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 (a drastic reduction in the number of red blood cells), 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
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
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

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
welders
window shade makers
wire insulators

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

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, and
—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, and
—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 (hemotologic toxins),

—ionizing radiation exposures (which also affect the blood-forming organs), and

—radiomimetic substances (those which imitate radiation effects).

Signs and Symptoms

Acute Poisoning

Acute benzene intoxification 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, tingling),

—staggering gait,

—incoherent speech, and

—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, and

—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, and
—tiredness, lassitude, weariness, fatigue and weakness.

Other symptoms may include:

—Pallor,
—tendency to bruise easily,
—bleeding gums,
—nose bleeds,
—retinal hemorrhages, and
—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 Evaluations

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

Blood

—arterial blood benzene level
—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
—drastic reduction in red blood cells (aplastic anemia)
—increased mean corpuscular volume (average volume of red blood cells)
—reduced hemoglobin (that part of the red blood cell that carries oxygen)

—increased plasma bilirubin (orange-colored or yellowish pigment in bile)
—hemosiderosis of the liver, spleen, kidneys, or bone marrow (a condition characterized by the deposition of an iron-containing pigment in these sites)
—the bone marrow may have reduced capacity to produce 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 capacity to produce 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 epidemiologic 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:[25]

Aksoy et al.[26] 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.[27] 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.[28] and Hartwick and Schwanitz[29] 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[30] collected, he concluded that cases showing less than 5,000 white blood cells per cubic millimeter should be considered positive; 7,500 to 9,000 was considered the normal count.

Hardy and Elkins[31] 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[32] 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.[33] 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[34] 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, and
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 absorption 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, and
4. absorption on silica gel.

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

1. Colorimetric evaluation, and
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 ½ 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). NOTE: This standard is currently being litigated in the U.S. Court of Appeals for the 5th Circuit. (American Petroleum Institute et al. vs. OSHA.)

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, and
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.

Carbon Monoxide

Introduction

Carbon monoxide is a colorless gas produced by incomplete burning of carbon-containing materials. On inhalation, it acts as an *asphyxiant*, causing a decrease in the amount of oxygen delivered to the body tissues. Carbon monoxide combines with hemoglobin (the oxygen carrier in the blood) to form carboxyhemoglobin, which reduces the oxygen carrying capacity of the blood.

The two main sources of carbon monoxide exposure are the internal combustion engine and cigarette smoking.

The blood of cigarette smokers contains between 3 and 10 percent carboxyhemoglobin (COHb) depending on the number of cigarettes smoked and the manner of smoking, inhaling or not inhaling. During smoking, the individual is being exposed to the equilvalent of 400-500 parts per million carbon monoxide. The COHb of non-smokers is approximately 0.5-0.8 percent. Thus, in evaluating occupational exposure to carbon monoxide, the smoking habits of the individual must be carefully evaluated.

An exposure to carbon monoxide is usually sudden and the symptoms are acute and rapid in onset. *Headache* and *dizziness* may rapidly progress to unconsciousness depending on the rate of build-up of COHb in the blood. Once the person is removed from the carbon monoxide exposure, the process is reversible and no permanent damage is known to occur.

Prolonged exposure and unconsciousness may cause brain damage and result in neurological disturbances.

If chronic carbon monoxide poisoning exists, it is not a clearcut, identifiable entity that can be diagnosed. Toxicologic and epidemiologic studies have not yielded adequate information to establish any physical impairment from chronic exposure to carbon monoxide.

Carbon monoxide is especially serious for persons with chronic heart or lung disease. The reason for this is that the carbon monoxide in the blood reduces the amount of oxygen available to an already damaged heart muscle.

