

**A GUIDE TO
THE WORK-RELATEDNESS
OF DISEASE**

Revised Edition

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Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health
January 1979**

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This guide is based upon the reports submitted in accordance with NIOSH Contract No. 210-75-0075 (J. E. Osterritter, M.D., Principal Investigator) and NIOSH Contract No. 210-77-0089 (Barbara M. O'Conner and George Christakis, M.D., authors).

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DHEW (NIOSH) Publication No. 79-116

PREFACE

The mission of the National Institute for Occupational Safety and Health (NIOSH) is to protect the health and safety of working men and women. Within the context of its program are NIOSH efforts that are directed toward the identification of those disease conditions that are closely related to or caused by the work environment as a necessary prerequisite to their prevention.

This guide, originally published in part in 1976, has been revised and expanded to reflect new knowledge as well as suggestions submitted by users of the guide. The guide is designed primarily as an aid to state agencies and others concerned with occupational disease compensation. It presents one method for assembling and evaluating evidence that may be relevant in determining the work-relatedness of a disease in an individual. Information on fourteen disease-producing agents is presented to illustrate the decision-making process. It should be noted that such information may not be complete and does not necessarily reflect the most recent data regarding health standards and epidemiologic studies.

NIOSH will welcome suggestions from users of the guide for its improvement.

ABSTRACT

This guide discusses various factors associated with establishing the relationship between disease and occupation. Prepared as an aid to state agencies, physicians, and others concerned with workers' compensation for occupational disease, the publication describes a method for collecting, organizing, and appraising medical, occupational, and other evidence with the aim of determining the probable work-relatedness of a given disease. Illustrative material on fourteen disease-producing agents is included. The guide also contains a list of occupations with potential exposure to selected agents, and other information that may be useful to those with decision-making responsibility in cases of occupational disease.

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ACKNOWLEDGMENTS

We wish to express appreciation to the following members of the advisory committee for their contributions during this program:

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Austin F. Henschel, Ph.D.; Lorice Ede; and Jim Carpenter of the Division of Technical Services, NIOSH, contributed substantially to reviewing, editing, and preparing the manuscript for publication.

INTRODUCTION

Background

Until this century, suing the employer was the only way for disabled workers or their families to obtain compensation for on-the-job injuries. Under common law, workers had to prove the employer's negligence in order to be compensated for work injuries. The injured worker, because of this narrow interpretation of law, found that compensation through the courts was seldom satisfactory.

By 1920, all but six states had passed workers' compensation statutes that sought to remedy past deficiencies and to avoid costly litigation by making employers responsible for the economic loss to workers due to injuries sustained at work.

Although the new laws established a more equitable compensation system, the system has not kept pace with the substantial changes that have taken place in the last half century in the labor force, in medical knowledge and techniques, and in industrial toxicology. In 1970, Congress established a National Commission on State Workmen's Compensation Laws to reexamine the adequacy of the compensation system in light of these changes. The Commission's published report to the Congress in 1972 lists the objectives of a modern workers' compensation program. Included is a statement that all work-related injuries or diseases should be covered by the compensation system. The report also states that coverage restricted to a list of specified occupational diseases is incompatible with complete protection.

Decision-Making

In order for the compensation system to treat both injury and disease in a uniform manner, disease must be related to the workplace as effectively as injury. For the decision-maker, however, establishing the causality of a disease is often a difficult task, especially when it becomes necessary to decide if an employee's disease resulted from, or was aggravated by, employment-related factors.

In contrast to a traumatic injury, which is readily apparent to the affected employee and to those around him, a cause-effect relationship between disease and an agent or conditions in the workplace may not be clear. Occupational disease may be slow to develop. Symptoms of disease may be confused with changes that are due to the aging process, or with the effects of smoking or alcohol abuse. Additionally, information on past work exposures is often unavailable, inadequate, or incomplete. Not all individuals react in the same way to similar exposures to disease-producing agents. Off-the-job exposures may contribute or be a primary cause of illnesses and accidents. These are but some of the factors which must be considered in the decision-making process.

The decision of the person responsible for determining the work-relatedness of a disease must be based on an evaluation of the available information. When appropriate evidence is presented in a logical and orderly sequence, when major issues are identified, and the basis for any presumption is defined, then the decision-making process is facilitated and an equitable decision is likely to result.

The following text outlines and describes a method for the collection, presentation, and evaluation of medical, occupational, and other evidence of occupational disease; presents selected information on fourteen disease-producing agents to illustrate the methodology; and discusses some problem areas associated with decision-making.

CHAPTER I—AN APPROACH TO DECISION-MAKING

Rationale

In the current workers' compensation system, the end result of the adjudicatory process is a decision that the claimant (employee) has or has not established that he has an occupational disease, that is, a disease condition resulting from, or aggravated by, his employment. In general, a disease is occupational if:

1. The medical findings of disease are compatible with the effects of a disease-producing agent or agents to which the worker has been exposed;
2. there exists in the worker's occupational environment (past or present) exposure to an agent or agents sufficient to have caused the disease; and
3. the weight of evidence supports that the disease is of occupational rather than non-occupational origin.

It would be convenient if a method could be devised which invariably led to a correct and unequivocal decision regarding the presence of an occupational disease. However, it is doubtful that such a system could be developed. A case in which the relationship of an illness to a documented agent exposure is clearly evident is not apt to be contested or to require the mechanism of a formal claims inquiry. The element of judgment is minimal and decision-making is relatively simple.

On the other hand, decision-making may be extremely difficult in many contested claims. Honest differences of opinion are common, "facts" may be subject to different interpretations,

and considerable judgment is necessary when data are lacking or incomplete.

This guide is an effort to define a step-by-step method for assembling and appraising evidence for the purpose of aiding the decision-making process. It is intended to be of particular assistance in cases where the suspected agent is not generally known to produce disease, and in those in which nonoccupational exposures must be considered.

The Method

This guide presents a suggested approach to decision-making that consists of six basic steps:

1. Consideration of evidence of disease,
2. consideration of epidemiologic data,
3. consideration of evidence of exposure,
4. consideration of validity of testimony,
5. consideration of other relevant factors, and
6. evaluation and conclusion.

Each of these steps is discussed fully in subsequent chapters. The importance of individual steps will vary according to the type of agent, the amount and quality of medical and occupational information available, and past experience with similar situations. Occasionally, one or more steps can be omitted. However, with occupational diseases, what appears to be "obvious" is often subject to controversy, and it is important to assemble complete information wherever possible in order to assure an equitable decision.

CHAPTER II—EVIDENCE OF DISEASE

The first consideration in determining the probability of a cause-effect relationship between an illness and an agent at the workplace is to establish:

1. That a disease condition does, in fact, exist, and
2. that the particular manifestations of the disease appear to be the result of exposure to a specific harmful agent.

The medical evidence which may be elicited in the course of the medical evaluation should cover the above points. Generally, a *medical evaluation* should include:

1. An analysis of the employee's medical, personal, family, and occupational histories;
2. a thorough physical examination and clinical evaluation (analysis of signs and symptoms); and
3. a laboratory evaluation (analysis of the results of specific tests).

Medical History

In order to determine the origin of illness, the worker's past medical history must be evaluated by the physician. A routine medical history includes the dates and details of:

- Onset of present illness,
- all previous illnesses (childhood, physical, mental),
- injuries,
- surgical procedures, and
- hospital admissions.

In addition, the medical history should include any details specific to a suspected occupational causative agent.

Personal History

This section of the history should give consideration to:

- Age, sex, marital status, number of children,
- name and location of all places of residence since birth,
- areas visited prior to onset of symptoms,
- alcohol and tobacco use (how much and how long),
- medications or drug use (past and present),
- recreation and hobbies,
- use of chemicals in the home (cleaning agents, aerosols, etc.), and
- details specific to a suspected causative agent.

Family History

This section of the history should consider, for each of the worker's parents, siblings, spouse, and children:

- Age, sex, and health status (if deceased, cause of death), and
- any chronic or occupational disease in the family or in persons in the worker's household.

Occupational History

The employee's complete occupational history, including military service, is also necessary in determining the origin of illness. The following factors regarding past and present occupations should be evaluated:

- Job titles,
- type of work performed (complete listing of actual duties),
- duration of each type of activity,
- dates of employment and worker's age for each job activity,
- geographical and physical location of employment,
- product or service produced,
- condition of personal protective equipment used (if any) and frequency and duration of periods of use, and
- nature of agents or substances to which worker is or has been exposed, if known. Include frequency and average

duration of each exposure situation. (See also Evidence of Exposure, page 11.)

Clinical Evaluation

This portion of the medical examination may vary somewhat with the type of illness but should include at least the following:

1. Routine examination of all physiological systems—
 - head and neck
 - eyes, ears, nose, and throat
 - endocrine
 - genitourinary
 - musculoskeletal
 - neurological
 - respiratory
 - cardiovascular
 - gastrointestinal,
2. observation and evaluation of behavior related to emotional status,
3. specific examination for health effects of suspected or possible disease agents (seek competent medical consultation),
4. comparison of date of onset of symptoms with occupational history,
5. evaluation of results of any past biological or medical monitoring (blood, urine, other sample analysis) and previous physical examinations, and
6. evaluation of laboratory tests: routine (complete blood count, blood chemistry profile, urinalysis) and specific tests for suspected disease agents (e.g., blood or urine test for specific agent, chest or other X-rays, liver function tests, pulmonary function tests).

CHAPTER III — EPIDEMIOLOGY

Epidemiology is the branch of medical science that deals with the incidence, prevalence, distribution, and control of the diseases that occur amongst human populations. It is the study of the distribution and determinants of disease frequency in man.

Epidemiology is concerned, among other things, with measuring the frequency of illnesses and deaths in certain population groups and with the study of the relationship between exposure and incidence of disease. Thus, studies of illness in groups of workers have made it possible to relate some diseases to various substances with which the workers had been in contact. Epidemiologic studies point up possible associations but do not prove cause-effect relationships.

Epidemiologic studies of coal miners demonstrated that prolonged exposure to coal mine dust could produce the crippling lung disease, coal workers' pneumoconiosis (black lung). Other studies have shown the relationship between workers' illness and exposure to sugar cane dust (bagassosis), cotton dust (byssinosis), silica dust (silicosis), and various fibrous silicates (asbestosis).

Epidemiologic studies have often revealed the carcinogenic action of certain substances and chemicals. Some studies were simply descriptive accounts of observed effects. Scrotal cancer was noted in English chimney sweeps two hundred years ago, and skin cancers in chromium workers at the turn of the last century. More recent studies have shown the carcinogenic properties of arsenic, vinyl chloride, ionizing radiation, and other agents.

Epidemiologic data documenting that groups of workers and other human populations exposed to a suspected agent have sustained certain types of illnesses may be extremely helpful in establishing the fact that the substance in question may cause an illness of a certain type. Whatever epidemiologic data is available should be included in the evidence presented.

CHAPTER IV — EVIDENCE OF EXPOSURE

Having heard evidence that establishes the medical condition of the claimant and its compatibility with known health effects of the suspected agent, and epidemiologic information about human populations with similar exposure histories, the examiner must consider evidence of exposure of the claimant to the suspected agent. Generally, occupational data will be presented for each relevant job or duty. The following information would be helpful:

1. Identification of the substances handled or used directly in operations in the area or in nearby areas;
2. any information from industrial hygiene studies, especially air sampling data, that indicate magnitude of worker exposure for the job or similar jobs (see specific guides);
3. data to be accumulated for work exposure evaluation:
 - a. inhalation exposure information—expert testimony should be obtained concerning general environmental conditions, especially when there are no industrial hygiene studies available as evidence. Such testimony should include reference to at least:
 - (1) Establishing the precise chemical or physical form of the agent (name the chemical; specify type of dust);
 - (2) a complete description of the operation *as performed by the worker* including materials handling practices, accessory equipment, operating procedures, and protective equipment;
 - (3) information on the particle size of the agent (for dusts) generated by the operation;

- (4) information about the solubility of the agent affecting absorption by the body;
 - (5) possible additional modes of entry of the agent into the body (inhalation, ingestion, skin absorption);
 - (6) available ventilation:
 - was general exhaust ventilation provided?
 - was local exhaust ventilation provided?
 - was it properly designed?
 - was it installed to design specifications?
 - was it properly maintained?
 - was it properly used by the operator?
 - was contaminated exhaust air recirculated into the plant?
 - (7) general housekeeping:
 - was dry sweeping done?
 - were spills cleaned up properly?
 - was equipment properly maintained and serviced?
 - were all plant areas regularly cleaned?
 - were materials stored properly to prevent spills or leaks?
 - (8) respiratory protection (While respirators are not the preferred method of protecting workers from inhalation of airborne toxic agents, they are sometimes used until other controls can be installed. They must be used properly to fulfill this function, and testimony directed toward this point should be elicited.):
 - was the proper type of respirator used? It should have been selected by an industrial hygienist for the specific agent involved, and approved by NIOSH or the Mine Safety and Health Administration.
 - were the respirators fitted properly? Leaks in the facepiece negate effectiveness.
 - did employee use the respirators?
 - were cartridges, filters, etc. changed at appropriate intervals?
 - were employees trained in the proper use, purpose, and care of respirators?
 - were the respirators periodically inspected and maintained?
- b. skin contact, skin absorption, and ingestion.
Evidence should include information regarding:

- (1) potential for skin contact
 - was the operation a “closed system”?
 - was personal protective clothing used?
 - was the proper type of clothing supplied?
 - was it used properly? laundered properly?
 - were change rooms available? protective skin creams? emergency washing facilities?
- (2) potential for ingestion
 - was smoking permitted in the work area?
 - were smoking materials permitted in the work area?
 - was eating permitted in the work area?
 - was food stored or prepared in the work area?
 - were there separate facilities for storing food and eating?
 - were proper washing facilities available?

Exposure Evaluation

The best evidence to confirm the exposure of a worker to an agent is measurements (such as air samples, noise levels, or radiation measurements) obtained at the worker's actual job stations, past and present. Factors which should be considered when evaluating the measurements are:

1. *Number of samples* (or duration of time covered by samples). In most cases, a few (two or three) samples covering only a small portion of a working day are not sufficient to establish degree of exposure. Generally, samples or measurements should be obtained covering most of a complete working day; covering several non-consecutive work days is even better. For very short duration samples or readings (less than 15 min.), a minimum of seven samples, spaced randomly over the workday, is advised.

2. *Location of samples.*

The best location for sample taking is in the breathing zone (within a few inches of nose and mouth) of the employee or a worker doing an identical job, under conditions identical to those under which the employee worked. Samples obtained at a stationary point in the work environment (area samples) can give an indication of possible exposure but can also be very misleading. For example, measuring noise levels a few inches from a

noisy machine when the worker is located several feet away may indicate erroneously high exposures. Obtaining air samples for a solvent at the center of the room, when the worker must lean into a solvent tank, would indicate erroneously low exposures.

3. *Air sampling method.*

The methods mentioned in the illustrative agent section of this guide are those commonly used or accepted in the industrial hygiene profession. Other methods may exist and give satisfactory results. However, expert opinion should be obtained concerning their validity. All equipment used should be accurately calibrated.

4. *Laboratory analysis.*

Analysis of air samples is a difficult science and should be performed by experienced, competent persons. Laboratories can be accredited by the American Industrial Hygiene Association for these analyses. In any case, the laboratory's previous experience with the specific type of analysis should be ascertained. Certification of laboratory staff is another indication of competence.

CHAPTER V — AGGRAVATION OF PREEXISTING CONDITIONS

With regard to occupational disease, there is no generally accepted medical definition of aggravation. In the current system of workers' compensation, aggravation of a preexisting disease or physical impairment may be defined as any occupational occurrence, act, or exposure that will make worse, intensify, or increase the severity of any physical or mental problem known to exist before the occupational exposure. An example of aggravation would be the effects on an employee with known allergies exposed to allergens in the workplace resulting in frequent asthmatic attacks. In another example, a recovered alcoholic with mild liver damage is exposed to carbon tetrachloride at work, resulting in greater liver damage. This definition implies that if there is any occupational contribution to an existing disease, the disease can become compensable. However, this guide is concerned solely with the causation of disease and whether or not the causes are occupational.

The existence of a condition before exposure does not necessarily mean before employment. Many companies change processes and products from time to time. When such changes occur during an employee's period of employment, there may be an aggravation of a condition that was not adversely affected by prior work in the same job or plant.

Any stress may be an aggravating factor and has been so considered by the courts for such jobs as firefighting and police work.

Since most states hold that the employer accepts the worker "as is," such factors as age, sex, heredity, and obesity can be logically excluded from the list of causative factors. This leaves

those environmental (occupational) exposures—mechanical, chemical, physical, or biologic—which may occur at work or in the nonworking environment, as candidates for discussion of the “cause” of an aggravated disease or condition.

This consideration appears to lead to a very straightforward decision-making scheme to weigh the “percent contribution” of various factors in a specific case with the aim of awarding compensation on a contributory basis. Unfortunately, no such single approach is feasible.

Aggravation cases frequently have multiple causes, not all of which are known, and most of which are poorly understood. The table on page 17 lists some agents which may contribute to disease and aggravation of disease.

A problem with aggravation of chronic diseases is that there are many parameters involved. The causes, courses, and eventual outcome of these diseases are usually unknown and poorly understood. As chronic diseases progress, they may exhibit irregular periods of worsening and of improvement. This factor confounds the role of an aggravating agent, and it is therefore necessary to medically monitor these employees over several of the cycles of improvement-worsening. Furthermore, the time of life when symptoms of chronic disease develop often contributes to the complexity of the problem, since both the degenerative processes of aging and the appearance of chronic diseases are associated with the middle years.

Arthritis

Arthritis is a disease that is almost universally present in the older age group. Arthritis can cause effects that range from nuisance aches to severe incapacity. Certain abattoir workers are required to work in damp, cold conditions. Over the years, some of these workers develop a disabling form of arthritis, but some escape it entirely. Are the work conditions responsible for the disabling arthritis? The courts have most often held that they are, but since the cause of arthritis is unknown, these decisions are based on adjudicatory and administrative rulings supported by medical testimony.

CONTRIBUTORY AGENTS

Disease	Nonoccupational	Occupational
1. HEART DISEASE (cardio-vascular including coronary occlusion)	age heredity sex smoking diet obesity stress medication or drugs climate	various chemicals, solvents, gases pulmonary irritants unusual exertion stress temperature
2. HEARING LOSS	age heredity noise impacted cerumen (wax) foreign body in ear canal ear infection nasopharyngitis medication or drugs trauma	noise foreign body in ear canal trauma nasopharyngeal irritants
3. ARTHRITIS OR "RHEUMATISM"	age heredity diet trauma infection obesity stress	repeated articular movement trauma cold, damp work environment improper lifting work-required poor posture
4. PULMONARY (LUNG) DISEASES	age heredity sex smoking allergy air pollution infection climate	various dusts, gases, mists, etc. allergens wearing of respirators decreased oxygen supply temperature, humidity

Coronary Artery Disease

Coronary artery disease, which may lead to a heart attack, is one of the most frequent "preexisting conditions" cited as being aggravated by work. There are those who feel that heart attacks should never be compensable and, since they have such complicated etiology (causation), that they should be removed from the compensation system. (National Commission on Workmen's Compensation Laws, 1973. *Compendium on Workmen's Compensation*. Washington: GPO.)

In addition to the commonly accepted factors such as age, smoking, diet, heredity, etc., there are some chemicals that have profound effects on the heart and cardiovascular system. Aniline and nitrobenzene are myocardial (heart muscle) depressants. Ethylene, chloroform, and trichloroethylene are myocardial irritants. The azides produce severe vasodilation. Carbon disulfide induces atherosclerosis.

Carbon monoxide, cyanide, and certain insecticides can have damaging effects on individuals with impaired cardiac function or reduced cardiac reserve. Pulmonary irritants such as ammonia, chlorine, phosgene, and sulfur dioxide can be quite hazardous to the person with heart impairment. Silicosis, asbestos, and other pneumoconioses may result in right heart failure (cor pulmonale). Heat, cold, and electrical shock can seriously affect the impaired heart.

Heart attacks seem to occur at a lower rate in workers than in the population at large. This may result, in part, from the fact that the American worker is "selected," that is, he often receives a preplacement medical examination to place him in a job that is compatible with his health and physical abilities. He may also receive periodic follow-up examinations at work to monitor his health.

One researcher (Paffenberger, the American Journal of Epidemiology) studied longshoremen. Those performing heavy work had a lower rate of sudden death than workers doing light work, suggesting that perhaps heavy work may help to prevent sudden deaths from coronary artery disease rather than to cause them. These conclusions have been confirmed by many other studies.

In almost all heart attacks that go to litigation, the problem is that of causation. To make a determination that an employee was subjected to a stressor "sufficient to bring about the heart attack or reaction," is extremely difficult because of the limitations of medical knowledge as to etiology. Rarely can a physician state that a heart attack is related to a particular stress, nor can he point with certainty to the initiating process of any heart attack. Although the presence of some atherosclerosis may be granted, it is not possible to predict when particular coronary vessels will occlude and precipitate the myocardial infarction or a fatal arrhythmia.

The physician does have a role in informing the court that the worker did indeed have a heart attack and in presenting substantiating data. A final judgment must be rendered in accordance with the administrative and adjudicatory framework of the state.

Several of the more troublesome areas concerned with determination of aggravation of preexisting conditions have been discussed above. Causation and the lack of positive medical knowledge about causation are the most important deficits in this determination. That a specific disease state can be caused or aggravated by more than one stressor is another important factor in determination, inasmuch as not all stressors can be identified. The other factors in the determination can be identified and quantified by experts; for example, factors related to genetics, physical characteristics, personal habits, work exposure, work habits, work processes, contaminants, age, and sex. To assist in arriving at a just decision, it is suggested that qualified medical and other professional advice should be obtained during the decision-making process. Consideration should be given to:

- a. Using this guide, and other material, as sources of information which should be obtained to help support opinions and decisions; and
- b. using the services of an impartial advisory board made up of occupational medical specialists and other physicians and industrial hygienists. Participants should be selected by state or local medical societies and professional organizations.

