Fertilizer makers
Firemen
Flour bleachers
Flue cleaners
Food bleachers
Foundry workers
Fruit preservers
Fumigant makers
Fumigators
Furnace operators
Galvanizers
Gelatin bleachers
Glass makers
Glue bleachers
Grain bleachers
Heat treaters (magnesium)
Ice makers
Insecticide makers
Lead smelters
Magnesium foundry workers
Meat preservers
Mercury smelters
Metal refiners
Oil bleachers
Oil processors
Ore smelter workers
Organic sulfonate makers
Paper makers
Petroleum refinery workers
Pottery workers
Preservative makers
Protein makers (edible)
Protein makers (industrial)
Pyrites burners
Refrigeration workers
Sodium sulfite makers
Storage battery chargers
Straw bleachers
Sugar refiners
Sulfite makers
Sulfur dioxide workers
Sulfurers (malt and hops)
Sulfuric acid makers
Tannery workers
Textile bleachers
Thermometer makers (vapor)
Thionyl chloride makers
Tunnel workers
Vegetable preservers
Vulcanizers
Wicker ware bleachers
Wine makers
Wood bleachers
Wood pulp bleachers
Zinc smelters

## MEDICAL EVALUATION AND DIFFERENTIAL DIAGNOSIS

The following should be considered:

- any history of past disease of the eye or the cardiopulmonary system (of the heart and/or lungs) should be carefully evaluated to determine if present symptoms are, in fact, associated with a previous disease or injury.

- a respiratory questionnaire, such as that in A Guide to the Work-Relatedness of Disease (NIOSH Publication No. 77-123), Appendix C, can be useful in evaluating the extent and importance of respiratory symptoms, such as:

  -- breathlessness

  -- sputum production

  -- chest pain

  -- cough

  -- wheezing

### Nonoccupational Exposure

Exposure to sulfur dioxide may be from:

- air pollution

- hobbies involved with auto mechanics and exposure to exhaust gases from cars equipped with catalytic converters

- working as a volunteer fireman

### Signs and Symptoms

#### Acute Exposure

- irritation of nose and throat

- burning sensation in eyes

- secretion and discharge of tears (lacrimation)

- mucus flows from nose (rhinorrhea)

- cough

- choking sensation

- sneezing

- bronchoconstriction (reflex type)

- increased bronchial secretion
- increased pulmonary resistance
- rales, high pitched type
- prolonged expiratory phase
- bronchial asthma

In severe exposure, the above progresses to:

- chemical bronchopneumonia (inflammation of the terminal bronchioles and alveoli)
- bronchiolitis obliterans (irritation of the bronchioles that results in their closure)

Hypersensitive individuals will develop urticarial skin eruption (characterized by pale evanescent wheals or hives associated with severe itching) and swelling of the eyelids.

Signs and symptoms that liquid sulfur dioxide can cause in the eye are:

- corneal burns (may be painless)
- corneal opacification which may result in partial or complete loss of vision depending upon the severity of exposure

Chronic Exposure

Symptoms which will be experienced initially include:

- upper respiratory tract irritation
- cough
- nose bleeds (epistaxis)
- chest tightness
- expectoration of blood (hemoptysis)

After customary or continued exposure, the following can be observed:

- hacking cough
- morning cough
- nasal irritation
- nasal discharge
- expectoration

- chronic irritation of the nose and throat (nasopharyngitis)
- alteration in senses of smell and taste
- increased sensitivity to other irritants
- fatigue
- labored or difficult breathing (dyspnea) on exertion
- prolongation of common colds

Laboratory and Clinical Examinations

Additional tests that will assist in arriving at a correct diagnosis are:

Urine

- increased acidity due to increased excretion of sulfate

Pulmonary Function

- increased airway resistance
- decreased maximum expiration flow
- decreased 1 second forced expiratory volume ($FEV_1$)
- decreased forced vital capacity (FVC)
- decreased specific airway conduction
- increase in respiratory and pulse rate
- decreased tidal volume

Chest X-ray

- may show reticulation, nodulation, and enlarged hilar shadows after long-term exposure
- X-ray findings compatible with bronchiectasis, pulmonary edema, emphysema, bronchiolitis obliterans, asthma

An additional test result which will assist in arriving at a correct diagnosis is:

- inhibition of thyroid function

EPIDEMIOLOGY

Studies of workers exposed to sulfur dioxide in their work environment have suggested association with chronic nonspecific pulmonary disease. However, no quantitative exposure-effect relationships have been derived

from the published reports of occupational exposure, and mixed exposures have been the general rule.[77] This should be taken into consideration when evaluating the following material:

Smith et al[78] reported a study of 113 copper smelter workers who were exposed to concentrations of sulfur dioxide ranging from 1.6 to 45 ppm with the highest concentrations occurring close to the production source. Combination dust and gas masks were used intermittently when a worker experienced or expected irritation. Over the 2 year study period, the workers showed an excessive loss of pulmonary function averaging 74.5 milliliter loss of forced vital capacity (FVC) and 84.0 milliliter loss of 1 second forced expiratory volume ($FEV_1$) per year. Workers with $FEV_1$ below normal on initial measurements (based on their age and height) showed evidence of even greater loss of pulmonary function related to sulfur dioxide exposure. It was concluded that sulfur dioxide exposures greater than 1 ppm are associated with an accelerated loss of pulmonary function that could lead to chronic pulmonary disease if high exposures were continued for a sufficient period of time.

Kehoe et al[79] reported a study of the effect of prolonged exposure to sulfur dioxide on 100 workers who manufactured electric refrigerators. At the time of the study, atmospheric concentrations of sulfur dioxide averaged from 20 to 30 ppm with a range of 5 to 70 ppm. (5 years before the study, concentrations averaged 80 to 100 ppm.) Average length of employment exposure was 3.8 years, and 47 workers had 4 to 12 years employment exposure. A control group of 100 men, age-matched with the exposed group, was selected from parts of the same plant where there was no known exposure to sulfur dioxide or other known noxious gases, fumes, or dust. An incidence of slight chronic nasopharyngitis significantly higher than normal was found in exposed workers, and many of these workers suffered partial loss of sense of taste and smell. The susceptibility to ordinary colds was no higher than normal but their average duration was 2 to 3 times longer than the average for the control group. Other significant differences between the 2 groups were dyspnea on exertion and increased fatigue from work.

Skalpe[80] reported a study of 54 workers at 4 different pulp mills that was initiated by the observation that pulp mill workers very often complained of chronic cough. The workers were exposed to concentrations of sulfur dioxide ranging from 2 to 36 ppm but were reported to occasionally have much heavier exposure due to special procedures than was indicated by the analysis. The control group, 56 unexposed workers from the same industry and district, had no significant differences in age or in frequency of smokers. A significantly higher frequency of cough, expectoration, and dyspnea on exertion was found in the exposed group with the difference being greatest in age groups under 50 years. The average maximal expiratory flow rate was significantly lower (the difference in means, 42 liters per minute, was twice the standard error) in the exposed group than in the control group in the age groups under 50; there was no difference in values in the age group over 50. Vital capacity values showed no significant difference between the groups.