Occupations with Potential Exposures to Carbon Monoxide

acetic acid makers
airplane pilots
ammonia makers
arc welders
artificial abrasive makers
artificial gas workers
automobile users
bakers
blast furnace gas users
bisque-kiln workers

blacksmiths
blast furnace workers
blockers (felt hat)
boiler room workers
brass founders
brewers
brick burners
busdrivers
carbide makers
cable splicers
carbon monoxide workers
cement makers
charcoal burners
chauffeurs
chimney masons
chimney sweepers
coal distillers
coke oven workers
cupola workers
diesel engine operators
compressed air workers
divers
dock workers
drier workers
firemen
enamelers
Fischer-Tropsch process work
formaldehyde makers
foundry workers
furnace starters
furnace workers
garage mechanics
gas workers (illumination)
gasoline engine testers
gas station attendants
heat treaters
iron workers
Kraft recovery furnace workers
laundry workers
lift truck operators (propane and gasoline)
lime kiln workers
mercury smelters
metal oxide reducers
metal refiners
methanol makers
miners
mond process workers
monotypers
nickel refiners
nickel smelters
organic chemical synthesizers
oxalic acid makers
patent leather makers
police
producer gas workers
pottery kiln workers
sanitation workers
steel makers
sewer workers
stokers
solderers
toll collectors (highway)
traffic controllers
tunnel attendants
tunnel workers
warehouse workers
water gas workers

welders
wood distillers
zinc white makers

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

In the medical history, the following should be considered:
—Neurological diseases, and
—it is important to note that persons with anemia, cardiovascular disease, and chronic lung disease have a decreased ability to resist the effects of carbon monoxide.

Occupational History

Potential nonoccupational sources of carbon monoxide include:
—Air pollution (particularly in areas of high motor vehicle use),
—cigarette smoking,
—cooking with charcoal in enclosed areas,
—burning carbon-containing materials in enclosed space,
—hobbies involved with the operation of automobiles or gasoline engines,
—working as a volunteer fireman,
—malfunctioning stove, furnace or heater, and
—faulty auto exhaust system.

Signs and Symptoms

Acute Carbon Monoxide Poisoning

—headache
—dizziness
—nausea
—vomiting
—drownsiness
—loss of consciousness

Initially, there is lack of color in the skin (skin pallor). Later, the skin and mucous membrane may be cherry red due to carboxyhemoglobin formation. Breathlessness upon exertion, rapid throbbing or fluttering of the heart (palpitation), and pain on the surface of the chest in the heart area (precordial pain) may be present. Excess fluid in the lung tissues (pulmonary edema) may also occur, or the victim may develop pneumonia.

It is important to ascertain the circumstances associated with carbon monoxide poisoning since the action of carbon monoxide is favored by conditions of heat, humidity, and a greater amount of muscular activity.

Chronic Carbon Monoxide Poisoning

There is conflicting opinion concerning the chronic effects of carbon monoxide. Other than increased carboxyhemoglobin levels in the blood, there are a few objective signs. Persons with chronic exposure to low levels of carbon monoxide develop a tolerance for it. However, the following have been described as characteristic symptoms of chronic carbon monoxide poisoning:

—loss of muscular strength and mental alertness,
—persistent headache,
—constant dizziness and light headedness, and
—auditory nerve damage.

Exposures to low levels of carbon monoxide may cause or enhance myocardial alterations (heart changes) in persons with coronary heart disease.

Laboratory and Clinical Examinations

(See Decision-Making Process)

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

—Blood carboxyhemoglobin of 10 percent or more,
—hemoglobin value may be increased,
—electrocardiogram may show sinus tachycardia and ST segment changes, and
—electroencephalogram may show focal and diffuse epileptiform (resembling epilepsy) changes which later disappear.

Epidemiology

Acute carbon monoxide poisoning from inhalation is well documented in the scientific literature. It is the most common poisoning in industry and may occur wherever internal combustion engines are in use. However, the question of whether chronic carbon monoxide poisoning exists has not been resolved in spite of numerous studies conducted by various researchers.

Many of the reports dealing with carbon monoxide (CO) toxicity are in terms of carboxyhemoglobin (COHb) percentage in blood. The percent of COHb depends on many factors including CO concentrations in air, total time of exposure to various air concentrations of CO, diffusion rate of CO through the lungs, ventilation rate, type of activity being done, metabolic rate, barometric pressure, and temperature. NIOSH[35] recommends an allowable level for CO of 35 ppm based on an 8-hour time-weighted average exposure so that COHb percent does not exceed five. The current allowable limit of 50 ppm CO based on an eight-hour time-weighted average exposure is designed to maintain COHb less than 10%.