Other measures that may ultimately enhance the equitable handling of cases involving possible aggravation of disease are:

- a. Encourage research on the causes of chronic disease and the relative degree of contribution of various factors;**
- b. encourage research into the possibility of removing cases of aggravation from the "all or nothing" decision realm. While this approach has certain drawbacks, it may also make possible partial compensation for diseases not previously held compensable. (This is being done through second injury funds established in some states.)**
- c. encourage preventive medicine through preplacement medical examinations and job selection procedures to place workers in jobs which will not aggravate any of their preexisting health conditions.**

CHAPTER VI — VALIDITY OF TESTIMONY

Non-professional persons cannot be expected to collect and evaluate all of the information needed. In most cases, physicians will provide testimony on medical conditions and laboratory and other medical tests; industrial hygienists will testify concerning evidence of exposure; epidemiologists give testimony on epidemiologic data. These professionals must consider all pertinent points in their area of expertise in order to present an accurate and meaningful evaluation of the available data. The hearing examiner, board, commissioner, or officer should verify:

1. The professional qualifications of those testifying, and
2. the basis of the testimony, that is, the importance attributed to various areas of the information reviewed, and the conclusions that were drawn.

Medical

The phrase "competent medical person" is frequently used in both the lay and professional literature, including this guide. But what does it mean? Who is a competent medical person? Board certification (other than in occupational medicine) and academic status do not in themselves confer expertise in occupational disease. An expert in a specific medical field is not necessarily medically competent to render clinical judgment on an entire case, but only on that portion which is within his or her area of expertise. No rigid rules for judging competency can be defined. Because of the many variables, some guidelines are offered to aid the decision-maker in judging who is or might be considered a "competent medical person."

A competent medical person is:

- 1. A physician, judged competent in one of the several disciplines of medicine, and**
- 2. specially trained in the particular expertise required for the testimony to be presented. In determining occupational causation of disease, such expertise would include intimate knowledge of the work environment.**

For compensation purposes, a medical specialist—such as an internist, pathologist, surgeon, specialist in chest diseases, or an occupational health physician—is usually a competent medical person, but not in all instances.

For example, in a compensation case involving a question of occupational lung disease, the chest specialist can certainly use his or her expertise to diagnose a chest condition. But unless such a specialist is familiar with the work history and exposure of the employee, and has the background to coordinate and evaluate toxicological, epidemiologic, and industrial hygiene information in terms of the medical condition, that specialist should not be considered competent to render an expert opinion regarding the occupational origin of the disease condition.

Generally, a physician certified in occupational medicine is a competent medical person. Occasionally, however, the physician's particular work experience does not include an understanding of the exposure issues involved, such as carcinogenic factors. In the examples given, two physicians may be required to provide the expert opinion.

It is important for the medically competent person to maintain impartiality and to have an understanding of labor and industry. Almost all persons, medical and otherwise, who testify in compensation cases have some degree of bias. This does not invalidate their testimony. However, the examiner should consider the extent, nature, and effect, if any, of expert bias in arriving at his decision.

It is the duty and responsibility of a compensation hearing officer, lawyer, or any interested person to be aware of the requirements for medical competency in order to assure sound decisions. The following should be considered in judging medical competence:

- 1. Is the physician certified in occupational medicine by the American Board of Preventive Medicine?**

2. Is the medical expert's specialty directly related to the type of disease in question (cardiologist for heart disease, pulmonary specialist for lung disease, etc.)?
3. Does the physician have industrial experience? In what industries? Does this include experience in diagnosing the disease in question?
4. What is the expert's formal training in occupational medicine?

Exceptions: Although the competent medical person is a physician, there are some instances when the physician's testimony will be supplemented by testimony from a dentist, anatomist, toxicologist, occupational health nurse, or industrial hygienist concerning special health issues in their area of expertise. In such circumstances, these professionals are considered "competent experts" for the purposes of the particular adjudicatory proceedings. The testimony of such non-physicians should not be permitted to be substituted for the medical testimony of a physician. In addition, the qualifications of such individuals should be ascertained as is done in qualifying any expert in any court case.

Industrial Hygienist

According to the American Industrial Hygiene Association, a professional industrial hygienist is "a person possessing either a Baccalaureate Degree in Engineering, Chemistry, or Physics, or a Baccalaureate Degree in a closely related biological or physical science from an accredited college or university, who has, in addition, a minimum of three years of industrial hygiene experience. A completed Ph.D or Sc.D. in a related physical or biological science or an M.D. can be substituted for two years of the three year requirement." Further, it is suggested that all industrial hygienists consulted be professionally certified by examination of the American Board of Industrial Hygiene.

The following should be considered when judging an industrial hygienist's competence:

1. Is the industrial hygienist certified by the American Board of Industrial Hygiene or under the direction of a certified industrial hygienist?

2. Is the area of specialty of the industrial hygienist related to the evidence being given (comprehensive, engineering, toxicology, acoustics, air pollution, chemistry)?
3. Does the industrial hygienist have experience with the particular occupation involved?

Whenever possible, reports of past industrial hygiene studies pertinent to the case should be relied upon to provide basic environmental evidence. To be credible, personnel conducting industrial hygiene studies for use as evidence should be professionals trained in industrial hygiene or be under the direction of such professionals.

CHAPTER VII — CONCLUSIONS

Evidence presented by qualified professionals according to the method described in the preceding chapters will generally be sufficient for the hearing examiner to answer the following questions to his satisfaction:

1. Has a disease condition been clearly established?
2. Has it been shown that the disease can result from the suspected agent(s)?
3. Has exposure to the agent been demonstrated? (by work history, sampling data, expert opinion?)
4. Has exposure to the agent been shown to be of sufficient degree and/or duration to result in the disease condition? (by scientific literature, epidemiologic studies, special sampling, replication of work conditions?)
5. Has nonoccupational exposure to the agent been ruled out as a causative factor?
6. Have all special circumstances been weighed?

Occasionally, special circumstances must be considered. Were there any unusual events at work that reduced the effectiveness of protective equipment? Of ventilation? Of safe work practices? If the employee is a woman, are there special risks to women from exposure to the agent? If so, this factor must be evaluated.

7. Has the burden of proof been met—did the evidence prove that the disease resulted from, or was aggravated by, conditions at work?

If the answer to all of the above is "Yes," the decision can be made that the disease is occupational in origin.

CHAPTER VIII — EXAMPLES OF THE METHOD

The following text of the guide presents information on fourteen selected disease-producing agents to illustrate the use of the decision-making method previously described. The examples presented are antimony, inorganic arsenic, asbestos, benzene, carbon monoxide, coke oven emissions, cotton dust, inorganic lead, inorganic mercury, nitrogen dioxide, noise, crystalline silica, sulfur dioxide, and toluene diisocyanate.

Different and additional agents could have been presented, and consideration will be given to such publication if experience with the guide indicates a demand for such agent information. As a group, however, the above agents exemplify both acute and chronic effects. They represent different physical forms: Solid fibers, physical agents, particulates, fumes, and vapors. In their health effects these agents involve many organ systems: Respiratory system, central nervous system, hepatic system, genitourinary system, blood forming (hematopoietic) organs, and other systemic effects, as well as carcinogenic action. The organization of the agent material presented in the following pages can serve as a guide for collecting and recording pertinent information about other disease-producing agents.

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 and/or skin absorption (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

Chemical Name	Common Names
antimony pentachloride	antimonie chloride, antimony perchloride, butter of antimony
antimony pentafluoride	antimony fluoride
antimony pentaiodide	antimony iodide
antimony pentasulfide	antimonie sulfide, antimonial saffron, antimony red, antimony persulfide, antimony sulfide, golden antimony sulfide
antimony pentoxide	antimonie anhydride, antimonie acid, antimonie 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
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

Chemical Name	Common Names
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	cable splicers
antimony workers	ceramic makers
babbitt metal workers	compositors
battery workers, storage	copper refiners
brass founders	dye makers
britannia metal workers	electroplaters
bronzers	explosives makers
burnishers	fireworks makers
flameproofers	pewter workers
foundry workers	pharmaceutical workers
glass makers	phosphor makers
glaze dippers, pottery	pigment makers
gold refiners	plaster cast bronzers
insecticide makers	porcelain workers
insulators, wire	pottery workers
lake color makers	printers
lead burners	pyrotechnics workers
lead hardeners	rubber makers
lead shot workers	semiconductor workers
linotypers	solder makers
match makers	stereotypers
metal bronzers	stibnite miners
miners	storage battery workers
monotypers	textile dryers
mordanters	textile flameproofers
organic chemical	textile printers
synthesizers	type metal workers
paint makers	typesetters
painters	vulcanizers
perfume makers	zinc refiners

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

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), and
- 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, and
- 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 or constipation,
- headache,
- dizziness, and
- chest pain.

Respiratory symptoms are:

- Inflammation of the larynx (laryngitis),
- inflammation of the trachea (tracheitis),
- cough, and
- difficulty in breathing (dyspnea).

Antimony and its compounds are generally regarded as primary skin irritants. Lesions usually appear on exposed, moist area 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), and
- nodular ulcers on the neck and/or moist areas of the body, e.g., axilla or groin.

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), and
- 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 Evaluations

Additional data 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. Epidemiologic 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.

Renes³ 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 milligrams 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.

Taylor⁸ 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 Souchere⁹ reported 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 Luvoni¹⁰ reported 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 and
2. electrostatic precipitation.

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

1. Rhodamine B and
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, and
4. blood antimony levels in excess of 2.0 milligrams per 100 grams.

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).

Epidemiologic data (experience with groups of people) show a relationship between exposure to arsenic and 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, realgar, red arsenic, red arsenic sulfide, red arsenic glass, ruby arsenic
arsenic hemiselenide	
arsenic oxychloride	

Chemical Name	Common Names
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
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

Chemical Name	Common Names
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 orthoarsenate, 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

Chemical Name	Common Names
lead arsenate	acid lead arsenate, arsenate of lead, lead salt of arsenic acid, dibasic lead arsenate, lead orthoarsenate, plumbous arsenate
lead arsenite	lead m-arsenite, lead o-arsenite, lead metaarsenite
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 Exposure to Inorganic Arsenic

acetylene workers	electroplaters
acid dippers	electrolytic copper workers
alloy makers	enamellers
aniline color makers	enamel makers
aniline workers	etchers
arsenic workers	exterminators
artificial flower makers	farmers
babbitt metal workers	feather workers
bleaching powder makers	ferrosilicon workers
boiler operators	fertilizer makers
book binders	fireworks makers
brass makers	flypaper makers
bronze makers	galvanizers
bronzers	glass makers
cadmium workers	gold extractors

candle (colored) makers	gold refiners
canners	hair remover makers
carpet makers	herbicide makers
carroters, felt hat	hide preservers
cattle dip workers	ice makers
ceramic makers	illuminating gas workers
ceramic enamel workers	ink makers
commercial artists	insecticide makers
copper smelters	japan makers
crop dusters	japanners
defoliant applicators	jewelers
defoliant makers	lead burners
dimethyl sulfate makers	lead shot makers
disinfectant makers	lead smelters
drug makers	leather workers
dye makers	lime burners
metal cleaners	soda makers
metal refiners	soil sterilizer makers
miners	solderers
mordanters	submarine workers
nitrocellulose makers	sulfuric acid workers
ore smelters	tanners
organic chemical synthesizers	tar workers
paint makers	taxidermists
painters	textile printers
paper hangers	tinners
paper makers	tree sprayers
petroleum refinery workers	type metal workers
pharmaceutical makers	varnish makers
pigment makers	vine dressers
plastic workers	wallpaper printers
plumbers	warfare gas makers
preservative makers	water weed controllers
printing ink workers	weed sprayers
pyrotechnics workers	wine makers
rayon makers	wire drawers
rodenticide makers	wood preservative makers
sealing wax makers	wood preservers
semiconductor compound	zinc chloride makers
makers	
sheep dip workers	zinc miners
sign painters	zinc refiners
silver refiners	

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

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, and
- 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, and
- 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, and
- 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) and
- 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, and
- extreme general weakness.

Signs and symptoms which may occur later include:

- Nausea,
- vomiting, and
- 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), and
- 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), and
- 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, and
- 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, and
- 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, and
- pulmonary function 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 52. 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 epidemiologic 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

Butzengeiger¹³ reported a study of "180 vinedressers and cellar-men 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 precede 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, Butzengeiger¹⁴ 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 Fraumeni¹⁵ 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 Strong¹⁸ 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 and
- 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, and
- molybdenum blue method.

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

Allowable Exposure Limits

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 standard adopted by the Occupational Safety and Health Administration (OSHA) establishes a permissible exposure limit of 10 micrograms per cubic meter air. This represents the lowest level which OSHA believes is feasible. The existence of a safe level of exposure to inorganic arsenic has

not yet been demonstrated. In the absence of a demonstrated safe level, OSHA will not assume that one exists because of the irreversibility and long latency period for lung cancer.

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, and
3. analysis of urine (or nails) for arsenic is of value in confirming exposure but is not diagnostic in itself.

Asbestos

Introduction

Asbestos is a mineral fiber, and is the name given to about thirty silicate compounds. Of these, only the following 5 are of significance in industry:

Chrysotile (white asbestos)	Crocidolite (blue asbestos)
Amosite	Anthophyllite
Tremolite	

Chrysotile accounts for about 97 percent of all the asbestos used in this country.

Asbestos is widespread in the environment because of its extensive use in industry and the home. Over 3,000 products contain asbestos.

Because of this wide usage, it may be difficult at times to determine if a disease arising from asbestos is occupational in origin. For example, the air of some relatively new apartment buildings has been found to contain more asbestos fibers than the maximum recommended levels in industry. The source of the fibers in the apartment buildings is the insulating materials used in the ventilation system.

Exposure to asbestos can produce a lung fibrosis called *asbestosis*. The onset of asbestosis is usually gradual, developing over a period of 10 to 30 years of exposure to significant concentrations of asbestos. Occasionally, from very massive exposures, it may develop more quickly.

Asbestos is also a *cancer* producing agent (bronchogenic carcinoma, mesothelioma) and can cause certain specific skin diseases (asbestotic subcutaneous granulomatosis and asbestotic cutaneous verruca). Heavy exposure to dust containing asbestos can cause *skin irritation*. Epidemiologic studies (experience with groups of people) and animal studies have shown that increased exposure to any of the types of asbestos increases the risk of lung cancer (bronchial carcinoma). This carcinoma appears to be related to the degree of exposure to asbestos, the type of asbestos, and cigarette smoking. It is also

significant that cigarette smoking in men and women greatly increases the risk of lung cancer in those who are exposed to asbestos. Smoking is a factor that should be considered when determining whether lung cancer is caused, wholly or in part, by an occupational exposure to asbestos.

Mesothelioma, a rare malignant tumor of the membrane which lines the chest cavity and the abdominal cavity, is occurring with increasing frequency in workers with exposure to asbestos. The development of this tumor apparently is not related to the amount of asbestos inhaled and it is found in persons not having asbestosis. Levels of exposure which are within accepted standards for protection against asbestosis may not protect against mesothelioma.

An increased incidence of malignancy of the stomach and colon has been reported among insulation workers using asbestos.

Occupations with Potential Exposure to Asbestos

acoustical product makers	crushers (asbestos)
acoustical product installers	fiberizers (asbestos)
air filter makers	fireproofers
asbestos-cement products makers	firemen
asbestos-cement product users	furnace filter makers
asbestos-coating makers	gasket makers
asbestos-coatings users	heat resistant clothing makers
asbestos-grout makers	insulation workers
asbestos-grout users	inert filter media workers
asbestos-millboard makers	ironing board cover makers
asbestos-millboard users	laboratory hood installers
asbestos-mortar makers	ladders
asbestos-mortar users	paint makers
asbestos millers	pipe insulators
asbestos miners	plastics makers
asbestos-paper makers	pump packing makers
asbestos-paper users	roofers
asbestos-plaster makers	roofing materials makers
asbestos-plaster users	rubber compounders
asbestos sprayers	shingle makers

asbestos workers
asphalt mixers
automobile repair
garage workers
brake lining makers
building demolition workers
carders (asbestos)
caulking compound makers
caulking compound users
clutch facing makers
cobblers (asbestos)
construction workers

ship builders
ship demolition workers
spinners (asbestos)
talc miners
talc workers
textile flameproofers
textile workers
undercoaters
vinyl-asbestos tile makers
vinyl-asbestos tile installers
weavers (asbestos)

Medical Evaluation and Differential Diagnosis

(Also, See Decision-Making Process)

In addition to the usual medical history, the following should be considered:

1. Any history of diseases of the heart or lung or abnormal tissue growth should be carefully evaluated to determine the relationship between the previous disease and the claimant's present condition.
2. A respiratory questionnaire, a sample of which is shown in Appendix C, can be useful in evaluating the extent and importance of respiratory symptoms such as:
 - Breathlessness,
 - phlegm (sputum) production,
 - chest pain,
 - cough, and
 - wheezing.

Asbestosis

Shortness of breath upon exertion is usually the first symptom, frequently accompanied by a dry cough. This symptom develops after several years of progressive pulmonary fibrosis. As asbestosis progresses, the following signs and symptoms are observed:

- Cough with production of sputum,
- anorexia (loss of appetite),

- secondary respiratory infections that are difficult to control,
- rapid breathing,
- repetitive end-inspiratory crackles (crackling sounds heard in the lower part of the lungs through stethoscope when employee completes each of a series of inhaled breaths),
- orthopnea (breathing difficulty in a recumbent position),
- cyanosis (change in skin color to bluish, grayish, slate-like or dark purple),
- decrease of chest expansion,
- digital clubbing (rounding of the ends, and swelling of the fingers and/or toes), and
- sequelae (other resultant diseases) including cor pulmonale (right heart failure), bronchogenic carcinoma (lung cancer), stomach or intestinal cancer, or pleural carcinoma (cancer of the membrane lining the chest).

Fibrosis results in alveolo-capillary block (impaired ability of the lungs to transfer oxygen into the blood). This impairment is often more severe than is indicated by chest X-rays.

Mesothelioma

In cases of mesothelioma, the rare malignancy noted above, there may be a long latent period, as much as 40 years, between initial exposure to asbestos and the development of the tumor.

Mesothelioma of the *peritoneum* (membrane surrounding the abdominal organs) is usually accompanied by abdominal swelling and pain that is not concentrated in a particular area. Signs and symptoms of this type of tumor (which *may* be associated with asbestos exposure) include:

- Weight loss,
- obstruction of the bowel, and
- excessive accumulation of fluid in the abdominal cavity (ascites) is almost always present.

This malignant tumor of the peritoneum may spread to the chest cavity.

With mesothelioma of the *pleura*, complaints include chest pain and breathlessness. Signs and symptoms of pleural mesothelioma include:

- Pleural effusion (accumulation of fluid in the space around the lungs),
- the tumor may grow outward through the chest wall in the form of a lump beneath the skin (subcutaneous lump),
- the tumor may spread to involve bone, lymph glands (nodes), mediastinum (area between the right and left lungs), and pericardium (the sac enclosing the heart). As a result, the supraclavicular nodes may become enlarged, ribs may develop tumors, and obstruction of the superior vena cava (major vein draining the upper portion of the body) may occur, and
- in addition, pericardial effusion (fluid in the heart cavity) may occur, causing tamponade (acute compression of the heart).

Laboratory and Clinical Examinations

(See Decision-Making Process)

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

Chest X-rays

Findings should be classified according to the ILO/UC 1971 Classification of the Radiographs of the Pneumoconioses.

Findings for asbestosis vary, but the usual picture shows a density in both lungs, with the lower one-third of the lungs involved. In the affected area there is a "ground glass" appearance.

As asbestosis progresses, more and more of the lung is involved, except the apices (tips of the lungs). The X-rays will show gradual obscuring of the border between the lungs and the diaphragm. It may show shadows from the presence of nodules.

X-ray findings usually will show the following as the asbestosis progresses:

- Reduced radiographic volume and
- formation of cysts combined with increased size of the heart, dilation (enlargement) of the proximal pulmonary arteries (arteries which lead from the heart to the lungs).

Lung Function Tests

Reduced lung capacities and other lung changes do not differ from those resulting from other forms of lung fibrosis, both occupational and nonoccupational. Therefore, the results of lung function tests alone or chest X-ray findings alone do not lead to diagnosis of asbestosis. Asbestos bodies in lymph nodes indicate exposure, but not necessarily asbestosis.

- Asbestosis causes a reduction in the vital capacity (VC) of the lungs and a reduction in total lung capacity (TLC). These capacities are further reduced as the disease progresses.
- The residual volume (RV) of the lungs will be normal or slightly increased.
- The lungs' diffusing capacity for carbon monoxide (D_L) will be reduced.

Other lung function test results which are found in asbestosis include:

- Increased minute ventilation (amount of air breathed in one minute),
- reduced oxygenation of the arterial blood (arterial hypoxemia),
- increased static transpulmonary pressures, and
- decreased lung compliance.

An exercise test will result in an increased amount of air required during physical effort and decreased oxygen in the blood, leading to cyanosis.

Sputum Examination

Asbestos fibers or bodies may be found in the sputum. These indicate asbestos exposure, but not necessarily asbestosis. Where cancer cells are present in the sputum, and chest X-ray findings are normal, bronchoscopy may be necessary to confirm and locate the lung tumor.

Skin Tests

The following tests should be performed by the physician to exclude possible infectious diseases:

1. PPD (tuberculin test)
2. blastomycin
3. histoplasmin
4. coccidioidin

Epidemiology

Various epidemiologic studies have demonstrated the relationship between asbestos and lung disease, including mesothelioma, in such trades and occupations as mining, insulation installation, textiles, paint, electrical industries, and many other occupations as a result of the widespread use of this substance.

The available information indicates evidence of a dose-response relationship for asbestos exposure and the risk of asbestosis and/or bronchogenic carcinoma. However, much of this information is epidemiologic in nature and there is little correlation between epidemiologic data and environmental exposure data. For this reason and others, including the long latent period for the development of carcinomas, it is difficult to develop a specific dose-response relationship. This should be taken into consideration when referring to the following material:

Enterline¹⁹ has reported an exposure-response relationship between asbestos exposure (evaluated as millions of particles per cubic foot years) and the risk of malignant and nonmalignant respiratory disease. Enterline's data indicate that the risk of respiratory cancer increased from 166.7 (standardized mortality ratio) at minimum exposure to 555.6 at cumulative exposures exceeding 750 million particles per cubic foot years. Enterline's data is summarized in a table by NIOSH²⁰.

Murphy²¹ reported that asbestosis was 11 times more common among pipe coverers in new ship construction than in a control group. The first asbestosis was found after 13 years of exposure to an estimated cumulative dose of about 60 million particles per cubic foot years. After 20 years, asbestosis prevalence was 38%. Murphy reported no asbestosis for men exposed to 60 mppcf years but 20% asbestosis in men exposed to 75-100 mppcf years. Murphy reports atmospheric dust concentrations ranged from 0.8 - 10.0 mppcf depending on the different operations evaluated. Asbestosis was considered present if the worker had at least three of the following: Vascular rales in two or more sites, clubbing of the fingers, vital capacity of less than 80% predicted, roentgenography consistent with moderately advanced or advanced asbestosis, and shortness of breath on climbing one flight of stairs.