Skalpe stated that the probable explanation for the high frequency of respiratory disease symptoms in the age group under 50 was because respiratory disease is rare in this age group. Therefore, the effect of small external insults would be easier to detect than in the older age group where respiratory disease from other causes is more common, so that a small addition would be less noticeable.

In a mortality study of 8047 copper smelter workers exposed to arsenic trioxide and sulfur dioxide, Lee and Fraumeni[81] hypothesized that an interaction between exposure to high levels of arsenic trioxide and to sulfur dioxide (or other unidentified chemicals in the work environment) may be responsible for the excessive number of respiratory cancer deaths among smelter workers.

## EVIDENCE OF EXPOSURE

### Sampling and Analysis

The NIOSH approved air sampling method uses mechanical filtration in series with impingement. Two previous methods used are:

1. continuous automatic reading instruments
2. a series of two scrubbers (impingers)

Direct reading detector tubes are still in use for spot sampling and analysis.

The NIOSH approved method for air sample analysis is titration using an indicator to determine the end point.

Previous impingement sample analysis methods also used titration plus an indicator.

These methods for sampling and analysis are not intended to be exclusive. However, it is recommended that other methods be justified.

### Allowable Exposure Limits

The Occupational Safety and Health Act (OSHA) has recommended limiting exposure to sulfur dioxide to 5 ppm of air by volume based on an 8 hour time-weighted average exposure. (NOTE: NIOSH has proposed a reduction in the standard to 2 ppm based on an 8 hour time-weighted average exposure. At this level, workers are not expected to be adversely affected.)

## CONCLUSION

It is difficult to attribute observed symptoms specifically to sulfur dioxide exposure since it is frequently associated with other atmospheric contaminants in industry.

Diagnosis of occupational disease due to sulfur dioxide exposure rests on meeting the following composite pictures:

1. confirmed history of occupational exposure to sulfur dioxide
2. clinical findings comparable to those outlined above
3. lung function test results indicating lung impairment
4. increased urinary sulfate ion concentration is not diagnostic but may indicate degree of exposure

NOTE: See Appendix V for a complete listing of the mandatory physical examinations and medical and biological monitoring required by OSHA.

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APPENDIX III
JOBS AND POTENTIAL EXPOSURES

| OCCUPATION | AGENT(S) |
|---|---|
| Abrasive blasters | Silica |
| Abrasive makers | Silica |
| Acetylene workers | Arsenic |
| Acid dippers | Arsenic, nitrogen dioxide |
| Acid finishers | Lead |
| Actors | Lead |
| Adhesive makers | Benzene |
| Agriculture | Lead, silica |
| Airplane dope makers | Benzene |
| Alcohol workers | Benzene |
| Alkali-salt makers | Sulfur dioxide |
| Alloy makers | Arsenic |
| Amalgam makers | Mercury |
| Aniline color makers | Arsenic |
| Aniline makers | Benzene, nitrogen dioxide |
| Aniline workers | Arsenic |
| Antimony ore smelters | Antimony |
| Antimony workers | Antimony |
| Arsenic acid makers | Arsenic, nitrogen dioxide |
| Arsenic workers | Arsenic |
| Art glass workers | Benzene |
| Artificial flower makers | Arsenic |
| Artificial leather makers | Benzene, nitrogen dioxide |
| Artists, commercial | Lead, mercury |
| Asbestos product impregnators | Benzene |
| Asphalt mixers | Benzene |
| Auto body shop workers | Lead |
| Auto garage workers | Silica, nitrogen dioxide |
| Auto painters | Nitrogen dioxide |
| Automotive workers | Benzene, sulfur dioxide |
| Babbitters | Lead |
| Babbitt metal workers | Antimony, arsenic |
| Bactericide makers | Mercury |
| Barometer makers | Mercury |
| Battery makers | Lead, mercury |
| Battery makers, dry | Benzene |
| Battery workers, storage | Antimony |
| Beaming operators, cotton mill | Cotton dust |
| Beet sugar bleachers | Sulfur dioxide |
| Belt scourers | Benzene |
| Benzene hexa-chloride makers | Benzene |
| Benzene workers | Benzene |

| OCCUPATION | AGENT(S) |
|---|---|
| Bisque-kiln workers | Silica |
| Blacksmiths | Lead |
| Blast furnace workers | Sulfur dioxide |
| Bleaching powder makers | Arsenic |
| Blueprinters | Nitrogen dioxide |
| Boiler makers | Mercury |
| Boiler operators | Arsenic |
| Boiler water treaters | Sulfur dioxide |
| Bone extractors | Sulfur dioxide |
| Bookbinders | Arsenic, lead |
| Bottle cap makers | Lead |
| Brakelining makers | Benzene |
| Brass cleaners | Nitrogen dioxide |
| Brass founders | Antimony |
| Brass makers | Arsenic |
| Brass polishers | Lead |
| Braziers | Lead, nitrogen dioxide |
| Brewery workers | Sulfur dioxide |
| Brick layers | Lead, silica |
| Brick makers | Lead, silica, sulfur dioxide |
| Bright dip workers | Nitrogen dioxide |
| Britannia metal workers | Antimony |
| Bronze cleaners | Nitrogen dioxide |
| Bronze makers | Arsenic |
| Bronzers | Antimony, arsenic, benzene, lead, mercury |
| Broommakers | Sulfur dioxide |
| Brush makers | Lead |
| Buffers | Silica |
| Buhrstone workers | Silica |
| Burnishers | Antimony, benzene |
| Cable makers | Lead |
| Cable splicers | Antimony, lead |
| Cadmium workers | Arsenic |
| Calibration instrument makers | Mercury |
| Can makers | Benzene |
| Candle makers | Arsenic |
| Canners | Arsenic, lead |
| Cap loaders, percussion | Mercury |
| Carbolic acid makers | Benzene, sulfur dioxide |
| Carbon brush makers | Mercury |
| Carborundum makers | Silica |
| Carders, cotton mill | Cotton dust |
| Carding machine operators, cotton mill | Cotton dust |