SYMPTOMS CAUSED BY VARIOUS AMOUNTS OF CARBON MONOXIDE HEMOGLOBIN IN THE BLOOD[36]

BLOOD SATURATION % COHb	SYMPTOMS
0-10	No symptoms.
10-20	Tightness across forehead, possible slight headache, dilation of cutaneous blood vessels.
20-40	Headache and throbbing in temples. Severe headache, weakness, dizziness, dimness of vision, nausea, vomiting, collapse.
40-50	Same as previous item with more possibility of collapse and syncope. Increased respiration and pulse.
50-60	Syncope, increased respiration and pulse, coma with intermittent convulsions and Cheyne - Stokes respiration.
60-70	Coma with intermittent convulsions. Depressed heart action and respiration and possible death.
70-80	Weak pulse and slow respiration, respiratory failure and death.

TIME FOR VARIOUS CONCENTRATIONS OF CARBON MONOXIDE TO PRODUCE 80% EQUILIBRIUM VALUE OF BLOOD SATURATION

CO IN AIR ppm	BLOOD SATURATION % (80% of Approx. Equil. Values)	TIME (Hours)
200-300	23-30	5-6
400-600	36-44	4-5
700-1,000	47-53	3-4
1,100-1,500	55-60	1½-3
1,600-2,000	61-64	1-1½
2,100-3,000	64-68	½-¾
3,100-5,000	68-73	20-30 Min.
5,000-10,000	73-76	2-15 Min.

There have been a number of reports showing evidence of behavioral effects in man on exposure to low levels of CO. The results of these studies indicate that exposure to low concentrations of CO could affect a worker's ability to work safely. McFarland[37] reported difficulties in visual discrimination at 5% COHb (similar results were reported by Halperin[38]). Horvath[39] reported significantly impaired vigilance at 6.6% COHb. Schulte[40] indicated various physiological and behavioral tests were effected by COHb levels as low as 5%. Beard[41,42] in two reports showed exposure to CO in concentrations ranging from 50-250 ppm caused a deterioration in the ability to discriminate auditory stimuli and exposures to 50 ppm caused impairment in time discrimination. Trouton[43] reported impairment in muscle limb coordination at COHb levels of approximately 5%. There have been a number of studies made relating carbon monoxide exposures to cardiovascular ramifications. NIOSH[44] concludes that the results of these studies provide sufficient evidence so that "based on cardiovascular alterations which could prove to be of severe physiological consequences for persons with CHD (coronary heart disease), a significant portion of who are in the worker population, it seems advisable that levels of COHb (carboxyhemoglobin) in excess of 5% should be avoided."

Evidence of Exposure

Air Sampling and Analysis

There are a variety of direct-reading field instruments for the evaluation of carbon monoxide in air including Hopcalite-type carbon monoxide meters and detector tubes. Air samples can also be collected for carbon monoxide by techniques including adsorption on silica gel. Analysis may be performed by calorimetric, infrared spectrophotometric, and gas chromatographic techniques.

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

Allowable Exposure Limits

The Occupational Safety and Health Administration (OSHA) limits carbon monoxide to 50 parts per million parts of air by volume based on an eight-hour time-weighted average exposure.

See Reference 29-38, Epidemiologic Data, Appendix D.

Conclusion

Diagnosis of occupational carbon monoxide exposure is based on the following:

1. Confirmed history of occupational exposure to carbon monoxide,
2. carboxyhemoglobin in excess of 10 percent, and
3. clinical findings compatible with carbon monoxide poisoning.

One medical researcher (Hunter, D. 1969. *The Diseases of Occupations*, 4th ed. Boston: Little, Brown and Co.) states that claims of impaired health from exposure to carbon monoxide are unjustified unless three conditions can be established:

1. At least a 50 percent saturation of the blood with carbon monoxide (not carboxyhemoglobin) or evidence of enough carbon monoxide in the air to produce it,
2. an exposure of at least three hours, and
3. continuous and complete unconsciousness for at least six hours after return to fresh air.