The Pennsylvania Department of Health²² reported a study of asbestos dust concentrations in two plants (one from 1930-1967 and the other from 1948-1968). Sixty-four cases of asbestosis were reported. In the two plants, the study indicates that the air concentrations of particulates were generally less than five mppcf and in many cases less than two mppcf.

Epidemiologic evidence is also available relating the development of mesothelioma with exposure to asbestos. Selikoff^{23,24} reported 14 deaths from mesothelioma in 532 asbestos insulation workers from 1943-1968. No deaths from mesothelioma would be expected from the same number of individuals in the general population.

Evidence of Exposure

Historically, there have been two air sampling and analysis methods to determine the quantity of asbestos in the workplace environment. The earlier light field impinger count method allowed only a measure of the overall dust level in the air rather than focusing on the amount of asbestos fibers in the air. The current fiber count method satisfactorily determines the amount of asbestos fibers in the air. It is performed by collecting airborne materials on a membrane filter and then counting the fibers using a phase contrast microscope at a 400 to 450 times magnification ratio (400X-450X).

Asbestos fibers occur in varying lengths and diameters. As of the publication of the guide, the Occupational Safety and Health Administration (OSHA) establishes maximum allowable limits for asbestos fibers greater than five micrometers (um) in length. OSHA limits such asbestos fibers to no more than 2 fibers per cubic centimeter of air (based on an eight-hour time-weighted average exposure).

OSHA further requires that no workers be exposed to more than 10 asbestos fibers (greater than five um in length) during any one 15-minute period of time.

For samples collected by the field impinger count method, results may be compared to the pre-1970 limit (TLV) of five million particles per cubic foot of air.

Occupational exposure to asbestos fibers five um in length or greater, at quantities averaging more than 2 fibers per cubic centimeter of air or frequent exposures to more than 10 such fibers during a 15-minute period of time is evidence of a possible causal relationship between disease and occupation.

Toxicological

(See References 19-24 Appendix A)

Conclusion

The diagnosis of occupational asbestosis is based on meeting the following criteria:

1. Confirmed history of occupational exposure to asbestos.
2. X-ray findings compatible with those indicating asbestosis according to ILO/UC 1971 "Classification of Radiographs of the Pneumoconioses."
3. Pulmonary impairment, particularly a decrease in lung diffusing capacity and an increase in alveolar-arterial oxygen difference, as demonstrated by lung function tests.

The diagnosis of occupational mesothelioma is based on meeting the following criteria:

1. Confirmed history of occupational exposure to asbestos.
2. Pathological evidence of mesothelioma.

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 dermatitis.

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

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
enamellers
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 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, 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 hematological 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 Schwanitz²⁹ 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 Greenberg³⁰ 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 Elkins³¹ 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."

Juzwiak³² 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)

Sherwood³⁴ 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 $\frac{1}{2}$ 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). 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 carboxy-hemoglobin, 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 equivalent 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
enamellers
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
- drowsiness
- 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)

Additional 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. NIOSH³⁵ 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 BLOOD³⁶

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. McFarland³⁷ reported difficulties in visual discrimination at 5% COHb (similar results were reported by Halperin³⁸). Horvath³⁹ reported significantly impaired vigilance at 6.6% COHb. Schulte⁴⁰ 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. Trouton⁴³ 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. NIOSH⁴⁴ 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 (carboxy-hemoglobin) 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 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 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.

Additional 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

Doll⁴⁶ reported an "81% excess of lung cancer deaths among gas works pensioners (gas retort workers) in comparison with the general population." Lloyd⁴⁷ reported that "coke oven workers had an average lung cancer mortality rate of 2½ times that predicted by the experience of all steelworkers."

Lloyd⁴⁷ 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 America⁵⁰ 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. Chromatography,
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 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, 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)	openers (cotton mill)
carders (cotton mill)	pickers (cotton mill)
carding machine operators (cotton mill)	press box operators (cotton mill)
cleaner operators (cotton mill)	roving frame operators (cotton mill)
cleaners (cotton mill)	slashing operators (cotton mill)
combining machine operator (cotton mill)	spindle pickers (cotton)
drawing frame operators (cotton mill)	spinners (cotton mill)
dryer operators (cotton mill)	spooling operators (cotton mill)
gin stand operators (cotton gin)	stripper operators (cotton)
ginners	stripper operators (cotton mill)
grinders (cotton mill)	twisters (cotton mill)
handpickers (cotton)	weavers (cotton mill)
lint cleaner operators (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 (FEV₁) has been devised to classify ventilatory impairment. The mean of the two highest values of FEV₁ 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; FEV₁ 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; FEV₁ 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; FEV₁ is greater than 80% of the predicted value,
- Function (F) 2: evidence of a slight to moderate irreversible impairment of ventilatory capacity; FEV₁ is 60 to 79% of the predicted value, and
- Function (F) 3: evidence of a moderate to severe irreversible impairment of ventilatory capacity; FEV₁ 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 Higgins⁵¹ 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 (FEV₁). 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_{1,8}).

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.

Shilling⁵³ 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) F (36)	2	5	2	9 (15%)
	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 FEV₁ decrease of 0.82 liter. In 6 men in both mills who had worked for less than 1 year, the reduction of FEV₁ on the first working day of the week ranged from 0.09 to 0.43 liter. FEV₁ 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"		Total		
		Range	Mean	Range	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 Gullett⁵⁵ 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 FEV₁ 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 FEV₁ would not identify all symptomatic workers.

Molyneux and Tombleson⁵⁶ 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 Suh⁵⁷ 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 FEV₁ 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 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)

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).

Inorganic Lead

Introduction

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

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

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

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

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

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

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

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

Lead and its compounds have numerous chemical and common names:

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

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

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

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

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

Occupations with Potential Exposures to Inorganic Lead

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

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

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

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

The following should be considered:

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

Nonoccupational Exposure

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

Common nonoccupational lead exposure sources include:

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

Signs and Symptoms

The early signs and symptoms of lead poisoning are non-specific and may resemble many diseases including influenza.

Early signs and symptoms are:

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

These symptoms are reversible, and complete recovery is possible.

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

Central nervous system symptoms are:

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

Gastrointestinal symptoms are:

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

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

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

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

- Basophilic stippling and
- deformed nuclei.

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

Kidney (renal) symptoms are:

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

Additional signs and symptoms which may be present are:

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

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

Laboratory and Clinical Examinations

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

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

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

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

Blood

- decreased hemoglobin — less than 13 gram %
- increased blood lead (PbB) — greater than 60 to 80 micrograms per deciliter
- decreased red blood count
- stippled basophilia and reticulocytosis
- increased free 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.⁵⁹ reported a study of subacute low level exposure to lead which occurred when a demolition company dismantled an elevated train network. The old steel structure was heavily coated with several coats of lead paint. Respirators were in use. Data from the report are presented in the following table.

LABORATORY AND CLINICAL DATA FROM 44 DEMOLITION WORKERS, BY EXPOSURE							
Group	Number of Workers	Average Lead Exposure (milligram per cubic meter of air)	Blood Lead (microgram per 100 grams)		Mean Hematocrit (%)	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 ^e
Nonburners	12	0.23	24-75	48.8	44.0	714 ^d	49.0 ^e
Normal Value						46.9 ± 14.9	54.09 ± 5.96

(Feldman et al., 1977)

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

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

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

Elkins⁶¹ assembled data available on lead in air and lead in urine and reported that a urinary lead level of 0.2 milligrams lead per liter of urine would correspond to an air concentration of 0.2 milligrams lead per cubic meter of air.

The data in the table on the next page relating average and median blood lead content with exposure and duration of employment have been adapted from Dreesen et al.⁶², the Committee on Biological Effects of Atmospheric Pollutants,⁶³ and the National Institute for Occupational Safety and Health⁶⁰.

Tola and Nordman⁶⁴ reported a study of 335 men representing the general population and 2,209 men occupationally exposed to lead. No association between blood lead concentrations and smoking was demonstrated in the men from the general population. A dose-response relationship was found between the amount of smoking and the blood lead concentrations of workers occupationally exposed to lead with smokers having statistically significant higher blood lead levels than nonsmokers.

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

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

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

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

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

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

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

(Lancranjan et al., 1975)

Cooper and Gaffey⁶⁷ reported a mortality study of 7,032 men who had been employed in 6 lead production facilities and 10 battery plants for 1 or more years over a 23 year period. Lead absorption in many of the men was greatly in excess of currently acceptable standards based upon urinary and blood lead concentrations available for a portion of the group. Although the workers had high levels of exposure, only small deviations from expected mortality were reported. Cooper and Gaffey predicted no detectable effect on the mortality of male adults from occupational exposure to lead controlled in conformity to currently recommended environmental and biological standards⁶⁰.

Evidence of Exposure

Air Sampling and Analysis

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

1. Electrostatic precipitator and
2. impingement.

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

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

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

Allowable Exposure Limits

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

Conclusion

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

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

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

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

Inorganic Mercury

Introduction

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

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

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

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

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

Mercury and its compounds have numerous chemical and common names:

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

mercuric-cuprous iodide	copper-mercury iodide, mercury-copper iodide
mercuric cyanate	fulminate of mercury, mercuric fulminate, mercury fulminate
mercuric cyanide	mercury cyanide
mercuric dichromate	mercuric dichromate, mercury bichromate
mercuric fluoride	mercury fluoride
mercuric fluorsilicate	
mercuric iodate	mercury iodate
mercuric iodide	mercuric biniodide, mercury biniodide, yellow mercury iodide, red mercuric iodide, red mercury iodide
mercuric iodide ammonobasic	
mercuric iodide, aquobasic- ammonobasic	
mercuric iodide diammine	
mercuric nitrate	mercury nitrate, mercury perni- trate, 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, trimeric 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 iodohydrogyrate
mercuric potassium thiosulfate	
mercuric salicylate	mercury subsalicylate, salicylated mercury
mercuric selenide	
mercuric sesquiodide	
mercuric silver iodide	mercury-silver iodide, silver-mercury iodide
mercuric subsulfate	basic mercuric sulfate, mercuric dioxysulfate, turbith mineral, turpeth mineral
mercuric sulfate	mercury bisulfate, mercury persulfate, mercury sulfate, mercury salt of sulfuric acid

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

mercurous iodate	
mercurous iodide	mercury iodide, mercury protoiodide, yellow mercury iodide
mercurous monohydrogen-o-arsenate	
mercurous nitrate	hydrated mercurous nitrate, mercury nitrate, mercury salt of nitric acid
mercurous nitrate, ammoniated	ammoniated mercury nitrate, black precipitate, Hahnemann's soluble mercury
mercurous nitrite	
mercurous oxalate	
mercurous oxide	black mercurous oxide
mercurous phosphate	neutral mercurous phosphate, normal mercurous phosphate, mercury phosphate, tertiary mercurous phosphate, trimercurous orthophosphate
mercurous sulfate	mercury sulfate
mercurous sulfide	
mercury	hydrargyrum, quick silver
mercury ammoniated	aminomercuric chloride, ammono-basic mercuric chloride, ammoniated mercuric chloride, ammoniated mercury chloride, ammoniated mercury, fusible white precipitate, Lamery's white precipitate, 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	farmers
bactericide makers	feltmakers
barometer makers	fingerprint detectors
battery makers, mercury	fireworks makers
boiler makers	fungicide makers
bronzers	fur preservers
calibration instrument makers	fur processors
cap loaders, percussion	gold extractors
carbon brush makers	hatters
caustic soda makers	histology technicians
ceramic workers	ink makers
chlorine makers	insecticide makers
cinnabar ore processors	investment casting workers
commercial artists	jewelers
dental amalgam makers	laboratory workers, blood
dentists	laboratory workers, chemical
direct current meter workers	lamp makers, fluorescent
disinfectant makers	lamp makers, mercury arc
disinfectors	manometer makers
drug makers	mercury workers
dye makers	miners, mercury
electric apparatus makers	neon light makers
electroplaters	paint makers
embalmers	paper makers
explosive makers	percussion cap makers
pharmaceutical workers	pesticide workers
photoengravers	tanners
photographers	taxidermists
pressure gage makers	textile printers
refiners, mercury	thermometer makers
seed handlers	vacuum pump makers
sign painters	vapor tube makers
silver extractors	vinyl chloride makers
switch makers, mercury	wood preservative workers

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

The following should be considered:

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

Nonoccupational Exposure

Potential nonoccupational sources of mercury include:

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

Signs and Symptoms

Acute Poisoning

Acute industrial toxicity is rare, and associated signs and symptoms are:

- Inflammation of the gums (gingivitis),
- salivation,
- loss of teeth,
- gastrointestinal upset (diarrhea),
- kidney failure (toxic nephrosis),
- cardiac function abnormalities, and
- tremor may exist as an isolated finding.

Chronic Poisoning

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

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

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

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

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

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

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

Central nervous system symptoms are:

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

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

Signs and symptoms associated with the eye are:

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

Ear symptoms are:

- Possible loss of hearing,
- vertigo, and
- hypo-excitability of vestibular function (middle ear insensitivity).

Skin symptoms are:

- A pallor that is unassociated with anemia and
- allergic hypersensitivity may occur.

Laboratory and Clinical Exposures

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

Blood

- excess lymph cells (lymphocytosis)
- increase in the number of cells that stain readily with the acid stain eosin (eosinophil count increase)
- electrophoretic pattern of serum proteins are consistent with nephrosis
- increased blood urea in nitrogen (BUN)
- increased 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 (rena) injury and decreased urinary excretion. Also, urinary mercury may be increased in workers exposed to mercury, but who may or may not exhibit symptomatology. However, urinary values are useful guides to early exposure.

Epidemiology

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

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

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

Shandar and Simson⁶⁹ 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 Seligman⁷⁰ reported a study of 9 mercury mine and mill locations. Average work area air concentrations between 0.08 and 0.73 milligrams of mercury per cubic meter were associated with clinical evidence of mercury poisoning found in 18 out of 83 workers examined. Symptoms included loss of teeth, sore gums, loose teeth, salivation, headaches, personality changes, and tremor. Workers exposed to average air concentrations less than 0.03 milligram of mercury per cubic meter displayed no symptoms.

The following table relating symptoms and exposure data has been adopted from Turrian et al.⁷¹ and NIOSH⁷²:

SYMPTOMS OBSERVED IN 58 MERCURY WORKERS			
	Air Concentration (milligrams of mercury per cubic meter)		
	0.01-0.06	0.05-0.23	0.3-0.6
Number of workers	26	15	17
Average exposure, years	9.1	16.7	7.4
Tremor	19%	20%	29%
Erethism	8%	33%	29%
Impaired memory	0%	13%	18%
Demographia	8%	27%	18%
Gingivitis	42%	40%	35%
Bad teeth or dentures	46%	67%	41%

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

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

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

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

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

**RELATIONSHIP OF MERCURY EXPOSURE
TO BLOOD MERCURY LEVELS***

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

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

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

**Time-weighted averages

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

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

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

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

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

**Time-weighted averages

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

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

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

Evidence of Exposure

Air Sampling and Analysis

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

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

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

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

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

Allowable Exposure Limits

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

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

Conclusion

Diagnosis of occupational mercury poisoning is based on the following:

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

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

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 nitrogen⁷⁵. This chapter is concerned with the chemical agent nitrogen dioxide (NO₂) which exists in equilibrium with nitrogen tetroxide (N₂O₄) 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
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
jewelry makers
lacquer makers
lithographers

auto garage workers
auto painters
blueprinters
brass cleaners
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

(See also Decision-Making Process)

The following should be considered:

- smoking history,
- periodic chest X-rays,
- periodic pulmonary function tests such as 1 second forced expiratory volume (FEV₁) and forced vital capacity (FVC), and
- methemoglobin studies.

The differential diagnosis includes:

- Pneumonia,
- acute bronchitis, and
- X-ray findings may mimic tuberculosis.

A respiratory questionnaire (Appendix C) can be useful in evaluating the extent and importance of respiratory symptoms such as:

- Breathlessness,
- sputum production,
- chest pain,
- cough, and
- 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, and
- use of a gas heater or stove.

Air pollution may also be a factor, 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 increased 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, and
- 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 data that will assist in arriving at a correct 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, and
- 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 or 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.

Muller⁷⁷ 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 (FEV₁) 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 Fatigue, moderate dyspnea Irritating cough Temperature Severe dyspnea 24 days hospitalization
2	none	none (gradual worsening of respiration)

3	18 days	Fatigue, dyspnea Irritating cough Temperature Suffocation 18 days hospitalization
4	4 days	Temperature Bronchitis and cough Moderate dyspnea a few days sick at home
5	2-1/2 days	Sanguinolent sputum Feeling of suffocation Acute dyspnea 39 days hospitalization
6	none	none
7	14 hours	Pressure in chest Vomiting Dyspnea 1 week sick at home

(Muller, 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-response 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.⁷⁸ 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 Bockman⁷⁹ 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 firemen who experienced every phase of the illness including the eventual development of chronic pulmonary insufficiency follow:

PULMONARY FUNCTION DATA

	Predicted Normal	Date		
		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
FEV₁^b (% of vital capacity)	79	51	61	70
FEV₃^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 ₂ O)	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
Pao₂^d (mm Hg) air	90-100	75	75	55
100% oxygen	550	560	550	550
Paco₂^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 ½ 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:⁸⁰

**PROBABLE RESULT OF SINGLE
EXPOSURE TO NITROGEN DIOXIDE**

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

(Emergency Exposure Limits, *AIHA J*, 1964)

Lowry and Schuman⁸¹ 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 4,000 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 and
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, and
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 Administration (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, and
7. pulmonary function tests.

X-ray findings and lung function tests 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.

Noise

Introduction

Occupational hearing loss is a slowly induced deafness produced by loud sound in the workplace, over a period of time varying from months to years. Hearing loss may also be immediate, such as that caused by a sudden, loud explosion.

Exposure to intense noise for an extended period of time causes hearing loss which is either temporary, permanent, or a combination. Hearing loss is referred to as temporary threshold shift (TTS) or permanent threshold shift (PTS).

Temporary hearing loss means that the person's ability to hear will return to normal when he is absent from the source of the noise for a period of time. In cases of permanent hearing loss, there is never a return of hearing to the previous threshold.

Disability from hearing loss results from the decreased ability to identify spoken words or sentences. Speech is composed of frequencies between the range of 250 and 3,000 Hertz (Hz). Hertz is a unit of measurement of the frequency, sometimes referred to as cycles per second (cps).

The hearing level for speech is a simple arithmetic average of the hearing levels at frequencies of 500, 1,000, and 2,000 Hz. (Sataloff, J.; and Michael, P. 1973. *Hearing Conversation* Springfield, Illinois: Charles C. Thomas Co.) Healthy young ears are able to hear sounds through the frequency range from 20 to 20,000 Hz.

Hearing loss from repeated exposure to excessively loud noise usually occurs in the 4,000 Hz. area. Since this is above the frequency range of the normal spoken voice, an individual may suffer a decrease in hearing and not be aware of it.

A person's ability to hear high frequencies decreases with age just as his ability to read fine print decreases with age. The hearing deficiency is called presbycusis. The effects of age on hearing and vision are not the same for all individuals. This adds to the problem of determining if a hearing loss is occupational in origin, or the result of the aging process. However,

presbycusis tends to start in the 8,000 Hz. frequencies, whereas hearing loss due to noise is usually in the 4,000 to 6,000 Hz. range. Recruitment is present in early cases of deafness due to excessive noise, but not in presbycusis. Recruitment is the inability to understand speech in the presence of surrounding noise. The louder the words are spoken, the more difficult it is to understand them. Noise induced hearing loss usually is bilateral (exists in both ears).

In cases of occupational hearing loss, any accompanying hearing loss due to presbycusis is usually accounted for by allowing a reduction of ½ decibel (dB) for each year of age over the age of 40. The decibel (dB) is a unit for measuring the loudness or intensity of sound. For example, the sound pressure level (loudness) of conversation is between 60 or 70 dBA, a compressor is in the range of 120 dBA, and a turbojet engine 160 dBA. Because noise is not one frequency but is composed of a mixture of many frequencies, the so-called A-weighted technique is used for measurement of intensity. It is an average of the intensity of the different frequencies and is expressed as dBA.

Excessive noise can cause physiological problems other than hearing loss. It can have an effect on emotions, produce irritability, increase blood pressure and heart rate, and produce nausea. These effects on the worker in a noisy environment are not well defined as an occupational illness, but may have an affect on the quality and efficiency of the work performed.

Occupations With Potential Exposures to Noise

boiler rooms	paper manufacture
chemical products manufacture	paper products manufacture
construction	petroleum refining
corrugated paper manufacture	plastics manufacture
demolition	plastic products manufacture
earth moving equipment operators	power plant operators
electrical equipment manufacture	printing
engine rooms	primary metal processing
fabricated metal product manufacture	quarrying
farm equipment operators	rubber manufacture
food processing	rubber products manufacture
	shipbuilding
	steel making
	stone products industries (cement mills)

foundries
furniture manufacture
glass manufacture
lumbering
metal forming
metal machining
metal working
mining — open pit
mining — underground
ordinance manufacturing

stone workers
textile manufacture
transportation equipment
operators
trucking
tunneling
wood products manufacture

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

In the Medical History, the following should be considered:

1. Any previous history of diseases or injury involving the auditory nerve, capable of causing hearing loss, either as a direct result of disease or injury, should be evaluated to determine if present findings are associated with previous disease or injury.
2. In cases of possible occupationally induced hearing loss, it is important to evaluate the claimant's medical history pertaining specifically to diseases and conditions of the ear and auditory nerve. Included are the following considerations:
 - Previous ear trouble and disease,
 - extent of known hearing loss,
 - dizziness,
 - tinnitus (ringing in the ears),
 - treatment with drugs (ototoxic drugs),
 - head injury, and
 - estimate of subject's own hearing ability.

In the occupational history, consider also that exposure to noise may be from a hobby or from home activities. Included are the following:

- Woodworking,
- metal working,
- loud music in any form from any source,

- auto repair,
- operating noisy equipment (tractors, lawn mowers, etc.),
- traffic,
- pistol, rifle, or shotgun firing,
- auto racing, and
- operating motorcycles, snowmobiles, or boats.

Signs and Symptoms

Early signs of hearing loss are:

- Inability to understand spoken words in a noisy environment,
- need to look at the person speaking to understand words,
- familiar music may not sound the same, and
- changes occur in routine audiometric examination.

NOTE: The American Academy of Ophthalmology and Otolaryngology *Guide for Determining Hearing Impairment* is a useful guide for assessing handicaps due to hearing loss.