| OCCUPATION | AGENT(S) |
|---|---|
| Carpet makers | Arsenic |
| Carroters, felt hat | Arsenic |
| Cartridge makers | Lead |
| Cast scrubbers, electroplating | Benzene |
| Casting cleaners, foundry | Silica |
| Cattle dip workers | Arsenic |
| Caustic soda makers | Mercury |
| Celluloid makers | Nitrogen dioxide |
| Cellulose makers | Sulfur dioxide |
| Cement makers | Silica |
| Cement mixers | Silica |
| Ceramic enamel workers | Arsenic |
| Ceramic makers | Antimony, arsenic, lead |
| Ceramic workers | Silica, mercury |
| Chemical equipment makers | Lead |
| Chemical glass makers | Silica |
| Chemical synthesis | Benzene |
| Chippers | Lead, silica |
| Chlorinated paraffin makers | Lead |
| Chlorine makers | Mercury |
| Chlorobenzene makers | Benzene |
| Chlorodiphenyl makers | Benzene |
| Cigar makers | Lead |
| Cinnebar ore processors | Mercury |
| Cleaner operators, cotton mill | Cotton dust |
| Cleaners, cotton mill | Cotton dust |
| Clutch disc impregnators | Benzene |
| Coal miners | Silica |
| Coal tar refiners | Benzene |
| Coal tar workers | Benzene |
| Cobblers | Benzene |
| Coke oven door cleaners, luterman | Coke oven emissions |
| Coke oven door machine operators | Coke oven emissions |
| Coke oven heater | Coke oven emissions |
| Coke oven larry car operators | Coke oven emissions |
| Coke oven lidmen, larrymen | Coke oven emissions |
| Coke oven maintenance men | Coke oven emissions |
| Coke oven patcher | Coke oven emissions |
| Coke oven pusher operators | Coke oven emissions |
| Coke oven quench car operators | Coke oven emissions |

| OCCUPATION | AGENT(S) |
|---|---|
| Coke oven tar chaser | Coke oven emissions |
| Coke oven workers | Benzene, coke oven emissions, sulfur dioxide |
| Combing machine operators, cotton mill | Cotton dust |
| Compositors | Antimony |
| Construction workers | Silica |
| Copper cleaners | Nitrogen dioxide |
| Copper refiners | Antimony |
| Copper smelters | Arsenic, sulfur dioxide |
| Cosmetics makers | Silica |
| Cotton bleachers | Nitrogen dioxide |
| Crop dusters | Arsenic, lead |
| Cutlery makers | Lead, silica |
| Cyclohexane makers | Benzene |
| DDT makers | Benzene |
| Decorators, pottery | Lead |
| Defoliant applicators | Arsenic |
| Defoliant makers | Arsenic |
| Degreasers | Benzene |
| Demolition workers | Lead |
| Dental amalgam makers | Mercury |
| Dental technicians | Lead |
| Dental workers | Nitrogen dioxide |
| Dentists | Mercury |
| Detergent makers | Benzene |
| Diamond polishers | Lead |
| Diatomaceous earth calciners | Silica |
| Dichlorobenzene makers | Benzene |
| Diesel equipment operators | Nitrogen dioxide |
| Diesel engine operators | Sulfur dioxide |
| Diesel engine repairmen | Sulfur dioxide |
| Dimethylsulfate makers | Arsenic |
| Diphenyl makers | Benzene |
| Direct current meter workers | Mercury |
| Disinfectant makers | Arsenic, benzene, mercury, sulfur dioxide |
| Disinfectors | Mercury, sulfur dioxide |
| Drawing frame operators, cotton mill | Cotton dust |
| Drug makers | Arsenic, benzene, mercury |
| Dry cleaners | Benzene |
| Dryer operators, cotton mill | Cotton dust |

JOBS AND POTENTIAL EXPOSURES

| OCCUPATION | AGENT(S) |
|---|---|
| Dye makers | Antimony, arsenic, benzene, lead, mercury, nitrogen dioxide, sulfur dioxide |
| Dyers | Lead |
| Electric apparatus makers | Mercury |
| Electric arc welders | Nitrogen dioxide |
| Electrolytic copper workers | Arsenic |
| Electronic device makers | Lead |
| Electronic equipment makers | Silica |
| Electroplaters | Antimony, arsenic, benzene, lead, mercury, nitrogen dioxide |
| Electrotypers | Lead |
| Embalmers | Mercury |
| Embroidery workers | Lead |
| Emery wheel makers | Lead, silica |
| Enamel burners | Lead |
| Enamel makers | Arsenic, lead |
| Enamelers | Arsenic, benzene, lead, silica |
| Engravers | Benzene |
| Etchers | Arsenic, nitrogen dioxide |
| Ethylbenzene makers | Benzene |
| Explosive makers | Antimony, benzene, lead, mercury, nitrogen dioxide |
| Explosives users | Nitrogen dioxide |
| Exterminators | Arsenic, sulfur dioxide |
| Farm workers | Arsenic, lead, mercury, nitrogen dioxide, silica |
| Farmers | Arsenic, lead, mercury, nitrogen dioxide, silica |
| Feather workers | Arsenic, benzene, sulfur dioxide |
| Feltmakers | Mercury |
| Ferrosilicon workers | Arsenic |
| Fertilizer makers | Arsenic, nitrogen dioxide, silica, sulfur dioxide |
| Fettlers | Silica |
| File cutters | Lead |
| Fingerprint detectors | Mercury |
| Firemen | Lead, nitrogen dioxide, sulfur dioxide |
| Fireworks makers | Antimony, arsenic, mercury |
| Flameproofers | Antimony |
| Flint workers | Silica |
| Flour bleachers | Nitrogen dioxide, sulfur dioxide |

| OCCUPATION | AGENT(S) |
|---|---|
| Flower makers, artificial | Lead |
| Flue cleaners | Sulfur dioxide |
| Flypaper makers | Arsenic |
| Food bleachers | Nitrogen dioxide, sulfur dioxide |
| Foundry workers | Antimony, lead, silica, sulfur dioxide |
| Fruit preservers | Sulfur dioxide |
| Fuel oil handlers | Benzene |
| Fumigant makers | Benzene, sulfur dioxide |
| Fumigators | Sulfur dioxide |
| Fungicide makers | Benzene, mercury |
| Fur preservers | Mercury |
| Fur processors | Mercury |
| Furnace liners | Silica |
| Furnace operators | Sulfur dioxide |
| Furniture finishers | Benzene |
| Fused quartz workers | Silica |
| Galvanizers | Arsenic, lead, sulfur dioxide |
| Garage mechanics | Lead |
| Gas shrinking operators | Nitrogen dioxide |
| Gas welders | Nitrogen dioxide |
| Gas workers, illumination | Benzene |
| Gelatin bleachers | Sulfur dioxide |
| Gin stand operators, cotton gin | Cotton dust |
| Ginners | Cotton dust |
| Glass blowers | Nitrogen dioxide |
| Glass makers | Antimony, arsenic, lead, silica, sulfur dioxide |
| Glass polishers | Lead |
| Glaze dippers, pottery | Antimony |
| Glaze mixers, pottery | Silica |
| Glost-kiln workers | Lead |
| Glue bleachers | Sulfur dioxide |
| Glue makers | Benzene |
| Gold extractors | Arsenic, mercury |
| Gold refiners | Antimony, arsenic, lead |
| Grain bleachers | Sulfur dioxide |
| Granite cutters | Silica |
| Granite workers | Silica |
| Grinders, cotton mill | Cotton dust |
| Grinding wheel makers | Silica |
| Grindstone workers | Silica |
| Gun barrel browners | Lead |
| Hair remover makers | Arsenic |