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. Epidemiologic stuides 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 list 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

(See also Decision-Making Process)

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, and

—a respiratory questionnaire, (Appendix C), 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 mucous 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, and
—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, and
—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, and
—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, and
—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:
  - -diffuse redness (erythema),
  - -swelling of body tissues (edema), and
  - -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), and
- —inflammation of follicles.

Systemic

- —Loss of appetite (anorexia),
- —nausea, and
- —vomiting.

Eye

—Inflammation of the membrane that lines the eyelids and the front of the eyeball (conjunctivitis).

Laboratory and Clinical Examinations

Additional data 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, and,
—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, and
—examination of the urine for cancer cells.

Skin

—Total removal of lesion, and microscopic (histological) examination.

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

Respiratory

—Chest X-ray and
—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 nontopside workers have a higher rate for carcinoma of the kidney.

The reports of disease response in parenthesis are from NIOSH:[45]

Doll[46] reported an "81% excess of lung cancer deaths among gas works pensioners (gas retort workers) in comparison with the general population." Lloyd[47] reported that "coke oven workers had an average lung cancer mortality rate of 2½ times that predicted by the experience of all steelworkers."

Lloyd[47] and Redmond et al.[48] 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 2½ times the expected rate, and for side oven only it is more than 1½ times the expected mortality. (All of these rates are based on 5 or more years exposure in the job category.)

Henry et al.[49] reported an "excess risk of bladder cancer among men employed at coal carbonization processes." Redmond et al.[48] did not observe an excessive incidence of bladder cancer in 4,661 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[50] 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, and
3. spectrophotometric techniques.

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

Allowable Exposure Limits

The standard adopted by the Occupational Safety and Health Administration (OSHA) provides that no employee in the regulated area may be exposed to coke oven emissions in excess of 150 micrograms per cubic meter of air for an eight-hour period. For the purpose of the Standard, coke oven emissions as defined as the benzene-soluble fraction

of total particulate matter present during the destructive distillation or carbonization of coal for the production of coke. The regulated area is the coke oven battery, including top side, punchside, coke side and their machinery, the wharf, and screening station. Beehive ovens have also been established as a regulated area.

Conclusion

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

1. Confirmed history of occupational exposure to coke oven emissions and
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 and
2. clinical findings of respiratory, genitourinary, or skin examination as outlined above and medical history.

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 casued 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 reponse) 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, 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 operator (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

(See also Decision-Making Process)

The following should be considered in the Medical Evaluation:

—Respiratory allergy,
—chronic lung disease,
—other diseases of the cardiopulmonary system, and
—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, and
—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 and
—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 Evaluations

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, and

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 workplace, 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 6,631 employees, Martin and Higgins[51] 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_{1s}).

Shilling et al.[52] 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[53] 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.[54] 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 ½[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%)
	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, or both.

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
	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
	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[55] 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 table on the next page, taken from the report, summarizes respiratory symptoms among the workers studied.

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[56] report a 3-year study of 1,359 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[57] 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.[58] 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.

RESPIRATORY SYMPTOMS IN MALE CARDERS, SPINNERS, AND WEAVERS WITH BYSSINOSIS AND IN THOSF 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)

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 standard adopted by the Occupational Safety and Health Administration (OSHA) provides that no employee in the textile industry shall be exposed to greater than 200 micrograms per cubic meter air of lint-free respirable cotton dust averaged over an eight-hour work shift, unless these employees are engaged in slashing or weaving, in which case they shall not be exposed to greater than 750 micrograms per cubic meter air of lint-free respirable cotton dust averaged over an eight-hour work shift. In all of the other industries where employees are exposed to cotton dust, no employee shall be exposed to greater than 500 micrograms per cubic meter air of lint-free respirable cotton dust averaged over an eight-hour work shift. OSHA has treated cotton gins separately and has not imposed a numerical permissible exposure limit. OSHA has, however, established requirements for work practices, respirator usage, medical surveillance, and recordkeeping. NOTE: This standard is currently (1978) being litigated in a number of U.S. Courts of Appeal.

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 workday following absences from exposure to cotton dust, and
3. a reduction of ventilatory capacity following return to work on the first workday and during the workday as demonstrated by lung function test.

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).