Laboratory and Clinical Evaluations

Other Tests

A thorough clinical examination of the ear should include the following:

- External ear examination for scars or malfunctions,
- otoscopic examinations of ear drum (typanic membrane) for any abnormalities,
- examination of nose, throat, and nasopharynx for any abnormalities,
- eye reflexes are noted (pupil and cornea),
- examination with tuning fork,
- pure tone audiometric examination,
- bone conduction studies,
- speech reception testing for threshold and discrimination,
- recruitment and tone decay studies, and
- other tests may be conducted.

If baseline and/or periodic audiometric examinations were conducted by the employer, these test results should be obtained for comparison with present audiometric test results.

In addition, the following should be considered:

The audiometric (pure tone) examination is one of the best clinical means of measuring hearing loss, although other examinations as referred to above should also be completed. The audiometric examination should be administered only by trained, competent personnel, and the test results interpreted by a competent otologist or audiologist.

The frequencies monitored by audiometry should cover the range of 250 Hz through 8,000 Hz. Factors which may alter audiometric test results include the following:

- Faulty or maladjusted equipment,
- inaccurate or misunderstood instructions from the test operator,
- wax in the ears,
- head cold or allergy, and
- exposure to intense noise 18 hours or less prior to the test.

Epidemiology

The sources of hearing loss and other auditory damage are well documented in the scientific literature, and many studies have shown the levels and durations of noise that are liable to cause such effects.

The following reports of dose-response relationships are taken from NIOSH⁸². NIOSH summarized audiometric surveys carried out between 1960-1970 in the United States and other countries. The following sections in quotes are all from NIOSH:

Coles and Knight⁸³ reported a study of workers in diesel-engine testing. "Maximum noise level 116 dB. Of six men who worked continuously in the intense noise of the two-stroke test-house (average period 3½ years) all had losses of 45-60 dB in one or both ears at 3.4 and 6 KHz, and none could be accounted for by an aging factor."

Yaffe and Jones⁸⁴ reported a study of Federal penitentiary workers (textiles, wood products, sheet metal, brush, shoe and clothing manufacture, and printing) where octave band noise levels ranged from 75-110 dB. "Those levels which exceeded octave band criteria produced significant hearing threshold shifts at 3, 4, and 6 KHz after 24 months exposure. The locations producing the largest shifts were cotton mill twist and weaving departments, woolen mill weaving departments, and furniture mills."

Schneider⁸⁵ reported a study of 294 jobs in chemical works involving 691 individuals. "Data divided into 4 noise exposure groups based on octave band criteria indicated that the group exceeding criteria more than 10% of the time experienced a permanent threshold shift of 1 dB per year at 2, 3, and 4 KHz. For the group near criteria exposure, most of the hearing loss occurred within the first five or so years."

Brohm and Zlamal⁸⁶ reported a study of noise in the cabs of heavy trucks ranging from 90-110 dB. Examinations were made of 51 truck drivers and in each case a loss of hearing was determined."

Mancini and Stancari⁸⁷ reported a study of 50 fettlers. "Men working in 9 foundries with noise levels of 92-100 dB. In men who had been working for more than 5-6 years in noisy conditions almost all frequencies were involved; those who had worked less than 2-3 months in noisy conditions showed a loss varying from 30 to 50 dB at 400 Hz." Chadwick⁸⁸ reported a study of 12 men exposed to noise from industrial gas-turbine engine noise. "Noise levels reached as high as 113 dB flat... the low-tone loss in just over two years was in the region of 10 dB and from 2,000-4,000 Hz was in the order of 20 dB... the average loss for the speech frequencies was... eight times more than that to be expected in a more conventional industry with a known noise hazard.

Filin⁸⁹ reported a study of drivers of self-propelled jumbos in underground ore mining. "Noise levels of 127 dB at frequencies between 1,000 Hz and 8,000 Hz. Hearing loss in 91 of 135 miners examined; after 10 years' work, 28 dB loss at 4,000 Hz."

Weston⁹⁰ reported a study of agricultural tractor drivers. "53 drivers of tractors of different horsepower; audiograms showed greater impairment in inland drivers where the tractors are of

higher power and exposure is for longer periods than on coast-plain farms. Noise levels ranged from 92 dB to 106 dB, occasionally as high as 114 dB."

Cohen⁹¹ reported a study where "hearing levels for heavy earth-moving equipment operators, paper bag workers, and airport ramp workers were compared with those of non-noise exposed groups. Noise encountered ranged from 80-120 dB (A-weighted sound level). The hearing loss levels of the heavy earth equipment operators were found to be significantly higher than the non-noise exposed groups. The paper bag workers had higher hearing loss levels but not as high as the earth equipment operators. The airport ramp personnel, however, had the lowest hearing loss levels, probably due to the intermittency of their exposures.

Burns⁹² reported a study of 759 employees in 32 various industrial factories with noise levels ranging from 78 to 109 dBA. "A relationship between noise level, exposure duration and hearing level was defined with two parameters: audiometric frequency and percentage of persons expected to exceed a specific hearing level. A-weighted sound level was found to be adequate for estimating hearing level for the industrial noises measured."

Stone⁹³ reported a study of "3,116 employees of 9 steam electric generating plants and 2 hydroelectric plants were tested. Noise levels from assorted equipment ranged from 91 to 127 dBA, the more intense values associated with coal hoppers, turbine generators and pumps, and forced draft fans. Prevalence of hearing impairment (defined by hearing levels averaging more than 15 dB (reASA 1951) at test frequencies of 0.5, 1, and 2 KHz) varied from 4.7 percent for the younger workers having less than two years of service to 31.9 percent for the oldest workers with 26 years or more experience. Boilermakers, heavy equipment operators, and conveyor car operators as classes had high incidences of hearing impairment."

Evidence of Exposure

Measurement Methods

The current basis for evaluating continuous industrial noise exposures is the A-weighted sound level measurement. The A-weighted network is one of the several standardized frequency

weighting networks on most sound measuring equipment. The A-scale is thought to rate noise in a similar manner as the human ear. Measurements are A-weighted, slow response for the evaluation of continuous noise. If only octave band analyses are available, equivalent A-weighted levels can be calculated for comparison to current standards.

There is a wide variety of instrumentation available for the evaluation of noise from very simple equipment to extremely sophisticated equipment used by acoustical engineers and consultants. The Occupational Safety and Health Administration (OSHA) proposes that noise level measurements for steady-state or continuous noise be made "with a sound level meter confirming as a minimum to the requirements of the ANSI Z1.4-1971, Type 2, and set to an A-weighted slow response or with an audio-dosimeter of equivalent accuracy and precision." Measurements should be taken as close as possible to the hearing zone of the worker whose noise exposure is being evaluated.

For the measurement of impact noise (such as that from a drop hammer), an impact noise meter with peak hold capability should be used. This type meter should conform to the requirements of ANSI Z1.4-1971, Type 1.

Sound level measuring instrumentation should be calibrated with an acoustical calibrator the day of the study, preferably before and after the noise measurements.

Allowable Exposure Limits

The OSHA allowable limits for continuous noise are as follows:

DURATION PER DAY HOURS	SOUND LEVEL dBA SLOW RESPONSE
8	90
6	92
4	95
3	97
2	100
1½	102
1	105
½	110
¼ or less	115

OSHA indicates that "when the daily noise exposure is composed of two or more periods of noise exposure of different levels, their combined effect should be considered, rather than the individual effect on each.

"If the sum of the following fractions $C_1/T_1 + C_2/T_2 + \dots C_n/T_n$ exceeds unity, then the mixed exposure should be considered to exceed the limit value. C_n indicates that the total time of exposure at a specified noise level, and T_n indicates the total time of exposure permitted at that level."

The OSHA allowable limit for impact noise should not exceed 140 dB peak sound pressure level. NOISE ABOVE these limits may cause damage, and the exact level of safety has not yet been determined.

Conclusion

A careful otologic examination and hearing evaluation as outlined above are necessary for an accurate diagnosis. Criteria for diagnosing occupational hearing loss due to exposure to noise include the following:

1. Time and nature of onset of the loss,
2. pattern of hearing loss for different frequencies,
3. confirmed history of occupational exposure of many months or years to noise level in excess of accepted standards, and
4. clinical findings of otologic examination and medical history.

Functional hearing impairment exists when there is no organic cause for the apparent deafness, and the inability to hear results chiefly from psychological or emotional factors.

Acoustic trauma is hearing loss resulting from a loud noise, such as an accidental explosion. If the causative noise occurs on the job, the hearing loss would be occupational.

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 pneumoconioses) 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	jasper
amethyst	muscovite
beach sand	pegmatite
chalcedony	quartz
chert	quartzite
chrysoprase	rock crystal
citrine quartz	rose quartz
cristobalite	sand
diatomaceous earth	sandstone
feldspar	sardonyx
flint	silica flour
free silica	silican hydride
ganister	tridymite
granite	tripoli
gritstone	

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

Trade Names

A 175	Cab-o-Sil H-5
Acticel	Cab-o-Sil L-5
Aerogel 200	Cab-o-Sil MS-7
Aerogel	Cab-o-Sil M-5
Aerosil	Cabosil N 5
Aerosil 175	Cabosil ST-1
Aerosil 200	Carplex
Aerosil 300	Carplex 30
Aerosil 380	Carplex 80
Aerosil A 175	Celite
Aerosil A 300	Celite Superfloss
Aerosil BS-50	Coloidal Silica
Aerosil E 300	Colloidal Silicon Dioxide
Aerosil K 7	Corasil II
Aerosil M-300	Crystallite A 1
Aerosil TT 600	Diatomaceous Silica
Aerosil-Degussa	Dicalite
Amorphous Silica Dust	Dri-die
Aquafil	Extrusil
C.I. 77811	Fossil Flour
C.I. Pigment White 27	Gasil
Cab-o-Sil	HK 125
HK 400	Silica (SiO ₂)
HI-Sil-C	Siliceous Earth
Iatrobeads 6RS8060	Silicic Anhydride
KS 160	Silicon Dioxide
KS 300	Silicon Oxide (SiO ₂)
KS 404	Silikil
Ludox	Silikolloid
Ludox HS 40	Siloxid
Manosil VN 3	Sipur 1500
Milowhite	Snowtex
Min-U-Sil	Snowtex 30
Minusil 5	Snowtex N
Minusil 30	Snowtex O
Nalcast P1W	Snowtex OL
Nalco 1050	Snowtex C
Nalfloc	Super-Cel
Nalfloc N 1050	Superfloss
Neosil	Suprasil
Neosyl	Syton 2X
Nipsil VN 3	Syton WL
OK 412	TK 900

Porasil
Positive Sol 130M
Positive Sol 232
Pregel
Protek-Sorb
Quiso G 30
Quiso 51
RD 8
Santocel CS
Santocel 62
Santocel Z
Si-O-Lite
Siflox
Silanox
Silanox 101

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	cement mixers
abrasives makers	ceramic workers
agriculture	chemical glass makers
auto garage workers	chippers
bisque-kiln workers	coal miners
brick layers	construction workers
brickmakers	cosmetics makers
buffers	cutlery makers
buhrstone workers	diatomaceous earth calciners
carborundum makers	electronic equipment makers
casting cleaners, foundry	enamellers
cement makers	farming
fertilizer makers	quartz workers
fettlers	refractory makers
flint workers	road constructors
foundry workers	rock crushers
furnace liners	rock cutters
fused quartz workers	rock drillers
glass makers	rock grinders
glaze mixers, pottery	rock screeners
granite cutters	rubber compound mixers
granite workers	sand cutters
grinding wheel makers	sand pulverizers
grindstone workers	sandblasters
hard rock miners	sandpaper makers
insecticide makers	sandstone grinders
insulators	sawyers

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

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

(See also Decision-Making Process)

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), and
- pulmonary hemosiderosis (iron deposits in lung tissue).

A respiratory questionnaire, such as that in 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, and
- 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, and
- 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, and
- 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 bacilli. However, silicosis may suppress the usual features of epithelioid cell proliferation, giant cell formation, and lymphocytic reaction.

Laboratory and Clinical Examinations

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

Pulmonary Function

- reduced forced vital capacity (FVC)
- decreased 1 second forced expiratory volume (FEV₁)
- 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 workers' 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 epidemiologic 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.⁹⁴ 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 (FEV₁). 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 FEV₁ and FVC in healthy men 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 FEV₁ and FVC of approximately 80 milliliters per year.⁹⁴

In a study of 727 mine workers from a representative group of metal mines, Dreessen et al.⁹⁵ 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.⁹⁶ reported a study of 2,516 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.⁹⁷

**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 Budlovsky⁹⁸ reported a study of 1,166 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 workplace dust concentrations ranging from 12 to 1,026 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 Potter⁹⁹ reported similar findings and concluded that excessive occupational hazards were not associated with brick making.

Theriault et al.^{100,101,102} 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, FEV₁, 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.¹⁰³ 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 Cralley¹⁰⁴ 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, and
3. electrostatic precipitator

The NIOSH approved methods for samples analysis are:

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

Three methods previously used are:

1. Electron microscopic,
2. exo-electron emission, and
3. differential thermoanalysis.

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

Allowable Exposure 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)

$$\frac{250^*}{\% \text{ SiO}_2 + 5} \quad \text{million particles per cubic foot}$$

- quartz (respirable mass fraction)
(gravimetric analysis)

$$\frac{10^{**}}{\% \text{ SiO}_2 + 2} \quad \text{milligrams per cubic meter}$$

- quartz (total dust)
(gravimetric analysis)

$$\frac{30}{\% \text{ SiO}_2 + 2} \quad \text{milligrams per cubic meter}$$

- 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 Pneumoconioses 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.

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 resolves 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	sulfurous anhydride
sulfur oxide	sulfurous oxide
sulfur oxide	sulphur dioxide
sulfurous acid anhydride	

Occupations with Potential Exposures to Sulfur Dioxide

alkali-salt makers	lead smelters
automotive workers	magnesium foundry workers
beet sugar bleachers	meat preservers
blast furnace workers	mercury smelters
boiler water treaters	metal refiners
bone extractors	oil bleachers
brewery workers	oil processors
brickmakers	ore smelter workers
broommakers	organic sulfonate makers
carbolic acid makers	paper makers
cellulose makers	petroleum refinery workers
coke oven workers	pottery workers
copper smelters	preservative makers
diesel engine operators	protein makers (edible)
diesel engine repairmen	protein makers (industrial)
disinfectant makers	pyrites burners
disinfectors	refrigeration workers

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

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

(See also Decision-Making Process)

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 Appendix C, can be useful in evaluating the extent and importance of respiratory symptoms, such as:
 - breathlessness,
 - sputum production,

- chest pain,
- cough, and
- 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, and
- working as a volunteer fireman.

Signs and Symptoms

Acute Exposures

- irritation of nose and throat,
- burning sensation in the eyes,
- secretion and discharge of tears (lacrimation),
- mucous flows from nose (rhinorrhea),
- cough,
- choking sensation,
- sneezing,
- bronchoconstriction (reflex type),
- increased bronchial secretion,
- increased pulmonary resistance,
- rales, high pitched type,
- prolonged expiratory phase, and
- bronchial asthma.

In severe exposure, the above progresses to:

- Chemical bronchopneumonia (inflammation of the terminal bronchioles and alveoli) and
- 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) and

- 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, and
- expectoration of blood (hemotysis).

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, and
- prolongation of common colds.

Laboratory and Clinical Examinations

Additional data 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 (FEV₁)
- decreased forced vital capacity (FVC)
- decreased specific airway conduction
- increase in respiratory and pulse rates
- 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.¹⁰⁵ This should be taken into consideration when evaluating the following material:

Smith et al.¹⁰⁶ 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 (FEV₁) per year. Workers with FEV₁ 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.¹⁰⁷ 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 ¹⁰⁸ 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 8,047 copper smelter workers exposed to arsenic trioxide and sulfur dioxide, Lee and Fraumeni¹⁰⁹ 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 and
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 Administration (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, and
4. increased urinary sulfate ion concentration is not diagnostic but may indicate degree of exposure.

Toluene Diisocyanate

Introduction

Toluene diisocyanate (TDI) is a liquid used in the manufacture of polyurethane. The liquid, vapor, and aerosol forms are powerful irritants to all tissue.

Skin contact with liquid toluene diisocyanate causes inflammation which may lead to a chemical dermatitis. Liquid in the eyes causes severe irritation with lacrimation (watering of the eyes). A chemical conjunctivitis with swelling of the cornea can result from exposure to the vapor.

The vapor is a potent *respiratory irritant* and *sensitizer*. In some cases where sensitization has occurred, violent respiratory symptoms can develop on exposure to very low concentrations. It is not now known if all or only some people may become sensitized.

The irritating effects of TDI include *rhinitis* (inflammation of the mucous membrane lining the nose), *pharyngitis* (inflammation of the pharynx), *bronchitis*, and in severe exposure, inflammation of the bronchioles. Occasionally the onset is with an attack of *asthma*. Usually the signs and symptoms of chest involvement subside when the exposure ceases. However, there is evidence that lung ventilatory capacity may be impaired in TDI foam workers even though they were symptomless and the maximum permissible concentrations had not been exceeded. Cigarette smokers and those with chronic lung disease show greater impairment.

Medical surveillance with frequent lung function tests, because of respiratory tract involvement, and eosinophil counts because of the allergenic properties of toluene diisocyanate are useful.

Occupations with Potential Exposures to Toluene

Diisocyanate

abrasion resistant rubber
makers

nylon makers
organic chemical synthesizers

adhesive workers	plastic foam makers
aircraft burners	plasticizer workers
foundry workers (core making)	polyurethane foam makers
insulation workers	polyurethane foam users
isocyanate resin workers	polyurethane sprayers
lacquer workers	ship burners
	mine tunnel coaters
	ship welders
	spray painters
	textile processors
	TDI workers
	upholstery makers
	wire coating workers

Medical Evaluation and Differential Diagnosis

(See also Decision-Making Process)

In the Medical History, the following should be considered:

1. Persons with any history of the following are at increased risk from inhalation of toluene diisocyanate:
 - Cigarette smoking
 - respiratory allergy
 - chronic obstructive lung disease
 - chronic bronchitis
 - emphysema
 - cardiopulmonary disease
2. a respiratory questionnaire, such as that in Appendix C, can be useful in evaluating the extent and importance of respiratory symptoms, such as:
 - Breathlessness,
 - sputum production,
 - chest pain,
 - cough, and
 - wheezing.

As part of the Occupational History, the results of any pre-employment and/or periodic lung function tests, as well as blood count and chemistry tests, should be evaluated.

Signs and Symptoms

The reactions encountered with inhalation of TDI vapor are:

1. Primary irritation to which all exposed persons are susceptible to some degree and
2. sensitization reaction, which occurs at much lower exposures in persons who have become sensitized to TDI during earlier exposure.

Primary Irritation

Inhaled toluene diisocyanate vapor causes:

- Burning of eyes, nose, and throat,
- dry, sore throat,
- choking sensation,
- nasal congestion,
- paroxysmal cough (cough which may occur in sudden, periodic attacks), and
- chest pain may occur.

If the TDI vapor concentration is high enough, the effects may progress to a chemical bronchitis with the following:

- Severe bronchospasm,
- feeling of tightness in chest, and
- rales and rhonchi.

This high dose-response may follow a clinical course similar to that of broncho-pneumonia from bacterial infection. In addition, the following may occur:

- Pulmonary edema (excess fluid in the lungs),
- headache,
- insomnia, and
- neurological and psychiatric symptoms.

Sensitization Reaction

- onset (usually without realization) of respiratory problems which become progressively worse with continuous exposure to TDI
- shortness of breath occurring at night (nocturnal dyspnea) and/or nocturnal cough followed by development of asthmatic bronchitis

- exposure of sensitized persons to TDI, even at low levels, can promote a severe asthmatic attack, and may cause death

In some instances, workers with only minimal respiratory symptoms or no apparent effects for several weeks at low level exposure may suddenly develop an acute asthmatic attack.

Acute respiratory effects from TDI exposure are often completely reversible, but continued exposure of affected workers to TDI vapor may result in:

- Asthmatic bronchitis,
- broncho-pneumonia, and
- chronic bronchitis, emphysema, and cor pulmonale (right heart failure).

Laboratory and Clinical Examinations

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

Lung Function Tests

- there is a decrease in the forced expiratory volume at one second (FEV₁)
- forced vital capacity (FVC) is decreased

Chest X-ray—findings are nonspecific. Corresponding changes will be seen if there is a broncho-pneumonia or pulmonary edema (excessive fluids in the lungs):

- absolute eosinophil count often is increased
- white blood count may be slightly increased
- lymphocyte transformation test is positive in sensitized persons

Epidemiology

When considering exposure to TDI, both the primary irritant effects and sensitization must be considered. There is sufficient information to conclude that the primary irritant effects of TDI are dose-related. However, once people are sensitized to TDI, there appears to be little or no dose-response relationship,¹¹⁰ and any further exposure may be extremely dangerous. This should be kept in mind when considering the following data:

There is a report of a study of 12 workers in an automobile plant making polyurethane foam crashpads.¹¹⁰ For the first 3 weeks the workers were exposed to air concentrations of TDI not exceeding 0.01 ppm. The next week, air concentrations of TDI rose to 0.03-0.07 ppm. At the latter exposure, all workers complained of respiratory symptoms including coryzal symptoms, continuous coughing, sore throat, dyspnea, fatigue, and night sweats. Subsequently, air concentration of TDI were reduced to 0.01-0.03 ppm. For the next 3½ months there were no further respiratory symptoms or complaints, and none of the workers appeared to have any permanent effects or became sensitized from the exposure.

Walworth and Virchow¹¹¹ report a study of workers' health for 2½ years in a polyurethane foam plant producing slabs. The average values of air concentrations of TDI were given as a range of 0.00-2.6 ppm with a time-weighted average level estimated in the range of 0.00-0.15 ppm (monthly). 83 workers developed illnesses attributed to TDI. 54 showed upper respiratory infection, 11 had tracheitis, 9 had bronchitis, and 9 had bronchial asthma. Most illnesses, it was reported, started between the third and fourth week of exposure. The report indicates evidence of sensitization.

Elkins¹¹² published a report on a 5-year study of TDI exposure in 14 plants. The author concluded that 0.01 ppm for TDI was "a not unreasonable limit." Elkin's data is summarized in the table found on page 183.