| OCCUPATION | AGENT(S) |
|---|---|
| Hairdressers | Benzene |
| Handpickers, cotton | Cotton dust |
| Hard rock miners | Silica |
| Hatters | Mercury |
| Heat treaters | Nitrogen dioxide |
| Heat treaters, magnesium | Sulfur dioxide |
| Herbicide makers | Arsenic, benzene |
| Hide preservers | Arsenic |
| Histology technicians | Benzene, mercury |
| Hydrochloric acid workers | Benzene |
| Ice makers | Arsenic, sulfur dioxide |
| Illuminating gas workers | Arsenic |
| Incandescent lamp makers | Lead |
| Ink makers | Arsenic, benzene, lead, mercury |
| Insecticide makers | Antimony, arsenic, benzene, lead, mercury, silica, sulfur dioxide |
| Insecticide users | Lead |
| Insulators | Silica |
| Insulators, wire | Antimony |
| Investment casting workers | Mercury |
| Japan makers | Arsenic, lead |
| Japanners | Arsenic, lead |
| Jet fuel makers | Nitrogen dioxide |
| Jewelers | Arsenic, lead, mercury, silica |
| Jewelry makers | Arsenic, lead, mercury, nitrogen dioxide, silica |
| Junk metal refiners | Lead |
| Jute workers | Silica |
| Kiln liners | Silica |
| Labelers, paint can | Lead |
| Laboratory workers, chemical | Antimony, arsenic, benzene, lead, mercury, nitrogen dioxide, silica, sulfur dioxide |
| Lacquer makers | Benzene, lead, nitrogen dioxide |
| Lake color makers | Antimony |
| Lamp makers, fluorescent | Mercury |
| Lamp makers, mercury arc | Mercury |
| Lead burners | Antimony, arsenic, lead |
| Lead counterweight makers | Lead |
| Lead flooring makers | Lead |
| Lead foil makers | Lead |
| Lead hardeners | Antimony |
| Lead mill workers | Lead |

JOBS AND POTENTIAL EXPOSURES

| OCCUPATION | AGENT(S) |
|---|---|
| Lead miners | Lead |
| Lead pipe makers | Lead |
| Lead salt makers | Lead |
| Lead shield makers | Lead |
| Lead shot makers | Antimony, arsenic, lead |
| Lead shot workers | Antimony, arsenic, lead |
| Lead smelters | Arsenic, lead, sulfur dioxide |
| Lead stearate makers | Lead |
| Lead workers | Lead |
| Leather workers | Lead |
| Leather makers | Benzene |
| Leather mordanters | Antimony |
| Leather workers | Arsenic |
| Lime burners | Arsenic |
| Linoleum makers | Benzene, lead |
| Linotypers | Antimony, lead |
| Linseed oil boilers | Lead |
| Lint cleaner operators, cotton mill | Cotton dust |
| Lithographers | Benzene, lead, nitrogen dioxide, silica |
| Lithotransfer workers | Lead |
| Magnesium foundry workers | Sulfur dioxide |
| Maleic acid makers | Benzene |
| Manometer makers | Mercury |
| Masons | Silica |
| Match makers | Antimony, lead |
| Meat preservers | Sulfur dioxide |
| Medical technicians | Nitrogen dioxide |
| Mercury smelters | Mercury, sulfur dioxide |
| Mercury workers | Mercury |
| Metal bronzers | Antimony |
| Metal buffers | Silica |
| Metal burners | Lead |
| Metal burnishers | Silica |
| Metal cleaners | Arsenic, nitrogen dioxide |
| Metal cutters | Lead |
| Metal grinders | Lead |
| Metal miners | Lead |
| Metal polishers | Lead, silica |
| Metal refiners | Arsenic, lead, sulfur dioxide |
| Metallizers | Lead |
| Millinery workers | Benzene |
| Mine workers | Nitrogen dioxide, silica |

JOBS AND POTENTIAL EXPOSURES

| OCCUPATION | AGENT(S) |
|---|---|
| Miners | Antimony, arsenic, mercury, nitrogen dioxide, silica |
| Mirror silverers | Benzene, lead |
| Monotypers | Antimony |
| Mordanters | Antimony, arsenic, benzene, lead |
| Mortar makers | Silica |
| Motormen | Silica |
| Musical instrument makers | Lead |
| Neon light makers | Mercury |
| Nitrate workers | Nitrogen dioxide |
| Nitric acid workers | Lead, nitrogen dioxide |
| Nitrite workers | Nitrogen dioxide |
| Nitrobenzene makers | Benzene |
| Nitrocellulose makers | Arsenic |
| Nitrocellulose workers | Benzene |
| Nitrogen dioxide workers | Nitrogen dioxide |
| Nitroglycerin makers | Lead |
| Nitrous acid workers | Benzene, nitrogen dioxide |
| Nuclear reactor workers | Lead |
| Nuclear technologists | Lead |
| Nurses | Nitrogen dioxide |
| Oil bleachers | Sulfur dioxide |
| Oil processors | Benzene, sulfur dioxide |
| Oil purifiers | Silica |
| Oilcloth makers | Benzene |
| Oilstone workers | Silica |
| Openers, cotton mill | Cotton dust |
| Optical equipment makers | Silica |
| Ore smelters | Arsenic |
| Ore smelting workers | Sulfur dioxide |
| Organic chemical synthesizers | Antimony, arsenic, benzene, nitrogen dioxide |
| Organic sulfonate makers | Sulfur dioxide |
| Oxalic acid makers | Nitrogen dioxide |
| Oxidized cellulose compound makers | Nitrogen dioxide |
| Paint makers | Antimony, arsenic, benzene, lead, mercury, sulfur dioxide |
| Paint mixers | Silica |
| Paint pigment makers | Lead |
| Painters | Antimony, arsenic, benzene, lead |
| Paper hangers | Arsenic, lead |

JOBS AND POTENTIAL EXPOSURES

| OCCUPATION | AGENT(S) |
|---|---|
| Paper makers | Arsenic, mercury, sulfur dioxide |
| Paraffin processors | Benzene |
| Patent leather makers | Lead |
| Pearl makers, imitation | Lead |
| Pencil makers | Benzene |
| Percussion cap makers | Mercury |
| Perfume makers | Antimony, benzene |
| Pesticide workers | Mercury |
| Petrochemical workers | Benzene |
| Petroleum refinery workers | Arsenic, benzene, sulfur dioxide |
| Pewter workers | Antimony |
| Pharmaceutical workers | Antimony, arsenic, benzene, mercury, nitrogen dioxide, silica, sulfur dioxide |
| Phenol makers | Benzene |
| Phosphor makers | Antimony |
| Phosphoric acid makers | Nitrogen dioxide |
| Photoengravers | Mercury, nitrogen dioxide |
| Photographers | Mercury |
| Photographic chemical makers | Benzene |
| Photography workers | Lead |
| Phthalic acid makers | Nitrogen dioxide |
| Physicians | Nitrogen dioxide |
| Pickers, cotton mill | Cotton dust |
| Picklers | Nitrogen dioxide |
| Picric acid makers | Benzene |
| Pigment makers | Antimony, arsenic |
| Pipefitters | Lead, nitrogen dioxide |
| Plasma torch operators | Nitrogen dioxide |
| Plaster cast bronzers | Antimony |
| Plastic workers | Arsenic, lead |
| Plumbers | Arsenic, lead |
| Policemen | Lead |
| Polish makers | Benzene |
| Polishing soap makers | Silica |
| Porcelain workers | Antimony, silica |
| Pottery decorators | Benzene |
| Pottery glaze dippers | Lead |
| Pottery glaze mixers | Lead |
| Pottery workers | Antimony, lead, silica, sulfur dioxide |
| Pouncers, felt hat | Silica |

