A GUIDE TO THE WORK-RELATEDNESS OF DISEASE

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health

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PREFACE

The goal 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 this program are NIOSH efforts that are directed toward the identification of those disease conditions that are causally related to occupation, as necessary prerequisite to their prevention.

This guide is presented primarily as an aid to State agencies and others concerned with occupational disease compensation. The Guide 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 five 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 for improvement of the Guide based upon experience with its use.

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 five 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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THE WORK-RELATEDNESS OF DISEASE

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, burdened with this narrow interpretation of law, found that compensation through the courts was seldom satisfactory.

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

Although the new laws established a more equitable compensation system, substantial changes 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 illness 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.

In contrast with 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 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 five 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 unarguable 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 epidemiological data.
3. Consideration of evidence of exposure.
4. Consideration of validity of testimony.
5. Consideration of other relevant factors.
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 <u>medical evaluation</u> 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

-- 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)

-- 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)

-- 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 determing 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

-- 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 9).

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.

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 tests, pulmonary function tests)

CHAPTER III - EPIDEMIOLOGIC DATA

Epidemiology is the branch of medical science that deals with causes 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 causative factors in 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, coalworkers' 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 last turn of the 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 the suspected agent have sustained certain types of illnesses may be extremely helpful in establishing the fact that the substance in question has been shown to cause 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 epidemiological 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, used, or used in operations in nearby areas;

2. any industrial hygiene studies available, 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 <u>as performed by the worker</u> 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 (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 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 MESA.

-- were the respirators fitted properly? Leaks in the facepiece negate effectiveness.

-- did employee use the respirators?

-- were cartriges, 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?

-- proper washing facilities available?

Exposure evaluation

The best evidence to confirm the exposure of a worker to an agent are 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 nonconsecutive 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 work day, 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.

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 the following page lists some agents which may contribute to disease and aggravation of disease.

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

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 abbatoir 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.

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, 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) 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 causing them.

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 on the preceding pages. In most cases, physicians will provide testimony on medical conditions and laboratory and other medical tests; industrial hygienists will testify concerning evidence of exposure. They or epidemiologists give testimony on epidemiological data, depending upon the technical areas covered. 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, epidemiological, 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, an occupational health physician 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 nonphysicians 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 by 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, audiology)?

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?
   (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?
   (scientific literature, epidemiological studies, special sampling, replication of work conditions)

5. Has non-occupational 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 five selected disease-producing agents, to illustrate the use of the decision making method previously described.

ASBESTOS was selected as an example of an agent that may lead to cancer of the membrane lining the chest cavity (pleural mesothelioma) or the abdominal cavity (peritoneal mesothelioma), as well as the better known dust disease, asbestosis.

CARBON MONOXIDE represents a very common industrial poison where internal combustion engines are in use, and it is also a hazard from nonoccupational exposures and affects oxygenation of blood.

INORGANIC LEAD was selected because it produces damage to both the nervous system and the blood forming organs, and because so many occupations and hobbies are potential sources of exposure.

NOISE represents a somewhat different type of agent that is also both occupational and nonoccupational. Furthermore, the effect of excessive noise (hearing loss) is a common manifestation of aging.

TOLUENE DIISOCYANATE is a good example of a potent respiratory irritant and sensitizer.

Different and additional agents could have been presented, and consideration will be given to such a 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, central nervous system, blood forming (hematopoietic) organs, and systemic effects (liver), as well as carcinogenic action. The organization of the agent material can serve as a guide in collecting pertinent information about other disease-producing agents.

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)
Amosite
Tremolite
Crocidolite (blue asbestos)
Anthophyllite

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 ventilating 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 verruc a). 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 increase the risk of lunk 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
Acoustical Product Installers
Air filter makers
Asbestos-cement products makers
Asbestos-cement products users
Asbestos-coatings makers
Asbestos-coatings users
Asbestos-grout makers
Asbestos-grout users
Asbestos-millboard makers
Asbestos-millboard users
Asbestos-mortar makers
Asbestos-mortar users
Asbestos millers
Asbestos miners
Asbestos-paper makers
Asbestos-paper users
Asbestos-plaster makers
Asbestos-plaster users
Asbestos sprayers
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
Cobbers (asbestos)
Construction workers
Crushers (Asbestos)
Fiberizers (Asbestos)
Fireproofers
Firemen
Furnace filter makers
Gasket makers
Heal resistant clothing makers
Insulation workers
Inert filter media workers
Ironing board cover makers
Laboratory hood installers
Laggers
Paint makers
Pipe insulators
Plastics makers
Pump packing makers
Roofers
Roofing materials makers
Rubber compounders
Shingle makers
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

(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, can be useful in evaluating the extent and importance of respiratory symptoms such as:

   - breathlessness
   - phlegm (sputum) production
   - chest pain
   - cough
   - 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, slatelike or dark purple)
- decrease of chest expansion
- digital clubbing (rounding of the ends, and swelling of the fingers and/or toes)
- sequelae (other resultant diseases) including cor pulmonale (right heart failure), bronch ogenic 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

- 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.

- in addition, pericardial effusion (fluid in the heart cavity) may occur, causing tamponade.

Laboratory

(See Decision-Making Process)

Additional tests 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. (Appendix B)

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

- 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

- Decreased lung compliance

An exercise test will result in an increased amount of air required during physical effort, 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

Epidemiological Data

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 epidemiological 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:

Enterline[1] 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 indicates 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 NIOSH[2].

Murphy[3] 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, shortness of breath on climbing one flight of stairs.

The Pennsylvania Department of Health[4] reported a study of asbestos dust concentrations in two plants (one studies from 1930-1967 and the other from 1948-1968). 64 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.