Glass and Thom¹¹³ report a study in 3 plants in New Zealand. In one plant where polyurethane foam was produced in a batch molding process, atmospheric TDI concentrations ranged from 0.003-0.0123 ppm and 3 cases of respiratory sensitization were reported in one year. In the second plant (similar to the first), TDI concentrations in air ranged from 0.005-0.100 ppm and two mild cases of coryzal symptoms, one case of possible sensitization, and one case of acute asthma attack on heavy exposure (with no evidence of sensitization) were reported. In the third plant, polyurethane foam was produced in the continuous slab process. Air concentrations of TDI ranged from 0.000-0.018 ppm in the third plant. Two cases of mild coryzal symptoms with no evidence of sensitization were reported (the men experiencing these symptoms wore canister-type respiratory protection).

Williamson¹¹⁴ reported a study of 18 workers exposed to air concentrations of TDI generally below 0.02 ppm except for a brief exposure (not more than 10 minutes) to at least 0.2 ppm after a spill. Over a 14-month period, no differences in ventilatory measurements were detected within a work-shift from Monday to Friday. It was reported that none of the men suffered illness attributed to TDI or developed TDI sensitization during this study.

Maxon¹¹⁵ reported a study of 7 workers exposed to TDI in a plastic varnish plant. Environmental data was minimal because only 3 measurements of TDI in air were made (0.08 ppm, 0.10 ppm, and 0.12 ppm). Symptoms developed within ½ hour to 3 weeks following initial exposure. All workers had cough and dyspnea and 4 had hemotysis. There was evidence that 4 workers had become sensitized to TDI.

Bruchner et al.¹¹⁶ reported a study of 26 workers exposed to a range of 0.0-2.4 ppm isocyanates and a range of median values of 0.0-0.033 ppm over an 11-year period. The workers were engaged in research and development and production of isocyanates, presumably including TDI. 5 workers showed minimal symptoms of mucous membrane irritation, 16 showed marked irritation of the respiratory tract, and 5 were sensitized. 4 of the 5 sensitized workers showed a positive lymphocyte transformation test (an indication of an immunologic allergic sensitization) using TDI-human serum albumin conjugate as the antigen.

Peters¹¹⁷ reported a long-term study of ventilatory measurements on workers repeatedly exposed to TDI. Initial atmospheric concentrations of TDI ranged from 0.0001-0.0030 ppm and later concentrations ranged from 0.000-0.0120 ppm. After exposure to TDI on the first day of this study, decreases were reported in the forced vital capacity (FVC), FEV 1.0, peak flow rate (PFR), and flow rate at 50% and 25% of vital capacity of all 38 workers studied. At the end of the first week, FVC had returned to baseline but mean FEV 1.0 was still depressed and mean flow rates were even more depressed. A follow-up was made six months later on 28 of the workers still available. As a group, the 28 showed decrease in mean FEV 1.0, FEV 1.0/FVC, and in flow rates. 8 workers had cough and phlegm. Continued decline in FEV 1.0 was reported in the workers studies at six month intervals for a total of two years.

**SUMMARY OF TDI CONCENTRATIONS IN AIR AND
CASES OF TDI INTOXICATION AT 14 PLANTS**

PLANT	YEAR	AIR ANALYSIS			NUMBER OF CASES		QUESTION- ABLE OR DISPUTED
		NUMBER OF TESTS	AVERAGE TDI CONCENTRA- TION (ppm)	WORKERS EXPOSED	ACCEPTED ESTABLISHED		
1	1957	-	-	2	1	1	
2	1957-8	14	0.005	50	3	28	
2	1960	33	0.028	100	14	25	
2	1961-2	55	0.015	50	3	2	
3	1958-60	12	0.009	25	-	-	
4	1958-62	21	0.004	40	5	15	
5	1958-61	11	0.008	6	1	?	
6	1958-61	28	0.015	40	8	-	
7	1961	4	0.001 (Less than)	4	-	-	
8	1961	5	0.001*	5	1	-	
9	1961	3	0.006	4	-	-	
10	1961	14	0.002**	3	2	-	
11	1961	14	0.54 **	4	4	-	
12	1962	6	0.009	6	-	1	
13	1962	4	Nil	20	-	1	
14	1962	6	0.000	20	-	-	
TOTAL		230		379	42	73	

* Probably not representative of exposure.

** Not representative of exposure.

Elkins, H.B.; et al. 1962

Evidence of Exposure

Sampling and Analysis

The two most commonly used methods for the collection of air samples for toluene diisocyanate are:

1. The Ranta method and
2. the Marcali method.

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

There are also available a number of field instruments for the determination of TDI concentrations in air. Many of them are based on modifications of the Marcali sampling method.

Allowable Exposure Limits

The Occupational Safety and Health Administration (OSHA) limits exposure to toluene diisocyanate to 0.02 parts per million parts of air by volume. This is a Ceiling Limit which should never be exceeded. These allowable levels may not be safe for all persons.

Conclusion

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

1. Confirmed history of occupational exposure to TDI vapor,
2. clinical findings compatible with the respiratory syndrome as outlined above,
3. progressive decrease in lung capacity, and
4. progressive increase in eosinophil count.

APPENDIX A-1

EPIDEMIOLOGIC REFERENCES

Antimony

1. Brieger, H.; et al. 1954. *Ind. Med. Surg.* 23:521.
2. Cooper, D. A.; et al. 1968. *Am. J. Roentgenol. Radium Ther. Nucl. Med.* 103:495.
3. Renes, L. E. 1953. *Arch. Ind. Hyg.* 7:99.
4. McCallum, R. I. 1963. *Ann. Occupat. Hyg.* 6:60.
5. McCallum, R. I. 1965. *Lancet.* 2:882.
6. McCallum, R. I. 1967. *Trans. Soc. Occup. Med.* 17:134.
7. Karajovic, D. 1957. Proc. 12th Internat. Congress Occupational Health, Helsinki. 370.
8. Taylor, P. J. 1966. *Brit. J. Industr. Med.* 23:318.
9. Rodier, J., and Souchere, G. 1955. *Bull. Inst. Hyg. Maroc* 15:93.
10. Gallina, R., and Luvoni, R. 1958. *Rass. Med. Ind.* 27:28.

Inorganic Arsenic

11. Pinto, S. S.; et al. 1976. *J. Occup. Med.* 18:677.
12. NIOSH. 1975. *Criteria for a Recommended Standard - Occupational Exposure to Inorganic Arsenic* (HEW Publication No. 75-149). Cincinnati: NIOSH.
13. Butzengeiger, K. H. 1940. *Klin. Wochenscher.* 19:523. German.
14. Butzengeiger, K. H. 1949. *Dtsch. Arch. Klin. Med.* 194:1. German.
15. Lee, A. M.; and Fraumeni, J. R. 1969. *J. Natl. Cancer Inst.* 42:1045.
16. Perry, K.; et al. 1948. *Brit. J. Industr. Med.* 5:6.
17. Birmingham, D. J.; et al. 1965. *Arch. Derm.* 91:457.
18. Milham, S., Jr.; and Strong, T. 1974. *Environ. Res.* 7:176.

Asbestos

19. Enterline, P.; et al. *A Study of the Dose-Response Relationship Asbestos Dust and Lung Cancer.* Unpublished manuscript.
20. NIOSH. 1972. *Criteria for a Recommended Standard - Occupational Exposure to Asbestos.* Cincinnati: NIOSH.

21. Murphy, R. L. H.; et al. 1971. *N. Eng. J. Med.* 285:1271.
22. Pennsylvania Dept. of Public Health. Unpublished Communication.
23. Selikoff, I. J.; et al. 1968. *JAMA.* 204:106.
24. Selikoff, I. J.; et al. 1964. *JAMA.* 188:22.

Benzene

25. NIOSH. 1974. *Criteria for a Recommended Exposure to Benzene* (HEW Publication No. 74-137). Cincinnati: NIOSH.
26. Aksoy, M.; et al. 1971. *Brit. J. Industr. Med.* 28:296.
27. Aksoy, M.; et al. 1976. *Acta haemat.* 55:65.
28. Forni, A. M.; et al. 1971. *Arch. Environ. Health.* 23:385.
29. Hartwick, G.; and Schwanitz, G. 1972. *Dtsch. Med. Wochenechr.* 97:45. German.
30. Greenberg, L. 1926. *Public Health Reports.* 41:1526.
31. Hardy, H. L.; and Elkins, H. B. 1948. *J. Ind. Hyg. Toxicol.* 30:196.
32. Juzwiak, I. 1969. *Med. Przemysłowa.* 20:67. Polish.
33. Pagnotto, L. D.; et al. 1961. *AIHA J.* 22:417.
34. Sherwood, R. J. 1972. *Am. Occup. Hyg.* 15:409.

Carbon Monoxide

35. NIOSH. 1972. *Criteria for a Recommended Standard-Occupational Exposure to Carbon Monoxide.* Cincinnati: NIOSH.
36. Sayers, R. R.; et al. 1929. *USPHS Bull.* 186. Washington: GPO.
37. McFarland, R. A. 1944. *J. Aviation Med.* 15:381.
38. Halperin, M. H.; et al. 1959. *J. Physiol.* 146:583.
39. Horvath, S. M. 1972. *Arch. Env. Health.* 23:343.
40. Schulte, J. H. 1963. *Arch. Env. Health.* 7:524.
41. Beard, R. R.; and Wertheim, G. 1967. *Am. J. Pub. Health.* 57:2012.
42. Beard, R. R.; and Grandstaff, N. W. 1970. *Proc. Ann. Conf. Env. Toxic.* 1:93.
43. Trouton, D.; and Eysewck, H. J. 1961. *Handbook of Abnormal Psychology.* New York: Basic Books.
44. NIOSH. 1972. *Criteria for a Recommended Standard-Occupational Exposure to Carbon Monoxide.* Cincinnati: NIOSH.

Coke Oven Emissions

45. NIOSH. 1973. *Criteria for a Recommended Standard - Occupational Exposure to Coke Oven Emissions*. (Publication No. HSM-11016). NIOSH: Cincinnati.
46. Doll, R. 1952. *Brit. J. Industr. Med.* 9:180.
47. Lloyd, J. W. 1971. *J. Occup. Med.* 13:53.
48. Redmond, C. K.; et al. 1972. *J. Occup. Med.* 14:621.
49. Henry, S. A.; et al. 1931. *J. Hyg.* 31:126.
50. Final Occupational Safety and Health Standard for Exposure to Coke Oven Emissions, Part III. October 22, 1976. *Federal Register*.

Cotton Dust

51. Martin, C. F.; and Higgins, J. E. 1976. *J. Occup. Med.* 18:455.
52. Shilling, R. S. F.; et al. 1955. *Brit. J. Industr. Med.* 12:217.
53. Shilling, R. S. F. 1956. *Lancet.* 2:261.
54. Zuskin, E.; et al. 1969. *Arch. Environ. Health.* 19:666.
55. Schrag, P. E.; and Gullett, A. D. 1970. *Am. Rev. Resp. Disease.* 101:497.
56. Molyneux, M. K. B.; and Tombleson, J. B. L. 1970. *Brit. J. Industr. Med.* 27:225.
57. Imbus, H. R.; and Suh, M. W. 1973. *Arch. Environ. Health.* 26:183.
58. Merchant, J. A.; et al. 1972. *Ann. Int. Med.* 76:423.

Inorganic Lead

59. Feldman, R. G.; et al. 1977. *Lancet.* 1:89. Published Communication.
60. NIOSH. 1972. *Criteria for a Recommended Standard - Occupational Exposure to Inorganic Lead*. Cincinnati: NIOSH.
61. Elkins, H. D. 1959. *The Chemistry of Industrial Toxicology*. 2nd ed. New York: John Wiley.
62. Dreeson, W. C.; et al. 1941. Public Health Bulletin 262. Washington: GPO.
63. National Academy of Sciences, Division of Medical Sciences, Committee on Biological Effects of Atmospheric Pollutants. 1971. *Airborne Lead in Perspective*.
64. Tola, S.; and Nordman, C. H. 1977. *Environ. Res.* 13:250.

65. Sakurai, H.; et al. 1974. *Arch. Environ. Health*. 29:157.
66. Lancranjan, I.; et al. 1975. *Arch. Environ. Health*. 30:396.
67. Cooper, W. C.; and Gaffey, W. R. 1975. *J. Occup. Med.* 17:100.

Inorganic Mercury

68. Miller, J. M.; et al. 1975. *AIHA J.* 36:725.
69. Shandar, A.; and Simson, R. E. 1971. *Med. J. Aust.* 2:1005.
70. Rentos, P. G.; and Seligman, E. J. 1968. *Arch. Environ. Health*. 16:794.
71. Turrian, H.; et al. 1956. *Schweiz. Med. Wchenschr.* 86:1091. German.
72. NIOSH. 1973. *Criteria for a Recommended Standard - Occupational Exposure to Inorganic Mercury* (Publication No. HSM 73-11024). Cincinnati:NIOSH.
73. McGill, C. M.; et al. 1964. *J. Occup. Med.* 6:335.
74. Smith, R. G.; et al. 1970. *AIHA J.* 31:687.
75. Baldi, G.; et al. 1953. *Med. Lav.* 44:161. Italian.

Nitrogen Dioxide

76. NIOSH. 1976. *Criteria for a Recommended Standard - Occupational Exposure to the Oxides of Nitrogen (Nitrogen Dioxide and Nitric Oxide)*. Cincinnati: NIOSH.
77. Muller, B. 1969. *Respiration*. 26:249.
78. Kosmider, S.; et al. 1972. *Zentralbl Arbeitsmed.* 22:362. German.
79. Tse, R. L.; and Bockman, A. A. 1970. *JAMA*. 212:1344.
80. Emergency Exposure Limits. 1964. *AIHA J.* 25:580.
81. Lowry, T.; and Schuman, L. M. 1956. *JAMA*. 162:153.

Noise

82. NIOSH. 1972. *Criteria for a Recommended Standard - Occupational Exposure to Noise*. Cincinnati: NIOSH.
83. Coles and Knight. 1960. *Ann. Occ. Hyg.* 2:267.
84. Yaffe and Jones. 1961. U. S. Public Health Service Publication 850. Washington: GPO.
85. Schneider et al. 1961. *AIHA J.* 22:245.
86. Brohm and Zlamal. 1962. *Cas. Lek. Ces.* 101:300. Czech.
87. Mancini and Stancari. 1962. *Rass. Med. Ind.* 31:239. Italian.

88. Chadwick. 1963. *J. Laryngol.* 77:467.
89. Filin. 1963. *Gog. Tr. Prof. Zabol.* 7:3. Russian.
90. Weston. 1963. *J. Aus. Inst. Agr. Sci.* 29:15.
91. Cohen et al. 1970. *Arch. Env. Health.* 20:614.
92. Burns and Robinson. 1970. *Hearing and Noise in Industry.* London: Her Majesty's Stationery Office.
93. Stone et al. 1971. *AIHA J.* 32:123.

Crystalline Silica

94. Musk, A. W.; et al. 1977. *Am. Rev. Respir. Dis.* 115:769.
95. Dreessen, W. C.; et al. 1942. Public Health Bulletin 277. Washington: GPO.
96. Flinn, R. H.; et al. 1939. Public Health Bulletin 244. Washington: GPO.
97. NIOSH. 1974. *Criteria for a Recommended Standard - Occupational Exposure to Crystalline Silica* (HEW Publication No. 75-120). Cincinnati: NIOSH.
98. Rajhans, G. S.; and Budlovsky, J. 1972. *AIHA J.* 33:258.
99. Keatinge, G. F.; and Potter, N. M. 1949. *Brit. J. Industr. Med.* 1:31.
100. Theriault, G. P.; et al. 1974. *Arch. Environ. Health.* 28:12.
101. Theriault, G. P.; et al. 1974. *Arch. Environ. Health.* 28:18.
102. Theriault, G. P.; et al. 1974. *Arch. Environ. Health.* 28:23.
103. UICC/Cincinnati classification of the radiographic appearances of pneumoconioses. 1970. *Chest.* 58:57.
104. Cooper, W. C.; and Cralley, L. J. 1958. Public Health Bulletin 601. Washington: GPO.

Sulfur Dioxide

105. NIOSH. 1974. *Criteria for a Recommended Standard - Occupational Exposure to Sulfur Dioxide* (HEW Publication No. 74-11). Cincinnati: NIOSH.
106. Smith, T. J.; et al. 1977. *Am. Rev. Respir. Dis.* 116:31.
107. Kehoe, R. A.; et al. 1932. *J. Industr. Hyg.* 14:159.
108. Skalpe, I. O. 1964. *Brit. J. Industr. Med.* 21:69.
109. Lee, A. M.; and Fraumeni, J. F. 1969. *J. Nat. Cancer Inst.* 42:1045.

Toluene Diisocyanate

110. NIOSH. 1973. *Criteria for a Recommended Standard - Occupational Exposure to Toluene Diisocyanate*. Cincinnati: NIOSH.
111. Walworth, H. T.; and Virchow, W. E. 1959. *AIHA J.* 20:205.
112. Elkins, H. B.; et al. 1962. *AIHA J.* 23:265.
113. Glass, W. I.; and Thom, N. G. 1964. *M. Z. Med. J.* 63:642.
114. Williamson, K. S. 1964. *Trans. Assoc. Ind. Med. Off.* 14:81.
115. Maxon, F. C. 1964. *Arch. Env. Health.* 8:755.
116. Bruckner, H. C.; et al. 1968. *Arch. Env. Health.* 16:619.
117. Peters, J. M.; et al. 1968. *Arch. Env. Health.* 16:642.

APPENDIX A-2

BIBLIOGRAPHY

Antimony

- Allison, A. C.; and Patton, G. R. 1972. *Mutation Res.* 16:332.
- Bromberger-Barnea, B.; and Stephens, N. L. 1965. *AIHA J.* 26:404.
- Browning, E. 1969. *Toxicology of Industrial Metals.* 2nd ed. London: Butterworth.
- Daum, S. M.; and Stellman, J. M. 1963. *Work is Dangerous to Your Health.* New York: Random House.
- Elkins, H. B. 1959. *The Chemistry of Industrial Toxicology.* 2nd ed. New York: John Wiley & Sons.
- Hunter, D. 1975. *The Diseases of Occupations.* 5th ed. Boston: Little, Brown.
- Hygiene Guide Series.* Akron: American Industrial Hygiene Association.
- Key, M. M.; et al. eds. 1977. *Occupational Diseases—A Guide to Their Recognition.* Washington: U.S. Government Printing Office.
- NIOSH. 1977. *Manual of Sampling Data Sheets, S2* (DHEW Publication No. 77-159). Cincinnati: NIOSH.
- OS&HR.* June 16, 1977. Washington: Bureau of National Affairs, Inc.
- Patty, F. A. ed. 1963. *Industrial Hygiene and Toxicology.* 2nd ed. New York: John Wiley & Sons.
- Plunkett, E. R. 1976. *Handbook of Industrial Toxicology.* New York: Chemical Publishing Co., Inc.
- Sax, N. I. 1975. *Dangerous Properties of Industrial Materials.* 4th ed. New York: Reinhold Publishing Corporation.
- Schroeder, H. A. 1974. *The Poisons Around Us.* Bloomington: Indiana University Press.

Inorganic Arsenic

Daum, S. M.; and Stellman, J. M. 1973. *Work is Dangerous to Your Health*. New York: Random House.

Dinman, B. D. 1960. *J. Occup. Med.* 2:137.

Foreman, H. 1962. *Ann. Rev. Pharm.* 2:341.

Hunter, D. 1975. *The Diseases of Occupations*. 5th ed. Boston: Little, Brown.

Hygiene Guide Series. Akron: American Industrial Hygiene Association.

Jackson, R. 1975. *CMAJ*. 113:396.

Key, M. M.; et al. eds. 1977. *Occupational Diseases— A Guide to Their Recognition*. Washington: U.S. Government Printing Office.

NIOSH. 1977. *Manual of Sampling Data Sheets*, S309 (DHEW Publication No. 77-159). Cincinnati: NIOSH.

OS&HR. June 16, 1977. Washington: Bureau of National Affairs, Inc.

Patty, F. A. ed. 1963. *Industrial Hygiene and Toxicology*. 2nd ed. New York: John Wiley & Sons.

Pinto, S. 1976. *J. Occup. Med.* 18:677.

Plunkett, E. R. 1976. *Handbook of Industrial Toxicology*. New York: Chemical Publishing Co., Inc.

Sax, N. I. 1975. *Dangerous Properties of Industrial Materials*. 4th ed. New York: Reinhold Publishing Corporation.

Schroeder, H. A. 1974. *The Poisons Around Us*. Bloomington: Indiana University Press.

Asbestos

Arena, J. M. 1970. *Poisoning, Toxicology, Symptoms, Treatments*. Springfield: Charles C. Thomas.

Council on Occupational Health. 1963. *Arch. Env. Health*. 7:130.

Hamilton, A.; and Hardy, H. L. 1974. *Industrial Toxicology*. 3rd ed. Acton: Publishing Sciences Group.

Morgan, W. K. C.; and Seaton, A. 1975. *Occupational Lung Diseases*. Philadelphia: W. B. Saunders.

Advisory Committee on Asbestos Cancers. 1972. *The Biological Effects of Asbestos*. Delivered to World Health Organization, Lyon, Oct. 5-6.

NIOSH. 1972. *Criteria for a Recommended Standard—Occupational Exposure to Asbestos*. Cincinnati: NIOSH.

Yater, W. M.; and Oliver, W. F. 1961. *Symptom Diagnosis*. 5th ed. New York: Appleton-Century-Croft.

Benzene

Aksoy, M. 1975. *Blut*. 30:255.

Aksoy, M.; et al. 1974. *Blut*. 28:293.

Daum, S. M.; and Stellman, J. M. 1973. *Work is Dangerous to Your Health*. New York: Random House.

Deichmann, W. 1963. *Tox. App. Pharm.* 5:201.

Elkins, H. B. 1959. *The Chemistry of Industrial Toxicology*. 2nd ed. New York: John Wiley & Sons.

Key, M. M.; et al. eds. 1977. *Occupational Diseases— A Guide to Their Recognition*. Washington: U.S. Government Printing Office.

Gerardi, H. W. 1960. *Toxicology and Biochemistry of Aromatic Hydrocarbons*. New York: Elsevier Publishing Co.

Hunter, D. 1975. *The Diseases of Occupations*. 5th ed. Boston: Little, Brown.

Hygiene Guide Series. Akron: American Industrial Hygiene Association.

NIOSH. 1977. *Manual of Sampling Data Sheets, S311* (DHEW Publication No. 77-159). Cincinnati: NIOSH.

OS&HR. June 6, 1977. Washington: Bureau of National Affairs, Inc.

Patty, F. A., ed. 1963. *Industrial Hygiene and Toxicology*. 2nd ed. New York: John Wiley & Sons.

Plunkett, E. R. 1976. *Handbook of Industrial Toxicology*. New York: Chemical Publishing Co., Inc.