| OCCUPATION | AGENT(S) |
|---|---|
| Preservative makers | Arsenic, sulfur dioxide |
| Press box operators, cotton mill | Cotton dust |
| Pressure gage makers | Mercury |
| Printers | Antimony, benzene, lead |
| Printing ink workers | Arsenic |
| Protein makers, edible | Sulfur dioxide |
| Protein makers, industrial | Sulfur dioxide |
| Pulpstone workers | Silica |
| Putty makers | Benzene, lead |
| Pyrites burners | Sulfur dioxide |
| Pyrotechnics workers | Antimony, arsenic |
| Pyroxylin-plastics workers | Lead |
| Quarry workers | Silica |
| Quartz workers | Silica |
| Raw silk bleachers | Nitrogen dioxide |
| Rayon makers | Arsenic |
| Reclaimers, rubber | Benzene, lead |
| Refiners | Mercury |
| Refractory makers | Silica |
| Refrigeration workers | Sulfur dioxide |
| Resin makers | Benzene |
| Respirator makers | Benzene |
| Riveters | Lead |
| Road constructors | Silica |
| Rock crushers | Silica |
| Rock cutters | Silica |
| Rock drillers | Silica |
| Rock grinders | Silica |
| Rock screeners | Silica |
| Rocket fuel makers | Nitrogen dioxide |
| Rodenticide makers | Arsenic |
| Roofers | Lead |
| Rotogravure printers | Benzene |
| Roving frame operators, cotton mill | Cotton dust |
| Rubber buffers | Lead |
| Rubber cementers | Benzene |
| Rubber compound mixers | Benzene, lead, silica |
| Rubber gasket makers | Benzene |
| Rubber makers | Antimony, benzene, lead |
| Sand cutters | Silica |
| Sand pulverizers | Silica |
| Sandblasters | Silica |

| OCCUPATION | AGENT(S) |
|---|---|
| Sandpaper makers | Silica |
| Sandstone grinders | Silica |
| Sawyers | Silica |
| Scouring soap workers | Silica |
| Scrap metal workers | Antimony, lead |
| Sealing wax makers | Arsenic |
| Seed handlers | Mercury |
| Semiconductor compound makers | Antimony, arsenic, lead |
| Semiconductor workers | Antimony, lead |
| Service station attendants | Lead |
| Sheep dip workers | Arsenic |
| Sheet metal workers | Lead |
| Shellac makers | Benzene, lead |
| Ship dismantlers | Lead |
| Shoe factory workers | Benzene |
| Shoe finishers | Benzene |
| Shoe stainers | Benzene, lead |
| Shot makers | Lead |
| Sign painters | Lead, mercury |
| Silica brick workers | Silica |
| Silicon alloy makers | Silica |
| Silk weighters | Lead |
| Silo fillers | Nitrogen dioxide |
| Silver extractors | Mercury |
| Silver polishers | Silica |
| Silver refiners | Arsenic |
| Slashing operators, cotton mill | Cotton dust |
| Slate workers | Silica |
| Slushers, porcelain enameling | Lead |
| Smelters | Silica |
| Soap makers | Benzene |
| Soda makers | Arsenic |
| Sodium silicate makers | Silica |
| Sodium sulfite makers | Sulfur dioxide |
| Soil sterilizer makers | Arsenic |
| Solder makers | Antimony, lead |
| Solderers | Antimony, arsenic, lead |
| Solvent makers | Benzene |
| Spacecraft workers | Silica |
| Spindle pickers, cotton | Cotton dust |
| Spinners, cotton mill | Cotton dust |
| Spooling operators, cotton mill | Cotton dust |
| Stain makers | Benzene |

| OCCUPATION | AGENT(S) |
|---|---|
| Stainers | Benzene |
| Steel engravers | Lead |
| Stereotypers | Antimony, lead |
| Stibnite miners | Antimony |
| Stone bedrubbers | Silica |
| Stone cutters | Silica |
| Stone planers | Silica |
| Storage battery chargers | Sulfur dioxide |
| Storage battery workers | Antimony |
| Straw bleachers | Sulfur dioxide |
| Street sweepers | Silica |
| Stripper operators, cotton | Cotton dust |
| Stripper operators, cotton mill | Cotton dust |
| Styrene makers | Benzene |
| Submarine workers | Arsenic |
| Subway construction workers | Silica |
| Sugar refiners | Sulfur dioxide |
| Sulfite makers | Sulfur dioxide |
| Sulfur dioxide workers | Sulfur dioxide |
| Sulfurers, malt and hops | Sulfur dioxide |
| Sulfuric acid makers | Arsenic, nitrogen dioxide, sulfur dioxide |
| Switch makers | Mercury |
| Synthetic fiber makers | Benzene |
| Tanners | Arsenic, lead, mercury, sulfur dioxide |
| Tar workers | Arsenic |
| Taxidermists | Arsenic, mercury |
| Television picture tube makers | Lead |
| Temperers | Lead |
| Textile bleachers | Sulfur dioxide |
| Textile bleachers, rayon | Nitrogen dioxide |
| Textile dryers | Antimony |
| Textile flame-proofers | Antimony |
| Textile processors | Lead |
| Textile printers | Antimony, arsenic, mercury |
| Thermometer makers | Mercury |
| Thermometer makers, vapor pressure | Sulfur dioxide |
| Thionyl chloride makers | Sulfur dioxide |
| Tile makers | Lead, silica |
| Tin foil makers | Lead |
| Tinners | Arsenic, lead |
| Tobacco seedling treaters | Benzene |

| OCCUPATION | AGENT(S) |
|---|---|
| Tooth paste makers | Silica |
| Tree sprayers | Arsenic |
| Trinitrotoluol makers | Benzene |
| Tube mill liners | Silica |
| Tumbling barrel workers | Silica |
| Tunnel construction workers | Silica |
| Tunnel workers | Nitrogen dioxide, sulfur dioxide |
| Twisters, cotton mill | Cotton dust |
| Type cleaners | Benzene |
| Type founders | Antimony, lead |
| Type metal workers | Antimony, arsenic |
| Type setters | Antimony, lead |
| Vacuum pump makers | Mercury |
| Vanadium compound makers | Lead |
| Vapor tube makers | Mercury |
| Varnish makers | Arsenic, benzene, lead |
| Vegetable preservers | Sulfur dioxide |
| Vehicle tunnel attendants | Lead, nitrogen dioxide, sulfur dioxide |
| Vine dressers | Arsenic |
| Vinyl chloride makers | Mercury |
| Vulcanizers | Antimony, benzene, sulfur dioxide |
| Wallpaper printers | Arsenic, lead |
| Warfare gas makers | Arsenic, benzene |
| Water weed controllers | Arsenic |
| Wax makers | Benzene |
| Weavers, cotton mill | Cotton dust |
| Weed sprayers | Arsenic |
| Welders | Benzene, lead, nitrogen dioxide |
| Whetstone workers | Silica |
| Wicker ware bleachers | Sulfur dioxide |
| Window shade makers | Benzene |
| Wine makers | Sulfur dioxide |
| Wire drawers | Arsenic |
| Wire insulators | Benzene |
| Wood bleachers | Sulfur dioxide |
| Wood filler workers | Silica |
| Wood preservative workers | Arsenic, mercury |
| Wood pulp bleachers | Sulfur dioxide |
| Wood stainers | Lead |
| Zinc chloride makers | Arsenic |
| Zinc mill workers | Lead |
| Zinc miners | Antimony, arsenic |
| Zinc refiners | Antimony, arsenic |
| Zinc smelter chargers | Lead |
| Zinc smelters | Sulfur dioxide |

APPENDIX IV
GLOSSARY

Aberrations: Deviations from a normal course.

Acuity: Pertaining to the sensitivity of hearing or vision.

Acute: Sharp, severe; having rapid onset, severe symptoms, and a short course.

Addison's Disease: Disease resulting from deficiency in the secretion of adrenocortical hormones.

Adhesion: A holding together by new connective tissue produced by inflammation or injury.

Air Monitoring: The continuous sampling for, and measuring of, pollutants in the atmosphere.

Albuminuria: Presence of readily detectable amounts of albumin protein in the urine.

Allergy: An abnormal response of a hypersensitive person to chemical and physical stimuli.

Alopecia: Baldness or deficiency of hair, partial or complete, localized or generalized.

Alveolar: Concerning the air spaces within the lungs.

Alveoli: Air spaces within the lungs.

Amblyopia: Reduced or dimness of vision.

Anemia: Deficiency in the hemoglobin and/or red blood cells.

Angina: Any disease characterized by attacks of choking or suffocation.

Anorexia: Loss of appetite.

Anuria: Urinary suppression or failure of kidney function.

Aplastic Anemia: Failure of bone marrow to produce red blood cells.

Arthralgia: Pain in a joint.

Asphyxia: Suffocation from lack of oxygen.

Asymptomatic: Without symptoms.

Ataxia: Muscular incoordination.

Atelectasis: A collapsed or airless condition of the lung or a segment of the lung.

# GLOSSARY

Atrophy: Reduction in size.

Attenuation: Lessening.

Auricular Fibrillation: Extremely rapid incomplete contractions of the upper heart chambers (auricles of heart).

Basophilia: A pathological condition of the blood in which the erythrocytes develop basophilic granules.

Basophilic Stippling: Spotted condition of the red corpuscles; there are also some normal white blood cells called basophils.

Benign: Harmless.

Bilirubin: The orange-colored or yellowish pigment in bile carried to the liver by the blood.

Bioassay: Estimation of the strength of a drug on a test animal.

Biologic Monitoring: Periodic examination of blood, urine, or any other body substance to determine exposure to toxic materials.

Biopsy: Removal of small bits of living tissue from the body for study.

Blood Count: A count of the number of different blood cells circulating in the body.

Blood Dyscrasia: An abnormality of the blood or blood forming system.

Body Burden: The amount of a harmful material in the body at a given time.

Bone Marrow: The soft tissue of bone which is part of the blood forming system.

Bradycardic: Slow heart action.

Brain Dysfunction: Abnormal, inadequate, or impaired function of the brain.

Bronchial Tubes: Branches or subdivisions of the trachea (windpipe).

Bronchiectasis: Dilation, usually of the terminal bronchi, often associated with abnormal secretions.

Bronchiogenic: Originating in the bronchi.

Bronchiolar: Pertaining to the bronchioles.

Bronchiole: One of the finer subdivisions of the bronchial tree. The area where oxygen and carbon dioxide are exchanged between air and blood.

Bronchiolitis Fibrosa Obliterans: Irritation of the bronchioles that results in the closing of the bronchioles with fibrous tissue.

Bronchitis: Inflammation of the bronchial tubes.

Bronchoscope: An instrument used for visual examination of the interior of a bronchus.

Bronchoscopy: Examination of the bronchi through a bronchoscope.

Bronchospasm: Spasm of the bronchi or bronchioles.

Cancer: A malignant tumor characterized by proliferation of abnormal cells (carcinoma or sarcoma).

Caplan's Syndrome: The appearance of large nodules against the background of simple silicosis.

Carcinogen: Substance which is capable of causing cancer.

Carcinoma: New growth of malignant tumor.

Carcinomatous Lymphangitis: Inflammation of lymphatic channels or vessels due to cancer.

Cardiomyopathies: Inflammation of the heart muscle.

Ceiling Limit: The maximum level of an environmental contaminant which should not be exceeded for any period of time. OSHA has some exceptions to this rule.

Chelation: Combining of metallic ions with certain heterocyclic ring structures so that the ion is held by chemical bonds from each of the participating rings.

Chemosis: Swelling of the mucous membrane of the sclera.

Chronic: Long, drawn out; designating a disease showing little change or of slow progression and long continuance.

Chronic Obstructive Lung Disease: Disease processes which cause decreased pulmonary ventilation (e.g. pulmonary emphysema, pulmonary fibrosis, chronic asthma, and chronic bronchiolitis).

Cirrhosis: Progressive fibrosis of the liver.

Colic: Spasm in any hollow or tubular soft organ such as the colon accompanied by pain.

Coma: Prolonged unconsciousness.

Comedones: Blackheads or plugging of sebaceous gland of skin.

Compound: A chemical substance composed of two or more elements joined according to the laws of chemical combination. Each compound has its own characteristic properties different from those of its constituent elements.

Concomitant: Occurring at the same time.

Conjunctiva: Membrane that lines eyelids and covers the eyeball.

Conjunctivitis: Inflammation of the membrane that lines the eyelids and the front of the eyeball.

Contaminant: A material that is foreign to the normal medium.

Coproporphyrin: A porphyrin present in urine and feces.

Cor Pulmonale: Hypertrophy (enlargement) or failure of the right ventricle resulting from disorders of the lungs, pulmonary vessels, or chest wall.

Cornea: The transparent part of the eye.

Crackles: A crackling sound heard in certain diseases.

Creatinine: 1-Methylglycocyamidine, the end product of creatine metabolism, a normal alkaline constituent of urine and blood.

Cutaneous: Pertaining to, or affecting the skin.

Cyanosis: Slightly bluish, greyish, slatelike, or dark purple discoloration of the skin due to the presence of abnormal amounts of reduced hemoglobin in the blood.