Sax, N. I. 1975. *Dangerous Properties of Industrial Materials*. 4th ed. New York: Reinhold Publishing Corporation.

Thorpe, J. J. 1974. *J. Occup. Med.* 16:375.

Vigliana, E. C. 1976. *Environ. Res.* 11:122.

Carbon Monoxide

NIOSH. 1973. *Criteria for a Recommended Standard—Occupational Exposure to Carbon Monoxide*. Cincinnati: NIOSH.

Gafer, W. M. 1966. *Occupational Diseases—A Guide to Their Recognition*. Washington: U.S. Government Printing Office.

Breaker, W.; and Mossman, A. L. 1970. *Toxic Gases: First Aid and Medical Treatment*. Rutherford: Matheson Gas Products.

Hunter, D. 1969. *The Diseases of Occupations*. 4th ed. Boston: Little, Brown.

Arena, J. M. 1970. *Poisoning, Toxicology, Symptoms, Treatment*. Springfield: Charles C. Thomas.

Plunkett, E. R. 1966. *Handbook of Industrial Toxicology*. New York: Chemical Publishing.

Coke Oven Emissions

Daum, S. M.; and Stellman, J. M. 1973. *Work is Dangerous to Your Health*. New York: Random House.

English, J. D. 1975. *Position of United Steelworkers of America on Proposed Coke Oven Regulations*.

Hygiene Guide Series. Akron: American Industrial Hygiene Association.

Jackson, J. O.; et al. 1974. *AIHA J.* 35:276.

Key, M. M.; et al. eds. 1977. *Occupational Diseases—A Guide to Their Recognition*. Washington: U.S. Government Printing Office.

Lloyd, J. W.; and Ciocco, A. 1969. *J. Occup. Med.* 11:299.

Mittman, C.; et al. 1974. *Am. J. Med.* 57:92.

NIOSH. 1977. *Manual of Sampling Data Sheets*, 8 (DHEW Publication No. 77-159). Cincinnati: NIOSH.

OS&HR. August 7, 1975. Washington: Bureau of National Affairs, Inc.

OS&HR. June 16, 1977. Washington: Bureau of National Affairs, Inc.

Patty, F. A. ed. 1963. *Industrial Hygiene and Toxicology*. 2nd ed. New York: John Wiley & Sons.

Plunkett, E. R. 1976. *Handbook of Industrial Toxicology*. New York: Chemical Publishing Co., Inc.

Radford, E. P. 1976. *Ann. N.Y. Acad. Sci.* 271:228.

Sax, N. I. 1975. *Dangerous Properties of Industrial Materials*. 4th ed. New York: Reinhold Publishing Corporation.

Second Coke Worker Conference. 1975. *Studies of Coke Plant Workers*.

Cotton Dust

Barnes, L; and Simpson, G. R. 1976. *J. Occup. Med.* 18:551.

Bouhuys, A.; et al. 1970. *Arch. Environ. health.* 21:475.

Bouhuys, A. 1971. *Arch. Environ. Health.* 23:405.

Cavagna, G.; et al. 1969. *Brit. J. Industr. Med.* 26:314.

Daum, S. M.; and Stellman, J. M. 1973. *Work is Dangerous to Your Health*. New York: Random House.

Hunter, D. 1975. *The Diseases of Occupations*. 5th ed. Boston: Little, Brown.

Hygiene Guide Series. Akron: American Industrial Hygiene Association.

Key, M. M; et al. eds. 1977. *Occupational Diseases—A Guide to Their Recognition*. Washington: U.S. Government Printing Office.

Martin, C. F.; and Higgins, J. E. 1976. *J. Occup. Med.* 18:455.
Merchant, J. A.; et al. 1972. *Ann. Int. Med.* 76:423.

Morgan, W. K. C.; and Seaton, A. 1975. *Occupational Lung Diseases*. Philadelphia: W. B. Sounder's Company.

Musk, A. W.; et al. 1977. *Am. Rev. Respir. Dis.* 115:769.

NIOSH. 1977. *Manual of Sampling Data Sheets*, 50 (DHEW Publication No. 77-159). Cincinnati: NIOSH.

OS&HR. June 16, 1977. Washington: Bureau of National Affairs, Inc.

Plunkett, E. R. 1976. *handbook of Industrial Toxicology*. New York: Chemical Publishing Co., Inc.

Sax, N. I. 1975. *Dangerous Properties of Industrial Materials*. 4th ed. New York: Reinhold Publishing Corporation.

Taylor, G.; et al. 1971. *Brit. J. Industr. Med.* 28:143.

Tuma, J.; et al. 1973. *J. Occup. Med.* 15:409.

Inorganic Lead

Benson, G. I.; et al. 1976. *Brit. J. Industr. Med.* 33:29.

Browning, E. 1969. *Toxicity of Industrial Metals*. 2nd ed. New York: Appleton-Century-Crofts.

Casarett, L. J.; and Doull, J. eds. 1975. *The Basic Science of Poisons*. New York: MacMillan Publishing Co., Inc.

Goldwater, L. J.; and Hoover, A. W. 1967. *Arch. Environ. Health.* 15:60.

Hammond, P. B. 1969. *Essays Toxicol.* 1:116.

Hunter, D. 1975. *The Diseases of Occupations*. 5th ed. Boston: Little, Brown.

Hygiene Guide Series. Akron: American Industrial Hygiene Association.

Key, M. M.; et al. eds. 1977. *Occupational Diseases—A Guide to Their Recognition*. Washington: U.S. Government Printing Office.

NIOSH. 1972. *Criteria for a Recommended Standard—Occupational Exposure to Inorganic Lead*. Cincinnati: NIOSH.

NIOSH. 1977. *Manual of Sampling Data Sheets, S341* (DHEW Publication No. 77-159). Cincinnati: NIOSH.

OS&HR. June 16, 1977. Washington: Bureau of National Affairs, Inc.

Plunkett, E. R. 1976. *Handbook of Industrial Toxicology*. New York: Chemical Publishing Co., Inc.

Sax, N. I. 1975. *Dangerous Properties of Industrial Materials*. 4th ed. New York: Reinhold Publishing Corporation.

Schroeder, H. A. 1974. *The Poisons Around Us*. Bloomington: Indiana University Press.

Inorganic Mercury

Clarkson, T. W. 1972. *Crit. Rev. Toxicol.* 1:203.

Daum, S. M.; and Stellman, J. M. 1973. *Work is Dangerous to Your Health*. New York: Random House.

Elkins, H. B. 1959. *The Chemistry of Industrial Toxicology*. 2nd ed. New York: John Wiley & Sons.

Hunter, D. 1975. *The Diseases of Occupations*. 5th ed. Boston: Little, Brown.

Hygiene Guide Series. Akron: American Industrial Hygiene Association.

Key, M. M.; et al. eds. 1977. *Occupational Diseases—A Guide to Their Recognition*. Washington: U.S. Government Printing Office.

Lee, S. D. ed. 1977. *Biochemical Effects of Environmental Pollutants*. Ann Harbor: Ann Harbor Science Publishers, Inc.

Magos, L. 1975. *Brit. Med. Bull.* 31:241.

Nadi, N. A.; and Yassin, A. K. 1974. *J. Top. Med. Hyg.* 77:128.

NIOSH. 1977. *Manual of Sampling Data Sheets*, 20 (DHEW Publication No. 77-159). Cincinnati: NIOSH.

OS&HR. June 16, 1977. Washington: Bureau of National Affairs, Inc.

Patty, F. A. ed. 1963. *Industrial Hygiene and Toxicology*. 2nd ed. New York: John Wiley & Sons.

Plunkett, E. R. 1976. *Handbook of Industrial Toxicology*. New York: Chemical Publishing Co., Inc.

Sax, N. I. 1975. *Dangerous Properties of Industrial Materials*. 4th ed. New York: Reinhold Publishing Corporation.

Schroeder, H. A. 1974. *The Poisons Around Us*. Bloomington: Indiana University Press.

Selikoff, I. J. ed. 1971. *Environ. Res.* 4:1.

Nitrogen Dioxide

Daum, S. M.; and Stellman, J. M. 1973. *Work Is Dangerous to Your Health*. New York: Random House.

Dowell, A. R.; et al. 1971. *Arch. Intern. Med.* 128:74.

Ehrlich, R.; and Henry, M. C. 1968. *Arch. Environ. Health.* 17:860.

Freeman, G.; et al. 1974. *Arch. Environ. Health.* 29:203.

Key, M. M.; et al. eds. 1977. *Occupational Diseases—A Guide to Their Recognition*. Washington: U.S. Government Printing Office.

Hunter, D. 1975. *The Diseases of Occupations*. 5th ed. Boston: Little, Brown.

Hygiene Guide Series. Akron: American Industrial Hygiene Association.

Lee, S. D. ed. 1977. *Biochemical Effects of Environmental Pollutants*. Ann Harbor: Ann Harbor Science Publishers, Inc.

NIOSH. 1977. *Manual of Sampling Data Sheets*, 32 (DHEW Publication No. 77-159). Cincinnati: NIOSH.

Oreshek, J.; et al. 1976. *J. Clin. Investig.* 57:301.

OS&HR. June 16, 1977. Washington: Bureau of National Affairs, Inc.

Plunkett, E. R. 1976. *Handbook of Industrial Toxicology*. New York: Chemical Publishing Co., Inc.

Ramirez-R. J.; and Dowell, A. R. 1971. *Ann. Inter. Med.* 74:569.

Sax, N. I. 1975. *Dangerous Properties of Industrial Materials*. 4th ed. New York: Reinhold Publishing Corporation.

Scott, E. G.; and Hunt, Jr., W. B. 1973. *Chest*. 63:701.

Noise

American Medical Association. 1955. *J.A.M.A.* 157:1408.

Sataloff, J.; and Michael, P. 1973. *Hearing Conservation*. Springfield: Charles C. Thomas.

Robinson, D. W.; and Burns, W. 1970. *Hearing and Noise In Industry*. London: Her Majesty's Stationery Office.

Kryter, K. D. 1970. *The Effects of Noise on Man*. New York: Academic Press.

American Academy of Ophthalmology and Otolaryngology. 1961. *Guide for the Evaluation of Hearing Impairment*.

Gafafer, W. M. 1966. *Occupational Diseases—A Guide to their Recognition*. Washington: U.S. Government Printing Office.

Crystalline Silica

Daum, S. M.; and Stellman, J. M. 1973. *Work Is Dangerous to Your Health*. New York: Random House.

- Heppleston, A. G. 1968. *Brit. Med. Bull.* 25:282.
- Hunter, D. 1975. *The Diseases of Occupations*. 5th ed. Boston: Little, Brown.
- Hygiene Guide Series*. Akron: American Industrial Hygiene Association.
- Jacobson, G.; and Lainhart, W. S., eds. 1972. *Med. Radiogr. Photogr.* 48:65.
- Key, M. M.; et al. eds. 1977. *Occupational Diseases—A Guide to Their Recognition*. Washington: U.S. Government Printing Office.
- Montgomery, C. H.; and Synder, H. B. 1975. *J. Occup. Med.* 17:523.
- Morgan, W. K. C.; and Seaton, A. 1975. *Occupational Lung Diseases*. Philadelphia: W. A. Saunders Company.
- NIOSH. 1977. *Manual of Sampling Data Sheets, S315* (DHEW Publication No. 77-159). Cincinnati: NIOSH.
- OS&HR. June 16, 1977. Washington: Bureau of National Affairs, Inc.
- Patty, F. A. ed. 1963. *Industrial Hygiene and Toxicology*. 2nd ed. New York: John Wiley & Sons.
- Pintar, K.; et al. 1976. *Arch. Pathol. Lab. Med.* 100:535.
- Plunkett, E. R. 1976. *Handbook of Industrial Toxicology*. New York: Chemical Publishing Co., Inc.
- Sax, N. I. 1975. *Dangerous Properties of Industrial Materials*. 4th ed. New York: Reinhold Publishing Corporation.
- Suratt, P. M.; et al. 1977. *Am. Rev. Respir. Dis.* 115:521.
- Vitums, V. D.; et al. 1977. *Arch. Environ. Health.* 34:62.

Sulfur Dioxide

- Amdur, M. O.; et al. 1953. *Lancet.* 265:758.
- Andersen, I.; et al. 1974. *Arch. Environ. Health.* 28:31.
- Anderson, A. 1950. *Brit. J. Industr. Med.* 7:82.
- Daum, S. M.; and Stellman, J. M. 1973. *Work Is Dangerous to Your Health*. New York: Random House.
- Hunter, D. 1975. *The Diseases of Occupations*. 5th ed. Boston: Little, Brown.

Hygiene Guide Series. Akron: American Industrial Hygiene Association.

Key, M. M.; et al. eds. 1977. *Occupational Diseases—A Guide to Their Recognition*. Washington: U.S. Government Printing Office.

Lee, S. D. ed. 1977. *Biochemical Effects of Environmental Pollutants*. Ann Harbor: Ann Harbor Science Publishers, Inc.

NIOSH. 1977. *Manual of Sampling Data Sheets*, 19 (DHEW Publication No. 77-159). Cincinnati: NIOSH.

OS&HR. June 16, 1977. Washington: Bureau of National Affairs, Inc.

Plunkett, E. R. 1976. *Handbook of Industrial Toxicology*. New York: Chemical Publishing Co., Inc.

Sax, N. I. 1975. *Dangerous Properties of Industrial Materials*. 4th ed. New York: Reinhold Publishing Corporation.

Skalpe, I. O. 1964. *Brit. J. Industr. Med.* 21:69.

Smith, T. J.; et al. 1977. *Am. Rev. Respir. Dis.* 116:31.

Speizer, F. E.; and Frank, N. R. 1966. *Arch. Environ. Health.* 12:725.

Wolff, R. K.; et al. 1975. *Arch. Environ. Health.* 30:521.

Toluene Diisocyanate

NIOSH. 1974. *Criteria for a Recommended Standard—Occupational Exposure to Toluene Diisocyanate*. Cincinnati: NIOSH.

Gafafer, W. M. 1966. *Occupational Diseases—A Guide to Their Recognition*. Washington: U.S. Government Printing Office.

Johnstone, R. T.; and Miller, S. E. 1960. *Occupational Diseases and Industrial Medicine*. Philadelphia: W. B. Saunders.

Malten, K. E.; and Zielhuis, R. L. 1964. *Industrial Toxicology and Dermatology in the Production and Processing of Plastics*. New York: Elsevier.

APPENDIX B

CASE HISTORIES

This section contains two detailed examples of investigations of occupational disease claims, illustrating the application of the decision-making process. To illustrate the types of situations which may arise, the following brief examples are offered:

1. *An obvious occupational disease—*
A disease which occurs commonly in the workplace and a confirmed history of exposure to an agent causing the disease. Medical examination, X-ray, and lung function tests indicate probable silicosis, a disease of the lungs caused by inhalation of dust containing the mineral silica. The worker's past and present job: hard-rock miner. Evidence is presented showing dust exposures in the mine in which he works and at his job are in excess of current standards. There is no question that this is an occupational disease.
2. *An obvious nonoccupation disease—*
A disease occurring commonly in the general population with no occupational agent exposure. Medical examination and laboratory tests diagnose tuberculosis. The worker's past and present job: filing clerk. Investigation shows no other cases of tuberculosis in the office where the worker is employed. This is clearly a nonoccupational disease.
3. *A possible occupational disease and an unknown exposure*
A worker has an anemia and is employed as a spray painter. If the anemia is an *aplastic* anemia, it could be caused by exposure to benzene, a solvent that may be present in some paints. Both the exact type of anemia and the chemical content of the paints used must be investigated to make a decision.

To illustrate this type of situation, where decision-making is more difficult, the following two case histories are offered:

Occupational Disease Case History

Complaint: Malaise, increasing fatigue, and "pins and needles" sensation in the feet.

Medical Evaluation

Evaluation of complaint: Past few days noticed a "pins and needles" sensation in his feet and some weakness of the lower legs. For several weeks or longer he has generally felt weak and tired and not himself. In general he has not been feeling well for quite some time. He has had some weight loss but has not been eating well because of lack of appetite. For a time he has had intermittent periods of nausea and vomiting, but they "come and go." Insomnia and rather frequent headaches have been occurring. Remaining systemic review is negative.

Medical History

General health has always been good. Tonsils and adenoids removed as a child; usual childhood diseases; occasional colds but nothing serious.

Personal History

Age 36, white male, married with children, boy 13 and girl 11. Drinks 8 to 10 ounces of alcohol a day and smokes one pack of cigarettes a day. Lived all his life in Brooklyn, New York. Graduated from high school at age 18. Mother and father and two siblings living and well—mother has diabetes. As a hobby he gardens and has many house plants, but does not use insecticides.

Occupational History

Present occupation: Handyman—works with five other people in a small shop where arts and crafts are made. The work entails mixing pigments and dyes used in printing textiles and for coloring enamels and glazes; generally keeps the shop clean and in order.

Previous occupations: Took two courses of arts and crafts, pottery-making and glazing in high school. Worked part-time as a grocery clerk while in school. After graduation worked for five years as a ship cutter; exposed to lead, asbestos and iron oxide.

Building superintendent, two years. No known exposures to agents but perhaps some polishes, detergents and disinfectants.

Painter, four years. Exposed to pigments found in paint such as lead, chromium and arsenic.

Gardener, three years. Exposed to insecticides and weed killers. Knows that some had pyrethrums, arsenic and parathion-like substances in them.

Present job, four years. Some of the pigments he mixes contain nickel, lead, arsenic, iron and other chemicals. He cleans with a vacuum cleaner, wears no protection and there is some dust.

He has no secondary job.

Clinical Evaluation

The examination revealed a well developed male who appeared tired. His face was pale and the skin over the trunk appeared somewhat pigmented. Examination of the head, eyes, ears and throat showed them to be normal. The nasal septum was inflamed. No adenopathy. The thyroid was normal. Chest expanded symmetrically and percussion and auscultation were normal.

The pulse was 78 and regular, the blood pressure was 128/82. Heart sounds were normal and no evidence of enlargement. There was slight tenderness on palpation of the right upper quadrant but the liver edge was not palpable.

External genitalia was normal. Peripheral circulation was normal. On examination of the extremities a hyperkeratosis of the palms of the hands and soles of the feet were found. There was decreased sensation to touch and vibration in the feet. Patella and ankle reflexes were decreased; those of the wrist and elbow were normal.

Laboratory Evaluation

CBC and Differential:	RBC 4.0 million/cubic mm
	Hb. 12 g/100 ml
	Hct. 40 percent
	WBC 4,000 per cubic mm
Chest X-Ray: 14" x 17"	Normal
Electrocardiogram:	Normal
SMA-12:	Normal

Urinalysis:	Normal
Thyroid function tests:	Normal
Blood Lead:	0.03 mg/100 gms
Urinary Arsenic:	0.9 mg/liter

Epidemiologic Findings

The workplace was surveyed (see Table 1). It was found that the atmosphere contained levels of arsenic in excess of the Occupational Safety and Health Act (OSHA) standards. At breathing level, where the patient worked at mixing the pigments, arsenic levels often were much too high. Dust on the floor and walls contained arsenic and when cleaning, larger than recommended amounts of airborne arsenic were found. Even though pigment containing arsenic was not mixed daily, there was cumulative exposure.

The literature contains ample evidence to indicate that such exposure to arsenic dust could produce arsenic intoxication.

TABLE 1

***ATMOSPHERIC METAL DUST AND
FUME CONCENTRATIONS***
October 1, 1975
ABC ARTS & CRAFTS
ANYTOWN, U.S.A.

SAMPLE NUMBER	LOCATION	TIME START/ STOP	RESULTS IN MILLIGRAMS PER CUBIC METER OF AIR			
			Arsenic	Nickel	Lead	Chrom- ium
OSHA Allowable Limits			0.5	1	0.2	0.5
<i>Operator's Breathing Zone:</i>						
1	John Doe-General Work in stockroom weighing pigments.	0700/ 1900	0.47	<0.001	<0.001	<0.001

2 John Doe-Weighing and mixing pigments.	0900/ 1100	1.33	<0.001	0.021	0.007
3 John Doe-Mixing and packaging pigments; 30 minute lunch.	1100/ 1300	1.21	<0.001	0.050	0.042
4 John Doe-Plant cleanup	1300/ 1500	0.75	<0.001	0.027	0.003
Time-Weighted Average Exposures:		0.94	<0.001	0.025	0.013

<Denotes less than.

It can be clearly seen from Table I that the employee's exposure to arsenic was the only exposure evaluated which exceeded the allowable limit (in this case nearly twice the permitted exposure). Exposures to nickel, lead, and chromium were well within the eight hour time-weighted average limits and continued exposure at the levels evaluated should not result in any health hazards.

Contaminants in the Work Environment

Hyperpigmentation has been reported among employees exposed to arsenic concentrations ranging from 0.110-4.038 milligrams per cubic meter of air (0.562 milligrams per cubic meter was the mean exposure). (Dinman, B.D. 1960. *J. Occ. Med.* 2:137.) This would conform with the clinical evaluation in this specific case where the average exposure to arsenic was 0.94 milligrams per cubic meter of air and hyperpigmentation was observed.

Laboratory findings indicated absorption of arsenic by urinary arsenic levels of 0.9 milligrams per liter. Toxicological data would also imply increased urinary arsenic levels at the atmospheric concentrations evaluated as indicated by the report of an average urinary arsenic level of 0.23 milligrams per liter in workers exposed to mean air concentrations of 0.562 milligrams arsenic per cubic meter.

Conclusion

The differential diagnosis would include lead poisoning, hypothyroidism, anemia and chronic arsenic poisoning; the laboratory findings rule out lead poisoning and hypothyroidism and indicate an absorption of arsenic. Anemia would not account for all of the symptoms and could be part of the pathology of arsenic intoxication.

This history of the complaint, the symptoms and signs along with the laboratory information and the abnormal exposure to arsenic in the workplace, and no evidence of nonoccupational exposure make the diagnosis of chronic arsenic intoxication, occupational in origin.

Nonoccupational Disease Case History

Complaint: Cough of five years duration and shortness of breath.

Medical Evaluation

Evaluation of complaint: About five years ago started to notice a cough that seems to occur during sleeping and at work. He may awaken and raise a mouthful of white, clear sputum. There is no cough on arising in the morning but during the course of the day may bring up 1 or 2 mouthfuls of white sputum—never colored or blood streaked.

He has no dyspnea but states that he does become aware of shortness of breath after climbing 7 or 8 stairs. He can walk 2 or 3 flights slowly but without stopping.

He sleeps without a pillow and has no swelling of the ankles. There is no chest pain or wheezing. He has no history of allergy and there are no other symptoms. The rest of the systemic review is noncontributory. He has never sought medical attention for the cough or shortness of breath during the five years that he has been aware of it.