Cytology: Pertaining to the formation, structure, and function of cells.

Cytoscopy: Microscopic examination of cells for purpose of diagnosis.

Dermatitis: Inflammation of the skin from any cause.

Differential Blood Count: Determination of the number of (different) white blood cells in a cubic millimeter of blood.

Differential Diagnosis: Comparison of symptoms of two or more similar diseases to determine which disease the worker has.

Digital Clubbing: Rounding and swelling of the ends of the fingers.

Dysphagia: Inability to swallow or difficulty in swallowing.

# GLOSSARY

Dyspnea: Labored or difficult breathing.

Edema: A swelling of body tissues.

Electrophoretic: A method of analyzing the movement of charged protein particles.

Emphysema: A lung disease in which the walls of the air sacs (alveoli) have been stretched and broken down.

Emphysematous Bullae: Large blisters on lung surface filled with fluid caused by emphysema.

Eosin: An acid dye used for staining tissues for microscopic examination.

Eosinophil: A white blood cell containing granules that readily stain with the acid stain, eosin.

Epistaxis: Bleeding from the nose.

Epitheliomatous Ulceration: An open sore or lesion originating in the epidermis of the skin or in a mucous membrane.

Erethism: Triad of gingivitis, tremor, and emotional instability.

Erythema: Reddening of the skin.

Erythroblasts: Any form of nucleated red corpuscles.

Erythrocyte: The mature red blood corpuscle.

Erythroleukemia: Malignant growth of both red- and white-blood-cell forming tissues.

Erythropoiesis: The formation of red blood corpuscles.

Etiology: The study of the causes of disease.

Euphoria: An exaggerated feeling of well-being.

Exfoliative Dermatitis: The scaling off of dead skin.

Fasciculation: Small rapid movements of muscle fibers.

$FEV_1$: Forced expiratory volume in one second; a test of pulmonary function.

Fibrosing Alvelolitis: Fibrous tissue which replaces normal lung tissue following inflammation of the alveoli.

Fibrosis: A thickening, associated with growth of fibrous tissue.

Fibrotic: Abnormal formation of fibrous tissue.

FVC: Forced vital capacity; a test of lung function.

Gangrenous: Death and decomposition of body tissue due to failure of blood supply, injury, or disease.

Gastritis: Inflammation of the lining of the stomach.

Gastrointestinal: Pertaining to the stomach and intestine.

Genitourinary: Pertaining to the genitals, the urinary organs, and their accessories.

Gingivitis: Inflammation of the gums characterized by redness, swelling, and tendency to bleed.

Glycosuria: The presence of glucose in the urine.

Hematemesis: Vomiting of blood.

Hematocrit: The volume of red blood cells.

Hematologic Toxins: Poisonous substances affecting the blood or blood-forming tissues.

Hematology: The study of blood and the blood-forming organs.

Hematuria: Blood in the urine.

Hemoglobin: The red coloring matter of the blood which carries the oxygen.

Hemolysis: Breakdown of red blood cells with liberation of hemoglobin.

Hemolytic Anemia: Anemia resulting from the excessive destruction of red blood cells.

Hemopoietic: Pertaining to the formation of blood in the body.

Hemoptysis: Spitting blood or blood-stained sputum.

Hemorrhage: Profuse bleeding.

Hemosiderosis: A condition characterized by the deposition of iron containing pigment, from the disintegration of red blood cells, into the liver and spleen.

Hepatic: Pertaining to the liver.

Hepatic Injury: Damage to the liver.

Hepatitis: Inflammation of the liver.

Hepatomegaly: Enlargement of the liver.

Hilar Nodes: Nodes on the root of the lungs at level of fourth and fifth dorsal vertebrae.

Histological: Pertaining to the study of the microscopic structure of animal and plant tissue.

Hydrocephalus: Increased accumulation of cerebrospinal fluid within the ventricles of the brain.

Hyperaminoaciduria: An abnormal amount of amino acids in the urine.

Hyperemia: Congestion from an unusual amount of blood.

Hyperhidrosis: Excessive sweating.

Hyperkeratosis: Overgrowth of the horny layer of the skin.

Hyperpigmentation: Development of increased skin pigmentation.

Hyperplastic: Excessive proliferation of cells.

Hyperuricemia: Abnormal amount of uric acid in the blood.

Hypochromic Normocytic: A condition of the blood in which the red blood cells have a reduced hemoglobin content, but are normal in size.

Hypoplasia: Reduced development of tissue.

Hypoplastic: Reduced or defective production of cells.

Industrial Hygiene: The science that deals with the recognition, evaluation, and control of potential health hazards in the industrial environment.

Inflammation: The reaction of body tissue to injury.

Inorganic: Term used to designate compounds that do not contain the elements carbon, hydrogen, and oxygen.

Insidious: Working or spreading harmfully without symptoms.

Interstitial: Pertaining to the small spaces between cells.

Intravenous Pyelogram: A roentgenogram of the kidney, ureter, and pelvis.

Jaundice: A condition characterized by yellowness of skin and sclerae (white of eyes), mucous membranes, and body fluids due to deposition of bile pigment resulting from excess bilirubin in the blood.

Keratitis: Inflammation of the cornea.

Lacrimation: Secretion and discharge of tears.

Laryngitis: Inflammation of the larynx.

Larynx: Voice box.

Latent Period: The time which elapses between exposure and the first manifestation of symptoms.

Lesion: An injury, damage, or abnormal change in a tissue or organ.

Leukemia: A blood disease distinguished by a marked increase of white blood cells.

Leukemogen: Any substance or agent that produces or incites leukemia.

Leukocyte: A white blood cell.

Leukocytosis: An increase in the number of white blood cells.

Leukopenia: A reduction in the total number of white blood cells.

Lymphoblastic: A disease characterized by the presence of immature lymphocytes.

Malaise: A feeling of illness or depression.

Malignancy: A neoplasm or tumor that is cancerous.

Malignant: Virulent or harmful.

Maturation: Maturing.

Mean Corpuscular Volume: A measurement of the volume of red corpuscles.

Medical Monitoring: Periodic evaluation of body functions to ascertain state of health.

Melanosis: Unusual deposit of black pigments in different parts of the body.

Melena: Black vomit due to action of intestinal juices on free blood.

Menorrhagia: Excessive bleeding during the menstrual period in number of days, amount of blood, or both.

Mesothelioma: A malignant tumor of the membrane which surrounds the internal organs of the body.

Metastasis: Spreading of cancer cells from one part of the body to another.

Methemoglobin: A form of hemoglobin wherein the ferrous iron has been oxidized to ferric iron.

Methemoglobinemia: A condition where more than 1% of the hemoglobin in the blood has been oxidized to the ferric form.

Morphological: Pertaining to the biological study of the form and structure of living organisms.

Mucopolysaccharides: Thick gelatinous material that is found many places in the body; glues cells together, lubricates joints, and is found in blood group substances.

Mucopurulent: Consisting of mucus and pus.