Medical History

General health always has been good. Tonsillectomy and adenoidectomy at age 7. Usual childhood diseases, no accidents or serious illnesses. He received \$2,000.00 from a

previous employer for dermatitis of the hands (Workers Compensation Insurance). The cause of the dermatitis was never determined nor has it recurred.

Personal History

Age 40, white male, married with one son age 20. Lived in Pennsylvania all his life except while in the Navy when he was stationed in New York. Drinks an occasional beer, never smoked in any form. He quit high school at age 15 after two years. Mother age 62, father age 63, and a brother age 42; all living and well.

Occupational History

Present occupation: Foreman in a warehouse; warehouse adjacent to operation where paper towels, napkins and toilet tissue are printed. Duties consist of general supervision of the warehouse. Exposure to paper dust and ink and oil mist as well as exhaust from trucks at shipping platform.

Previous occupations: Age 14-16, sold newspapers (1949-1951).

Age 16-19, worked as a printer in a printing shop; in contact with paper dust and ink (1951-1954).

Age 19-21, Navy—stationed in New York and worked as a cook. No contact with any hazardous materials except some smoke from cooking (1954-1956).

Age 21-30, warehouseman in charge of ticketing—directing correct merchandise to proper retail stores. In contact with dust and some exhaust from trucks (1956-1965).

Age 30-40, present job—foreman in warehouse operation. The company makes and prints paper towels, napkins, toilet tissue, etc. Warehouse is adjacent to printing operation. There is some paper dust, ink and oil mist as well as exhaust from trucks.

He has no secondary occupation.

Clinical Evaluation

Examination revealed a white male, somewhat overweight, in no acute distress. Skin and hair appear normal. Neck veins not prominent, no cervical adenopathy. No abnormalities of ears, eyes and throat. Nasal septum deviated to the right. Chest is clear to percussion and auscultation. No murmurs or enlargement—A2 = P2. Blood pressure 180/120 right arm; 170/110 left arm. PMI within midclavicular line. Abdomen—no masses or organs palpable. Slight tenderness in left lower quadrant on deep palpation. Right testicle not palpable, inguinal rings firm. No clubbing of the fingers. Small varicosities on left lower leg. No ankle edema. Axillary and inguinal nodes not enlarged. Rectal examination reveals a normal prostate, no masses or other abnormalities palpable. Height 5'9"; Weight 180 lbs.

Laboratory Evaluation

Chest X-ray:	Heart size within normal limits; lung fields clear. Negative.
FVC and FEV:	(Repeated 3 times) within normal limits.
SMA-12:	All chemistries normal.
CBC and Differential:	Normal

Epidemiologic Data

There is no evidence in the scientific literature to indicate that working in this environment is hazardous. Others in the work area have occasional coughs—some with clear sputum production. These men all have negative clinical and laboratory findings. Epidemiologic evidence does exist to show that over 20 percent of the male and 9 percent of the female working population over 25 years of age in the United States have a chronic bronchitis (1970. *N.E.J. Med.* 270:894).

Contaminants in the Work Environment

The workplace was surveyed in 1973 (Table 2, a, b, c). Potential exposures are well within the allowable OSHA limits. The toxicity of carbon monoxide is well known, however, the levels of exposure in this case are far below toxic limits. Oil and ink mist have not been demonstrated to cause specific disease

entities. With very high concentrations animals have developed a chemical pneumonitis. Paper dust has not been found to be toxic, and is considered a nuisance dust.

TABLE 2 (a)
ATMOSPHERIC OIL MIST
PARTICULATE CONCENTRATIONS

October 10, 1973
XYZ VARIETY STORES
SOMETOWN, NY

SAMPLE NUMBER	LOCATION	TIME START/ STOP	RESULTS AS MILLIGRAMS PER CUBIC METER OF AIR
OSHA ALLOWABLE LIMIT			5.0
<i>Operator's Breathing Zone:</i>			
1	Jack White — performing normal duties.	0500/1000	0.25
2	Jack White — performing normal duties.	1000/1200	0.10
3	Jack White — performing normal duties.	1200/1400	1.2
4	Jack White — performing normal duties.	1400/1600	0.55
Time-Weighted Average Exposure			0.52

TABLE 2 (b)**ATMOSPHERIC PAPER DUST CONCENTRATIONS**

October 10, 1973
XYZ VARIETY STORES
SOMETOWN, NY

SAMPLE NUMBER	LOCATION	TIME START/ STOP	RESULTS AS MILLIGRAMS PER CUBIC METER OF AIR
OSHA ALLOWABLE LIMIT (Nuisance Particulates)			15
<i>Operator's Breathing Zone:</i>			
1	Jack White — performing normal duties.	0800/1200	2
2	Jack White — performing normal duties.	1200/1600	1.5
Time-Weighted Average Exposure			1.75

TABLE 2(c)

ATMOSPHERIC CARBON MONOXIDE CONCENTRATIONS

October 10, 1973

XYZ VARIETY STORES
SOMETOWN, NY

SAMPLE NUMBER	LOCATION	TIME START/STOP	RESULTS AS PARTS PER MILLION
OSHA ALLOWABLE LIMIT			50
<i>Operator's Breathing Zone:</i>			
1	Jack White - Paperwork at desk.	0810/0817	5
2	Jack White - Operating LPG Fueled Lift Truck.	0842/0849	20
3	Jack White - Loading platform (all docks filled with trucks).	0955/1002	10
4	Jack White - Same as 3	1131/1138	5
5	Jack White - Working approx. center of whse.	1159/1206	5
6	Jack White - Paperwork at desk.	1310/1317	< 5
7	Jack White - Approx. center of Printing Dept.	1418/1425	<5
8	Jack White - Operating LPG Fueled Lift Truck.	1430/1437	40

9 Jack White - Same as 8	1501/1508	25
10 Jack White - Working approx. center of whse.	1547/1554	<u>10</u>
Approximate Time-Weighted Average Exposure		13

< Denotes less than.

Conclusion

In the face of normal X-ray and pulmonary function studies with no abnormal lung findings on clinical examination, normal blood count and blood chemistries, no adverse epidemiologic or toxicologic evidence and the ambient work environment well below the recommended levels, this case must be considered nonoccupational in origin. There is no evidence to indicate that the worker's symptoms are occupational in origin.

He does, however, have hypertension. Sleeping flat and awakening to expectorate may signify a very early stage of hypertensive heart disease, and some orthopnea would be expected. Having symptoms for five years without ever seeking medical attention seems unusual. The conclusion in this case is that the disease is not bronchitis, but hypertension, and is nonoccupational.

APPENDIX C

SAMPLE RESPIRATORY QUESTIONNAIRE

Use the actual wording of each question. Put X in the appropriate space after each question. When in doubt, record "NO."

PREAMBLE: I am going to ask you some questions mainly about your chest. I should like you to answer 'YES' or 'NO' whenever possible.

YES NO N/A

1. Do you usually cough first thing in the morning or on getting up?

(Count a cough with first smoke or on first going out of doors. Exclude throat clearing or a single cough.)
2. Do you cough like this on most days for as much as three months each year?
3. Do you cough at work?
4. Do you usually bring up some phlegm from your chest first thing in the morning or on getting up?

(Count phelgm with the first smoke or on first going out of doors. Exclude phelgm from the nose. Count swallowed phlegm.)
5. Do you bring up phlegm like this on most days for as much as three months each year?

6. In the past three years, have you had a period of (increased) cough and phlegm lasting 3 weeks or more? ___ ___
7. Have you had more than one such period? ___ ___
8. Does your chest ever feel tight or your breathing become difficult? ___ ___
9. Do you get this apart from colds? ___ ___
- (If YES: specify ... (Interviewer to code))
- (a) With Exercise ___ ___
- (b) At Work ___ ___
- (c) Any Other Time ___ ___
- If disabled from walking by skeletal or other physical disability put 'X' here. ___
10. Are you troubled by shortness of breath, when hurrying on the levels or walking up a slight hill? ___ ___
- (If 'NO' omit questions 11 and 12)
11. Do you get short of breath walking with other people of your own age on level ground? ___ ___
- (If 'NO' omit question 12)
12. Do you have to stop for breath when walking at your own pace on level ground? ___ ___
13. Do you usually have a stuffy nose or catarrh at the back of your nose in the winter? ___ ___
14. Do you have this in the summer? ___ ___
- (If 'NO' to both questions 13 and 14, go to question 16)
15. Do you have this on most days for as much as three months each year? ___ ___

16. During the past 3 years have you had any chest illness which has kept you off work or from your usual activities for as much as a week? _____

17. Did you bring up more phlegm than usual in any of these illnesses? _____

18. Have you had more than one illness with phlegm like this in the last 3 years? _____

HAVE YOU EVER HAD:
(Give relevant details after each positive answer.)

19. An injury or operation affecting your chest? _____

20. Heart trouble? _____

21. Bronchitis? _____

22. Pneumonia? _____

23. Pleurisy? _____

24. Pulmonary Tuberculosis? _____

25. Bronchial Asthma? _____

26. Eczema? _____

27. Dermatitis? _____

28. Pneumoconiosis? _____

29. Byssinosis? _____

30. Other chest troubles? _____

31. Have you ever smoked? _____
(Record 'NO' if subject has never smoked as much as one cigarette a day, or 1 oz. tobacco a month, for as long as one year)

32. Age when stopped ____ years. Was this in the last month? _____
If 'YES' to 31 and 32, fill in figures below:

AMOUNT SMOKED
BEFORE
NOW _____ STOPPING

Cigarettes/day
(Average including weekends) _____

Oz. tobacco/week (handrolled) _____

Cigars/week (large) _____

Cigars/week (small) _____

Occupation (1st Interview Only)

(Record on lines the years in which subject has worked in any of these industries, e.g., 1960-1963)

YES NO

33. Have you ever worked in a dusty job? _____

34. In a coal mine _____

35. In any other mine? _____

36. In a quarry? _____
37. In a foundry? _____
38. In a pottery? _____
39. In a cotton, flax or hemp mill? _____
40. With asbestos? _____
41. In any other dusty job? _____
 If 'YES', specify _____
42. Have you ever been exposed regularly
 to irritating gas or chemical fumes? _____
 If 'YES', give details of nature and
 duration _____

Occupation (Follow-Up only)

43. What is your present job? _____
44. How long have you been doing it? _____
45. What was your previous job in the factory? _____

Taken with minor changes from Operating and Medical Codes of Practice for Safe Working with Toluene Diisocyanate, Health Advisory Committee, British Rubber Manufacturers' Association Ltd.

APPENDIX D

OCCUPATIONS AND SELECTED POTENTIAL EXPOSURES

Occupation	Agent(s)
Abrasive blasters	Silica
Abrasive makers	Silica
Abrasion resistant rubber makers	Tol, diisocyanate
Acetylene workers	Arsenic
Acid dippers	Arsenic nitrogen dioxide
Acid finishers	Lead
Actors	Lead
Acetic acid makers	Carbon monoxide
Acoustical product makers	Asbestos
Acoustical product installers	Asbestos
Adhesive makers	Benzene
Adhesive workers	Tol, diisocyanate
Agriculture	Lead, silica
Airplane dope makers	Benzene
Air filter makers	Asbestos
Aircraft burners	Tol, diisocyanate
Airplane pilots	Carbon monoxide
Alcohol workers	Benzene
Alkali-salt makers	Sulfur dioxide
Alloy makers	Arsenic
Amalgam makers	Mercury
Ammonia makers	Carbon monoxide
Aniline color makers	Arsenic
Aniline makers	Benzene, nitrogen dioxide
Aniline workers	Arsenic
Antimony ore smelters	Antimony
Antimony workers	Antimony
Aniline makers	Benzene nitrogen dioxide
Aniline color makers	Arsenic
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
Arc welders	Carbon monoxide
Arsine workers	Arsenic
Artificial abrasive makers	Carbon monoxide
Artificial gas workers	Carbon monoxide
Asbestos product impregnators	Benzene
Asphalt mixers	Benzene
Asbestos-cement products	Asbestos
Asbestos-cement products users	Asbestos
Asbestos-coating makers	Asbestos
Asbestos-coating users	Asbestos
Asbestos-Grout makers	Asbestos
Asbestos-Grout users	Asbestos
Asbestos-millboard makers	Asbestos
Asbestos-mortar makers	Asbestos
Asbestos-mortar users	Asbestos
Asbestos millers	Asbestos
Asbestos miners	Asbestos
Asbestos-paper makers	Asbestos
Asbestos-paper users	Asbestos
Asbestos-plaster makers	Asbestos
Asbestos-plaster users	Asbestos
Asbestos sprayers	Asbestos
Asbestos workers	Asbestos
Asbestos product impregnators	Asbestos
Auto body shop workers	Lead
Auto garage workers	Silica, nitrogen dioxide, sulfur dioxide, carbon monoxide, asbestos, lead
Auto painters	Nitrogen dioxide
Automotive workers	Benzene, sulfur dioxide, carbon monoxide, nitrogen dioxide
Automobile users	Carbon monoxide
Babbitters	Lead
Babbitt metal workers	Antimony, arsenic
Bactericide makers	Mercury
Barometer makers	Mercury
Battery makers	Mercury
Battery makers, dry	Benzene
Battery workers, storage	Antimony

Bakers	Carbon monoxide
Battery (dry) makers	Benzene
Beaming operators, cotton mill	Cotton dust
Beet sugar bleachers	Sulfur dioxide
Belt scourers	Benzene
Benzene hexachloride makers	Benzene
Benzene workers	Benzene
Bisque-kiln workers	Silica, carbon monoxide
Blacksmiths	Lead, carbon monoxide
Blast furnace workers	Sulfur dioxide, carbon monoxide
Bleaching powder makers	Arsenic
Blueprinters	Nitrogen dioxide
Blast furnace gas users	Carbon monoxide
Blockers (felt hat)	Carbon monoxide
Boiler makers	Mercury
Boiler operators	Arsenic
Boiler water treaters	Sulfur dioxide
Bone extractors	Sulfur dioxide
Bookbinders	Arsenic, lead
Bottle cap makers	Lead
Boiler rooms	Noise
Boiler room workers	Carbon monoxide
Brakelining makers	Benzene, asbestos
Brass cleaners	Nitrogen dioxide
Brass founders	Antimony, carbon monoxide
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
Brewers	Carbon monoxide
Brick burners	Carbon monoxide
Brickmakers	Sulfur dioxide
Buffers	Silica

Buhrstone workers	Silica
Burnishers	Antimony, benzene
Building demolition workers	Asbestos
Busdrivers	Carbon monoxide
Cable makers	Lead
Cable spicers	Antimony, lead, carbon monoxide
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
Carpenter 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
Carbide makers	Carbon monoxide
Carbon monoxide workers	Carbon monoxide
Carders (asbestos)	Asbestos
Caulking compound makers	Asbestos lead
Caulking compound users	Asbestos lead
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
Cellulose makers	Sulfur dioxide
Chemical equipment makers	Lead
Chemical glass makers	Silica
Chemical synthesis	Benzene
Chippers	Lead, silica
Chlorinated paraffin makers	Lead

Chlorine makers	Mercury
Chlorobenzene makers	Mercury
Chlorobenzene makers	Benzene
Chlorodiphenyl makers	Benzene
Charcoal burners	Carbon monoxide
Chauffeurs	Carbon monoxide
Chemical products manufacture	Noise
Chimney masons	Carbon monoxide
Chimney sweepers	Carbon monoxide
Cigar makers	Lead
Cinnebar ore processors	Mercury
Cleaner operators, cotton mill	Cotton dust
Cleaner, cotton mill	Cotton dust
Clutch disc impregnators	Benzene
Clutch facing makers	Asbestos
Coke oven tar chaser	Coke oven emissions
Coke oven workers	Benzene, coke oven emissions, sulfur dioxide, carbon monoxide
Combing machine operators, cotton mill	Cotton dust
Compositors	Antimony
Construction workers	Silica, asbestos, noise
Copper cleaners	Nitrogen dioxide
Copper refiners	Antimony
Copper smelters	Arsenic, sulfur dioxide
Cosmetics makers	Silica
Cotton bleachers	Nitrogen dioxide
Coal miners	Silica
Coal tar refiners	Benzene
Coal tar workers	Benzene
Cobblers	Benzene
Coke oven door cleaners, luteran	Coke oven emissions
Coke oven door machine operators	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
Coal distillers	Carbon monoxide
Cobblers (asbestos)	Asbestos
Coke oven workers	Benzene, carbon monoxide, sulfur dioxide
Colored glass maker	Chromic acid
Compressed air workers	Carbon monoxide
Corrugated paper manufacture	Noise
Crop dusters	Arsenic, lead
Crushers (asbestos)	Asbestos
Cutlery makers	Lead, silica
Cupola workers	Carbon monoxide
Cyclohexane makers	Benzene
Decorators, pottery	Lead
Defoliant applicators	Arsenic
Defoliant makers	Arsenic
Degreasers	Benzene
Demolition workers	Lead, noise
Dental amalgam makers	Mercury
Dental technicians	Lead
Dental workers	Nitrogen dioxide
Dentists	Mercury, nitrogen dioxide
Detergent makers	Benzene
Diamond polishers	Lead
Diatomaceous earth calciners	Silica
Dichlorobenzene makers	Benzene
Diesel equipment operators	Nitrogen dioxide
Diesel engine operators	Sulfur dioxide, carbon monoxide
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
Dippers (acid)	Chromic acid
Divers	Carbon monoxide
Dock workers	Carbon monoxide
Drawing frame operators, cotton mill	Cotton dust
Drug makers	Arsenic, benzene, mercury

Dry cleaners	Benzene
Dryer operators, cotton mill	Cotton dust
Drier workers	Carbon monoxide
Dye makers	Antimony, arsenic, benzene, lead, mercury, nitrogen dioxide, sulfur dioxide
Dyers	Lead
DDT makers	Benzene
Earth moving equipment operators	Noise
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
Electrical equipment manufacture	Noise
Embalmers	Mercury
Embroidery workers	Lead
Emery wheel makers	Lead, silica
Enamel burners	Lead
Enamel makers	Arsenic, lead
Enamellers	Arsenic, benzene, lead, silica, carbon monoxide
Engravers	Benzene
Enamellers	Silica
Engine rooms	Noise
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
Fabricated metal product manufacture	Noise
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, asbestos, carbon monoxide
Fireworks makers	Antimony, arsenic, mercury
Fiberizers (asbestos)	Asbestos
Fireproofers	Asbestos
Fischer-Tropsch process workers	Carbon monoxide
Flameproofers	Antimony
Flint workers	Silica
Flour bleachers	Nitrogen dioxide, sulfur dioxide
Flower makers, artificial	Lead
Flue cleaners	Sulfur dioxide
Flypaper makers	Arsenic
Food bleachers	Nitrogen dioxide, sulfur dioxide
Foundry workers	Antimony, lead, silica, sulfur dioxide
Formeldehyde makers	Carbon monoxide
Foundries	Noise
Foundry workers (core making)	Tol. Diisocyanate
Fruit preservers	Sulfur dioxide
Fruit bleachers	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
Furnace filter makers	Asbestos
Furnace starters	Carbon monoxide
Furnace workers	Carbon monoxide

Furniture workers	Carbon monoxide
Furniture manufacture	Noise
Galvanizers	Arsenic, lead, sulfur dioxide
Garage mechanics	Lead, carbon monoxide, nitrogen dioxide, sulfur dioxide
Gas shrinking operators	Nitrogen dioxide
Gas welders	Nitrogen dioxide
Gas workers, illumination	Benzene, carbon monoxide
Gas station attendants	Carbon monoxide, lead
Gasket makers	Asbestos
Gasoline engine testers	Carbon monoxide
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
Glass manufacture	Noise
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
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
Heat resistant clothing makers	Asbestos

Heat treaters	Carbon monoxide
Hide preservers	Arsenic
Histology technicians	Benzene, mercury
Hydrochloric acid workers	Benzene
Ice makers	Arsenic, sulfur dioxide
Illuminating gas workers	Arsenic
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
Incandescent lamp makers	Lead
Inert filter media workers	Asbestos
Insulation workers	Asbestos, Tol. Diisocyanate
Iron workers	Carbon monoxide
Ironing board cover makers	Asbestos
Isocyanate resin workers	Tol. diisocyanate
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
Kraft recovery furnace workers	Carbon monoxide
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
Laboratory hood installers	Asbestos
Lacquer workers	Tol. diisocyanate
Laggers	Asbestos
Laundry workers	Carbon monoxide

Lead burners	Antimony, arsenic, lead
Lead counterweight makers	Lead
Lead flooring makers	Lead
Lead foil makers	Lead
Lead hardeners	Antimony, lead
Lead mill workers	Lead
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, arsenic
Leather makers	Benzene
Lead mordanters	Antimony
Lead smelters	Arsenic, lead
Leather mordants	Antimony
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
Lift truck operators (propane and gasoline fueled)	Carbon monoxide
Lime kiln workers	Carbon monoxide
Lithographers	Benzene, silica
Lumbering	Noise
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, carbon monoxide
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
Metalizers	Lead
Metal forming	Noise
Metal machining	Noise
Metal working	Noise
Metal polishers	Silica
Metal refiners	Arsenic, sulfur dioxide
Metal oxide reducers	Carbon monoxide
Metal refiners	Carbon monoxide
Methanol makers	Carbon monoxide
Millinery workers	Benzene
Mine workers	Nitrogen dioxide, silica
Miners	Antimony, arsenic, mercury, nitrogen dioxide, silica
Mirror silverers	Benzene, lead
Mine-funnel coaters	Tol. diisocyanate
Mining open pit	Noise
Mining underground	Noise
Monotypers	Antimony
Mordanters	Antimony, arsenic, benzene, lead
Mortar makers	Silica
Mortormen	Silica
Mono process workers	Carbon monoxide
Musical 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
Nitroglycerine makers	Lead