Myalgia: Tenderness or pain in the muscles.

Myeloblastic: A condition where the bone marrow cell develops into a large cell in red bone marrow from which leukocytes are formed.

Myelofibrosis: Replacement of bone marrow by fibrous tissue.

Myeloid: Like marrow.

Myelopoieses: The development of bone marrow or formation of cells derived from bone marrow.

Myocardial: Concerning heart muscle.

Narcotic: Producing stupor or sleep.

Nasal Septum: A partition that divides the nasal cavity into two passages.

Nasopharyngitis: Inflamed condition of the pharynx directly behind the nasal cavity and above the soft palate.

Necrotic: Death of a portion of tissue.

Neoplasm: A new and abnormal formation of tissue, as a tumor or growth.

Nephritis: Inflammation in the kidneys.

Nephropathy: Any disease of the nerve.

Neuropathies: Any disease of the nerve.

NIOSH: National Institute for Occupational Safety and Health.

Node: A small round or oval mass of lymphoid tissue.

Nodular Ulcers: An open sore in a small aggregation of cells.

Nodule: A small node.

Nystagmus: Constant involuntary cyclical movement of the eyeball in any direction.

Opacities: Areas or spots that are not transparent.

Oropharyngeal: Concerning the central portion of the pharynx lying between the soft palate and upper portion of the epiglottis.

OSHA: Occupational Safety and Health Administration or Occupational Safety and Health Act.

Palpitation: Abnormal rhythm of the heart of which a person is acutely aware.

Pancytopenia: A reduction in all cellular elements of the blood.

Papillomas: Benign epithelial tumors.

Paresthesia: Abnormal sensation such as numbness, prickling, or tingling.

Particulate Matter: A suspension of fine solid or liquid particles in air, such as dust, fog, fume, mist, smoke, or sprays.

Pathological: Abnormal or diseased.

Percutaneous: Effected through the skin.

Peripheral Neuritis: Inflammation of terminal nerves.

Peritoneal: Concerning the serous membrane reflected over the viscera and lining the abdominal cavity.

Pernicious Anemia: Severe form of blood disease marked by progressive decrease in red blood corpuscles, muscular weakness, and gastro-intestinal and neural disturbances.

Peroneal Nerve: Nerve on the fibular side of the leg.

Phlegm: Thick mucus from the respiratory passages.

Plantar Keratosis: A horny growth on the sole of the foot.

Platelets: A round or oval disc, 2 to 4 micrometers in diameter, found in the blood of vertebrates.

Pleurisy: Inflammation of the lining of the lungs or chest cavity.

Pneumoconiosis: A condition of the respiratory tract due to the inhalation of dust particles.

Pneumonitis: Inflammation of the lungs.

Polymorphonuclear: A white blood cell consisting of several parts or lobes connected by fine strands.

Polyneuritis: A nerve inflammation involving two or more nerves.

Porphobilinogen: A substance sometimes found in the urine of patients with acute porphyria.

ppm: Parts of vapor or gas per million parts of air (by volume).

Preexisting Disease: A disease known to exist before the onset of current symptoms.

Preleukemic: A condition in which a group of nondiagnostic physical and blood abnormalities may indicate that leukemia will develop later.

Prognosis: Prediction of the future course of a disease.

Prostration: Absolute exhaustion.

Protoporphyrin: A derivative of hemoglobin containing four pyrrole nuclei.

Pruritis: Severe itching.

Pulmonary: Concerning or involving the lungs.

Pulmonary Hemosiderosis: A condition characterized by the deposition of iron containing pigment in the lungs.

Purpura: Hemorrhage into the skin or mucous membranes.

Pustular: Characterized by small elevations of the skin filled with pus.

Remission: Lessening severity or abatement of symptoms or signs.

Reticulocytosis: Increase in number of red blood cells containing a network of granules or filaments in circulating blood.

Reticuloendothelial System: Cells scattered throughout the body which have the power to ingest bacteria and colloidal particles.

Rhinitis: Inflammation of the nasal mucosa.

Rhinorrhea: Thin watery discharge from the nose.

Sanguinolent: Containing, or tinged with, blood.

Sarcoidosis: A chronic granulomatous disease of unknown etiology characterized by the formation of tubercle-like lesions in the organs such as skin, lymph nodes, lungs, and bone marrow.

Scalene Node: A particular group of lymph nodes in the neck.

Scotomas: Island-like gaps in the visual fields.

Serum: The watery portion of the blood after coagulation.

Sideroblasts: A ferritin-containing nucleated red blood corpuscle in the bone marrow.

Siderocyte: A red blood cell containing iron in a form other than hematin.

Skin Absorption: Penetration of the unbroken skin by a substance.

Sputum Cytology: Examination of the sputum cells.

Stomatitis: Inflammation of the mouth.

Striated: Skeletal muscle, consisting of fibers marked by crosswise series of streaks.

Subcutaneous: Beneath or to be introduced beneath the skin.

Supervene: The development of an additional condition as a complication to an existing disease.

Syncope: Fainting.

Synergism: Producing a total effect greater than the sum of separate effects.

Systemic: Spread throughout the body.

Tachycardia: Rapid heart action, usually defined as over 100 beats per minute.

Tachypnea: Rapid breathing.

Tex: Tex numbering system for measuring yarn size.

Threshold Limit Value (TLV): An atmospheric exposure level under which most people can work without harmful effects.

Thrombocytopenia: Decrease in number of the blood platelets.

Time-Weighted Average (Exposure): An average of several samples taken at various times during a working day. Usually more representative of the true exposure to a person for evaluation of long term effects from a harmful agent.

Toxic Nephrosis: Kidney failure due to toxic degeneration of the kidney or renal tubules.

Toxicology: Study of the effects of toxic or poisonous substances.

Trachea: Cylindrical tube from the larynx to the bronchial tubes.

Tracheitis: Inflammation of the trachea.

Tracheobronchial: Trachea or bronchial tubes.

Tracheobronchitis: Inflammation of the mucous membrane that lines the trachea or bronchi.

Trauma: An injury or a wound.

Tumor: A swelling or enlargement, may also refer to a spontaneous growth of new tissue.

Ulcerative: Causing ulcers.

Urinary: Pertaining to urine, its production, function, or excretion.

Urobilinogen: A colorless derivative of bilirubin from which it is formed by the action of intestinal bacteria.

Urticaria: A vascular skin reaction characterized by the eruption of pale evanescent wheals which are associated with severe itching.

Vascular: Blood vessels.

VC: Vital capacity; a test of lung function.

Ventricular Arrhythmias: A rhythmic disturbance arising in the ventricles or the lower chambers of the heart that pump blood into the arteries leading to the lungs and body.

Vertigo: Dizziness.

Viscera: Internal organs enclosed within a cavity such as the abdominal or thoracic cavities.

# OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION REGULATIONS

152.a

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Note: This information is to be supplied by NIOSH as per 1 May 1978 telecon with Stanley Kusnetz, Project Officer