Nitrous acid workers	Benzene, nitrogen dioxide
Nickel refiners	Carbon monoxide
Nickel smelters	Carbon monoxide
Nuclear reactor workers	Lead
Nuclear technologists	Lead
Nurses	Nitrogen dioxide
Nylon & makers	Tol. diisocyanate
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, lead
Ore smelting workers	Sulfur dioxide
Organic chemical synthesizers	Antimony, arsenic, benzene, nitrogen dioxide, carbon monoxide, tol. diisocyanate
Organic sulfonate makers	Sulfur dioxide
Ordnance manufacturing	Noise
Oxalic acid makers	Nitrogen dioxide, carbon monoxide
Oxidized cellulose compound makers	Nitrogen dioxide
Paint makers	Antimony, arsenic, benzene, lead, mercury, sulfur dioxide, asbestos
Paint mixers	Silica
Paint pigment makers	Lead
Painters	Antimony, arsenic, benzene, lead
Paper hangers	Arsenic, lead
Paper makers	Arsenic, mercury, sulfur dioxide
Paraffin processors	Benzene
Patent leather makers	Lead, carbon monoxide
Painters	Antimony, arsenic
Paper manufacture	Noise
Paper products manufacture	Noise
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, lead
Petroleum refinery workers	Benzene
Petroleum refining	Noise
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
Plastic cast bronzers	Antimony
Plastic workers	Arsenic, lead
Plumbers	Arsenic, lead
Plastic manufacture	Noise
Plastic products manufacture	Noise
Plastic foam makers	Tol. diisocyanate
Plasticizer workers	Tol. diisocyanate
Plastic workers	Arsenic
Plastic makers	Asbestos
Policemen	Lead, carbon monoxide
Polish makers	Benzene
Polishing soap makers	Silica
Pottery decorators	Benzene
Pottery glaze dippers	Lead
Pottery glaze mixers	Lead
Pottery workers	Antimony, lead, silica, sulfur dioxide
Pouncers, felt hat	Silica

Polyurethane foam makers	Tol. diisocyanate
Polyurethane foam users	Tol. diisocyanate
Polyurethane sprayers	Tol. diisocyanate
Ore smelters	Arsenic, lead
Ore smelting workers	Sulfur dioxide
Organic chemical synthesizers	Antimony, arsenic, benzene, nitrogen dioxide, carbon monoxide, tol. diisocyanate
Organic sulfonate makers	Sulfur dioxide
Ordnance manufacturing	Noise
Oxalic acid makers	Nitrogen dioxide, carbon monoxide
Oxidized cellulose compound makers	Nitrogen dioxide
Paint makers	Antimony, arsenic, benzene, lead, mercury, sulfur dioxide, asbestos
Paint mixers	Silica
Paint pigment makers	Lead
Painters	Antimony, arsenic, benzene, lead
Paper hangers	Arsenic, lead
Paper makers	Arsenic, mercury, sulfur dioxide
Paraffin processors	Benzene
Patent leather makers	Lead, carbon monoxide
Painters	Antimony, arsenic
Paper manufacture	Noise
Paper products manufacture	Noise
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, lead
Petroleum refinery workers	Benzene
Petroleum refining	Noise
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
Plastic cast bronzers	Antimony
Pottery kiln workers	Carbon monoxide
Power plant operators	Noise
Porcelain workers	Antimony, silica
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
Primary metal processing	Noise
Printing	Noise
Printers	Antimony, benzene
Producer gas workers	Carbon monoxide
Pulpstone workers	Silica
Putty makers	Benzene, lead
Pump packing makers	Asbestos
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	Tenzene, 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, asbestos
Rotogravure printers	Benzene
Roving frame operators, cotton mill	Cotton dust
Roofing materials makers	Asbestos
Rubber buffers	Lead
Rubber cementers	Benzene
Rubber compound mixers	Benzene, lead, silica
Rubber gasket makers	Benzene
Rubber makers	Antimony, benzene, lead
Rubber reclaimers	Lead
Rubber manufacture	Noise
Rubber compounders	Asbestos
Sand cutters	Silica
Sand pulverizers	Silica
Sandblasters	Silica
Sandpaper makers	Silica
Sandstone grinders	Silica
Sawyers	Silica
Sanitation workers	Carbon monoxide
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, benzene
Sewer workers	Carbon monoxide
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
Shingle makers	Asbestos
Ship burners	Tol. diisocyanate
Ship welders	Tol. diisocyanate
Ship builders	Asbestos
Ship demolition workers	Asbestos
Shipbuilding	Noise
Shoe stainers	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, lead, antimony, arsenic
Soap makers	Benzene
Soda makers	Arsenic
Sodium silicate workers	Silica
Sodium sulfite makers	Sulfur dioxide
Soil sterilizer makers	Arsenic
Solder makers	Antimony, lead
Solderers	Antimony, arsenic, lead, carbon monoxide
Solvent makers	Benzene
Solder makers	Antimony, lead
Spacecraft workers	Silica
Spindle pickers cotton	Cotton dust
Spinners, cotton mill	Cotton dust
Spooling operators, cotton mill	Cotton dust
Spinners (asbestos)	Asbestos
Stain makers	Benzene
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, lead
Straw bleachers	Sulfur dioxide
Street sweepers	Silica
Stripper operators, cotton	Cotton dust
Stripper operators, cotton mill	Cotton dust
Styrene makers	Benzene
Steel making	Noise
Steel makers	Carbon monoxide
Stokers	Carbon monoxide
Stone products industries (cement mills)	Noise, silica
Stone workers	Noise, silica
Submarine workers	Arsenic
Subway construction workers	Silica
Sugar refiners	Sulfur dioxide
Sulfite makers	Sulfur dioxide
Sulfur dioxide workers	Sulfur dioxide
Sulfurors, malt and hops	Sulfur dioxide
Sulfuric acid makers	Arsenic, nitrogen dioxide, sulfur dioxide
Sulfuric acid workers	Arsenic, sulfur dioxide
Switch makers	Mercury
Synthetic fiber makers	Benzene
Tanners	Arsenic, lead, mercury, sulfur dioxide
Tar workers	Arsenic
Taxidermists	Arsenic, mercury
Talc miners	Asbestos
Talc workers	Asbestos
Tannery workers	Lead, sulfur dioxide
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
Textile makers	Lead

Textile manufacture	Noise, cotton dust
Textile processors	Tol. diisocyanate
Textile workers	Asbestos, cotton dust
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
Tooth paste makers	Silica
Toll collectors (highway)	Carbon monoxide
Tooth paste makers	Silica
Tree sprayers	Arsenic
Trinitrotoluol makers	Benzene
Traffic controllers	Carbon monoxide
Transportation equipment operators	Noise, carbon monoxide
Trucking	Noise, carbon monoxide
Tube mill liners	Silica
Tumbling barrel workers	Silica
Tunnel construction workers	Silica, noise
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
TDI workers	Tol. diisocyanate
Undercoaters	Asbestos
Upholstery makers	Tol. diisocyanate
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, carbon monoxide
Vine dressers	Arsenic
Vinyl chloride makers	Mercury
Vinyl-asbestos tile makers	Asbestos

Vinyl-asbestos tile installers	Asbestos
Vulcanizers	Antimony, benzene, sulfur dioxide
Wallpaper printers	Arsenic, lead
Warfare gas makers	Arsenic, benzene
Water weed controllers	Arsenic
Wax makers	Benzene
Warehouse workers	Carbon monoxide
Water gas workers	Carbon monoxide
Weavers, cotton mill	Cotton dust
Weed sprayers	Arsenic
Welders	Benzene, lead, nitrogen dioxide
Weavers (asbestos)	Asbestos
Whetstone workers	Silica
Wicker ware bleachers	Sulfur dioxide
Window shade makers	Benzene
Wine makers	Sulfur dioxide, arsenic
Wire drawers	Arsenic
Wire insulators	Benzene
Wire coating workers	Tol. diisocyanate
Wood bleachers	Sulfur dioxide
Wood filler workers	Silica
Wood preservative workers	Arsenic, mercury
Wood pulp bleachers	Sulfur dioxide
Wood stainers	Lead
Wood products manufacture	Noise
Wood preservers	Arsenic
Wood distillers	Carbon monoxide
Zinc chloride makers	Arsenic
Zinc mill workers	Lead
Zinc miners	Antimony, arsenic, lead
Zinc refiners	Antimony, arsenic, lead
Zinc smelter chargers	Lead
Zinc smelters	Sulfur dioxide, lead
Zinc white makers	Carbon monoxide

APPENDIX E

GLOSSARY

Aberrations: Deviations from a normal course.

Acoustic, Acoustical: Containing, producing, or rising from, actuated by, related to, or associated with sound.

Acoustic Trauma: Hearing loss caused by sudden loud noise or by sudden blow.

Acuity: Pertaining to the sensitivity of the senses, such as hearing.

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 tissues produced by inflammation or injury.

Adsorption: The condensation of gases, liquids, or dissolved substances on the surfaces of solids.

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.

Asthenia: Lack or loss of strength: debility.

Asymptomatic: Without symptoms.

Ataxia: Muscular incoordination.

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

Atrophy: Reduction in size.

Attenuation: Lessening.

Audiogram: A test and recording of hearing ability.

Audiometer: An instrument for measuring hearing ability.

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

Basophilia: A pathological reaction of immature erythrocytes to basic dyes whereby the stained cells appear blue, gray, or contain bluish granules.

Basophilic Stippling: A spotted appearance of erythrocytes in Basophilia due to bluish granules.

Benign: Harmless.

Bilirubin: The orange-colored or yellowish bile pigment formed by the breakdown of heme (such as found in hemoglobin) circulated in the plasma and taken up by liver cells.

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 total number of blood cells circulating in the body. A complete blood count totals the number of different kinds of 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 Conduction Test: A hearing test conducted by placing a vibrating tuning fork on the bony portion of the head.

Bone Marrow: The soft tissue of bone which is a 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.
- Bronchiolitis Fibrosa Obliterans*: The closing of the bronchioles with fibrous tissue due to irritation.
- 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 in the lung, 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*: Diseases of the heart muscle.
- Ceiling Limit*: The maximum level of an environmental contaminant which should not be exceeded for any period of time.
- 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, permitting elimination from the body.
- Chemical Cartridge*: The type of absorption unit used with a respirator for removal of low concentrations of solvent vapors and certain gases.
- Chemosis*: Swelling of the mucous membrane of the sclera.

Chromatography: An analytical technique for the separation and identification of chemical compounds.

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.

Coalescence: Fusion of two or more parts.

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

Colorimetry: An analytical technique based on measuring color.

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.

Conductive Hearing Loss: Type of hearing loss not caused by noise exposure, but due to any disorder in the middle ear or external ear that prevents the sound from reaching the inner ear.

Conjunctiva: The membrane that lines the eyelids and covers the exposed surface of the sclera.

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

Consolidation: The act of becoming solid. Used in connection with the solidification of the lungs due to engorgement of the lung tissues, as occurs in acute pneumonia.

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

Coproporphyrin: A porphyrin present in urine and feces.

Cor Pulmonale: Hypertrophy (enlargement) and failure of the right ventricle resulting from disorders of the lungs, pulmonary vessels, or chest wall which involve an increase in pulmonary arterial pressure.

Cornea: The transparent part of the eye.

CPS: Cycles per second (Hertz); a measurement of frequency of sound or any other cyclic phenomenon.

Crackles: A crackling sound heard in the lung during certain diseases.

Creatinine: 1-Methylglycocyanidine, 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, slate-like, 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.

dB(A): Sound level in decibels read on the A-scale of a sound level meter.

Dicibel (dB): A unit used to measure sound intensity.

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.

Direct-Reading Instrument: An instrument which gives an immediate indication of concentration of an airborne contaminant by some means such as a meter or the changing color of a chemical.

Dysphagia: Inability to swallow or difficulty in swallowing.

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 sac (alveoli) have been stretched and broken down.

Emphysematous Bullae: Large blisters on lung surfaces 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.

Epithelioma: Carcinoma of the epithelial cells of the skin.

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: Skin disorder characterized by erythema, the scaling off of dead skin, itching, and loss of hair.

Fasciculation: Small rapid movements of muscle fibers.

FEV₁: Forced expiratory volume in one second; a test of pulmonary function.

Fibrosing Alveolitis: 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, to injury, or to 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 hemoglobin 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.

Hertz: Unit of frequency of sound.

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 blood.

Hyperhidrosis: Excessive sweating.

Hyperkeratosis: Overgrowth of the horny layer of the skin.

Hyperpigmentation: Development of increased skin pimentation.

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 are not derived from hydrocarbons.

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: The final stages of differentiation of cells, tissues, or organs.

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.

Mists: Liquid droplets suspended in air.

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

Mucopolysaccharides: A chief constituent of mucous.

Mucopurulent: Consisting of mucous and pus.

Myalgia: Tenderness or pain in the muscles.

Myeloblastic: A condition where the bone marrow cell develops into a large cell in bone marrow from which blood cells 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 kidney.

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.

Nuisance Dust: An innocuous dust.

Nystagmus: Constant involuntary cyclic 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.

Otologist: A physician who has specialized in surgery and diseases of the ear.

Ototoxic: Drugs which can affect hearing acuity.

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 or endothelial 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 peripheral 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 gastrointestinal and neural disturbances.

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

Phelegm: Thick mucous from the respiratory passages.

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

Platelet: A round or oval disc, 2 to 4 micrometers in diameter, found in the blood of vertebrates, and are concerned with the clotting of blood.

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.

Presbycusis: Hearing loss due to age.

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.

Pyelography: X-ray examination of the renal pelvis and ureter.

Radiomimetic Substance: A substance which imitates the biological effects of ionizing radiation.

Rafter Sample: A sample of dust taken from a rafter or other settling place. Representative of but not identical to dust suspended in air.

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 cross-wise series of streaks.

Subcutaneous: Beneath or to be introduced beneath the skin.

Substernal: Beneath the breastbone.

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.

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.

Tinnitus: A ringing sound in the ears.

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.

APPENDIX F

SAMPLE OSHA REGULATIONS FOR MANDATORY PHYSICAL EXAMINATIONS AND MEDICAL AND BIOLOGICAL MONITORING

MEDICAL SURVEILLANCE

I. Asbestos

(A) Medical Examinations

(1) General

The employer shall provide or make available at his cost, medical examinations relative to exposure to asbestos required by this paragraph.

(2) Preplacement

The employer shall provide or make available to each of his employees, within 30 calendar days following his first employment in an occupation exposed to airborne concentrations of asbestos fibers, a comprehensive medical examination, which shall include, as a minimum, a chest roentgenogram (posterior-anterior 14 X 17 inches), a history to elicit symptomatology of respiratory disease, and pulmonary function tests to include forced vital capacity (FVC) and forced expiratory volume at 1 second ($FEV_{1.0}$).

(3) Annual examinations

On or before January 31, 1973, and at least annually thereafter, every employer shall provide, or make available, comprehensive medical examinations to each of his employees engaged in occupations exposed to airborne concentrations of asbestos fibers. Such annual examination shall include, as a minimum, a chest roentgenogram (posterior-anterior 14 X 17 inches), a history to elicit symptomatology of respiratory disease, and pulmonary function tests to include forced vital capacity (FVC) and forced expiratory volume at 1 second ($FEV_{1.0}$).

(4) Termination of employment

The employer shall provide, or make available, within 30 calendar days before or after the termination of employment of any employee engaged in an occupation exposed to airborne concentrations of asbestos fibers, a comprehensive medical examination which shall include, as a minimum, a chest roentgenogram (posterior-anterior 14 X 17 inches), a history to elicit symptomatology of respiratory disease, and pulmonary function tests to include forced vital capacity (FVC) and forced expiratory volume for 1 second (FEV_{1.0}).

(5) Recent examinations

No medical examination is required of any employee, if adequate records show that the employee has been examined in accordance with this paragraph within the past 1-year period.

(6) Medical records

(i) Maintenance

Employers of employees examined pursuant to this paragraph shall cause to be maintained complete and accurate records of all such medical examinations. Records shall be retained by employers for at least 20 years.

(ii) Access

The contents of the records of the medical examinations required by this paragraph shall be made available, for inspection and copying, to the Assistant Secretary of Labor for Occupational Safety and Health, the Director of NIOSH, to authorized physicians and medical consultants of either of them, and, upon the request of an employee or former employee, to his physician. Any physician who conducts a medical examination required by this paragraph shall furnish to the employer of the examined employee all the information specifically required by this paragraph, and any other medical information related to occupational exposure to asbestos fibers.

II. VINYL CHLORIDE

(B) Medical Surveillance

A program of medical surveillance shall be instituted for each employee exposed, without regard to the use of respirators, to vinyl chloride in excess of the action level. The program shall provide each such employee with an opportunity for examinations and tests in accordance with this paragraph. All medical examinations and procedures shall be performed by or under the supervision of a licensed physician, and shall be provided without cost to the employee.

- (1) At the time of initial assignment, or upon institution of medical surveillance;
 - (i) A general physical examination shall be performed, with specific attention to detecting enlargement of liver, spleen or kidneys, or dysfunction in these organs, and for abnormalities in skin, connective tissues and the pulmonary system.
 - (ii) A medical history shall be taken, including the following topics:
 - (A) Alcohol intake;
 - (B) Past history of hepatitis;
 - (C) Work history and past exposure to potential hepatotoxic agents, including drugs and chemicals;
 - (D) Past history of blood transfusions; and
 - (E) Past history of hospitalizations.
 - (iii) A serum specimen shall be obtained and determinations made of:
 - (A) Total bilirubin;
 - (B) Alkaline phosphates;
 - (C) Serum glutamic oxalacetic transaminase (SGOT);
 - (D) Serum glutamic pyruvic transaminase (SGPT); and
 - (E) Gamma glutamyl transpeptidase.

- (2) Examinations provided in accordance with this paragraph shall be performed at least:
 - (i) Every 6 months for each employee who has been employed in vinyl chloride or polyvinyl chloride manufacturing for 10 years or longer; and
 - (ii) Annually for all other employees.
- (3) Each employee exposed to an emergency shall be afforded appropriate medical surveillance.
- (4) A statement of each employee's suitability for continued exposure to vinyl chloride including use of protective equipment and respirators, shall be obtained from the examining physician promptly after any examination. A copy of the physician's statement shall be provided each employee.
- (5) If any employee's health would be materially impaired by continued exposure, such employee shall be withdrawn from possible contact with vinyl chloride.
- (6) Laboratory analyses for all biological specimens included in medical examinations shall be performed in laboratories licensed under 42 CFR Part 74.
- (7) If the examining physician determines that alternative medical examinations to those required by paragraph (B) (1) of this section will provide at least equal assurance of detecting medical conditions pertinent to the exposure to vinyl chloride, the employer may accept such alternative examinations as meeting the requirements of paragraph (B) (1) of this section. If the employer obtains a statement from the examining physician setting forth the alternative examinations and the rationale for substitution. This statement shall be available upon request for examination and copying to authorized representatives of the Assistant Secretary and the Director.

SUPPLEMENTARY MEDICAL INFORMATION

When required tests under paragraph (B)(1) of this section show abnormalities, the tests should be repeated as soon as practicable, preferably within 3 to 4 weeks. If tests remain abnormal, consideration should be given to withdrawal of the employee from contact with vinyl chloride, while a more comprehensive examination is made.

Additional tests which may be useful:

- A. For kidney dysfunction: urine examination for albumin, red blood cells, and exfoliative abnormal cells.
- B. Pulmonary system: Forced vital capacity, forced expiratory volume at 1 second, and chest roentgenogram (posterior-anterior, 14 X 17 inches).
- C. Additional serum tests: Lactic acid dehydrogenase, lactic acid dehydrogenase isoenzyme, protein determination, and protein electrophoresis.
- D. For a more comprehensive examination on repeated abnormal serum tests; Hepatitis B antigen, and liver scanning.

III. CARCINOGENS

4-Nitrobiphenyl
Alpha-Naphthylamine
Methyl chloromethyl ether
3,3' - Dichlorobenzidine (and its salts)
bis-Chloromethyl ether
beta-Naphthylamine
Benzidine
4-Aminodiphenyl
Ethleneimine
beta-Propiolactone
2-Acetylaminofluorene
4-Dimethylaminoazobenzene
N-Nitrosodimethylamine

(C) Medical Surveillance

At no cost to the employee, a program of medical surveillance shall be established and implemented for employees considered for assignment to enter regulated areas, and for authorized employees.

(1) Examinations

- (i) Before an employee is assigned to enter a regulated area, a preassignment physical examination by a physician shall be provided. The examination shall include the personal history of the employee, family and occupational background, including genetic and environmental factors.**
- (ii) Authorized employees shall be provided periodic physical examinations, not less often than annually, following the preassignment examination.**
- (iii) In all physical examinations, the examining physician shall consider whether there exist conditions of increased risk, including reduced immunological competence, those undergoing treatment with steroids or cytotoxic agents, pregnancy and cigarette smoking.**

(2) Records

- (i) Employers of employees examined pursuant to this paragraph shall cause to be maintained complete and accurate records of all such medical examinations. Records shall be maintained for the duration of the employee's employment. Upon termination of the employee's employment, including retirement or death, or in the event that the employer ceases business without a successor, records, or notarized true copies thereof, shall be forwarded by registered mail to the Director.**
- (ii) Records required by this paragraph shall be provided upon request to authorized representatives of the Assistant Secretary or the Director; and upon request of any employee or former employee, to a physician designated by the employee, or to a new employer.**

- (iii) Any physician who conducts a medical examination required by this paragraph shall furnish to the employer a statement of the employee's suitability for employment in the specific exposure.

MEDICAL HISTORY EMPHASIS ITEMS

1. Previous exposures to benzene and any other hematologic toxin; blood dyscrasias including genetically related hemoglobin alterations, bleeding abnormalities and abnormalities in the function of formed blood elements; renal disease; liver disease; alcoholic intake and infection.
2. Respiratory symptoms, i.e, breathlessness, cough, sputum production and wheezing.
3. Nausea, vomiting, visual disturbances and use of alcohol and barbiturates.
4. G. I. symptoms and mental status. Review alcohol consumption.
5. Skin or pulmonary sensitization or a skin or mucous membrane condition that may promote response to chromic acid.
6. Potential skin or pulmonary sensitization, a skin or mucous membrane condition that may be exacerbated by chromium (VI), smoking habits and history of liver or kidney disease.
7. Presence and degree of respiratory symptoms (breathlessness, cough, sputum production and wheezing.
8. Respiratory allergy, chronic obstructive lung disease, cardio-pulmonary symptoms, smoking. (Respiratory questionnaire included).
9. Respiratory disease.
10. Respiratory and renal disease.
11. Musculoskeletal, pulmonary and G. I. symptoms and kidney dysfunction.

12. **Emphasis on signs or symptoms of unacceptable mercury absorption such as loss of weight, sleeplessness, tremors, personality change or other evidence of CNS involvement.**
13. **Occurrence of headache, dizziness, fatigue, pain in the limbs and irritation of the skin and eyes.**
14. **Respiratory symptoms, i.e., breathlessness, cough, sputum production, wheezing and tightness in the chest. Smoking history.**
15. **Headaches, nausea, or G.I. disturbance, dizziness, alcohol consumption. Particular attention to eye mucous membrane or skin irritation.**
16. **Preexisting disorders of the skin, respiratory tract, liver, and kidneys.**