Epidemiological evidence is also available relating the development of mesothelioma with exposure to asbestos. Selikoff[5,6] reported 14 deaths from mesothelioma in 532 abestos 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 Act (OSHA) establishes maximum allowable limits for asbestos fibers greater than five micrometers (um) in length. OSHA limits such asbestos fibers to no more than five 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 five 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 1-6, 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 finding;s 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.

CARBON MONOXIDE

Introduction

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

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

The blood of cigarette smokers contains between 3 and 10 percent carboxyhemoglobin (COHb) depending on the number of cigarettes smoked and the manner of smoking, inhaling or not inhaling. During smoking, the individual is being exposed to the 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

Furnace workers
Garage mechanics
Gas workers (illuminating)
Gasoline engine testers

Occupations with Potential Exposures to Carbon Monoxide (Cont.)

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
Chauffers
Chimney masons
Chimney sweepers
Coal distillers
Coke oven workers
Cupola workers
Diesel engine operators
Compressed air workers
Divers
Dock Workers
Drier workers
Fireman
Enamelers
Fischer-Tropsch Process work
Formaldehyde makers
Foundry workers
Furnace starters

Gas station attendants
Heat treaters
Iron workers
Kraft recovery furnace workers
Laundry workers
Lift truck operators (Profane 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

(See also Decision-Making Process)

In the medical history, the following should be considered:

- neurological diseases

- 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 behicle 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
- faulty auto exhaust system

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 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
- 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

(See Decision-Making Process)

Additional tests 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
- electroencephalogram may show focal and diffuse epileptiform (resembling epilepsy) changes which later disappear

Epidemological Data

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.

Carbon monoxide

Many of the reports dealing with carbon monoxide (CO) toxicity are in terms of carboxyhemoglobin (COHb) percentage in blood. The percent of COHb depends on many factors including CO concentrations in air, total time of exposure to various air concentrations of CO, diffusion rate of CO through the lungs, ventilation rate, type of activity being done, metabolic rate, barometric pressure and temperature. NIOSH[7] recommends an allowable level for CO of 35 ppm based on an 8 hour time-weighted average exposure so that COHb percent does not exceed five. The current allowable limit of 50 ppm CO based on an eight hour time-weighted average exposure is designed to maintain COHb less than 10%.

SYMPTOMS CAUSED BY VARIOUS AMOUNTS OF CARBON MONOXIDE HEMOGLOBIN IN THE BLOOD[8]

BLOOD SATURATION o/o COHb	SYMPTOMS
0-10	No symptoms.
10-20	Tightness across forehead, possible slight headache, dilation of cutaneous blood ve ssels.
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 o/o EQUILIBRIUM VALUE OF BLOOD SATURATION

CO IN AIR ppm	BLOOD SATURATION o/o (80 o/o of Approx. Equil. Values)	TIME (Hours)
200-300	23-30	5-6
400-600	36-44	4-5
700-1000	47-53	3-4
1100-1500	55-60	1½-3
1600-2000	61-64	1-1½
2100-3000	64-68	½-3/4
3100-5000	68-73	20-30 Min.
5000-10000	73-76	2-15 Min.

There have been a number of reports showing evidence of behavioral effects in man on exposure to low levels of CO. The results of these studies indicate that exposure to low concentrations of CO could affect a worker's ability to work safely. McFarland[9] reported difficulties in visual discrimination at 5°/o COHb (similar results were reported by Halperin[10]. Horvath[11] reported significantly impaired vigilance at 6.6°/o COHb. Schulte[12] indicated various physiological and behavioral tests were effected by COHb levels as low as 5°/o. Beard [13,14] in two reports showed exposure to CO in concentrations ranging from 50-250 ppm caused a deterioration in the ability to discriminate auditory stimuli and exposures to 50 ppm caused impairment in time discrimination. Trouton[15] reported impairment in muscle limb coordination at COHb levels of approximately 5°/o. There have been a number of studies made relating carbon monoxide exposures to cardiovascular ramifications. NIOSH [16] concludes that the results of these studies provide sufficient evidence so that "based on cardiovascula r 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 (carobxyhemoglobin) in excess of 5°/o 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 detectors 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 Act (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, Toxicological Data, Appendix D.

Conclusion

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

1. confirmed history of occupational exposure to carbon monoxide monoxide

2. carboxyhemoglobin in excess of 10 percent

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

3. continuous and complete unconsciousness for at least six hours after return to fresh air

INORGANIC LEAD

Introduction

Lead is a naturally occurring element found in some quantity in the human body. This Guide discusses only inorganic lead which, in industry, is usually absorbed into the body through inhalation of dust or fumes. Nonoccupational exposure may occur through ingestion, e.g. lead etched from the glaze of pottery used for food, paint from water pipes, or from food contaminated with lead.

Although lead may be absorbed into the body, absorption does not necessarily constitute lead poisoning. Body burden will affect individual tolerance. At a given body burden, some persons may have signs and symptoms of lead poisoning while others do not.

Lead may be stored in the body and, following an illness or some stress factor, be released into the system and produce symptoms of lead poisoning.

Lead and its compounds have numerous chemical and common names:

Chemical name	Common Names
lead	plumbum
lead acetate	normal lead acetate, sugar of lead, salt of Saturn
lead antimonate	Naple's yellow, antimony yellow
lead borate	
lead bromide	
lead butyrate	butyric acid lead salt
lead carbonate	basic lead carbonate, lead subcarbonate, white lead, flake lead, ceruse, cerussa, cerussite
lead chlorate	
lead chloride	cotunnite, matlockite
lead chromate	chrome yellow, Cologne yellow, King's yellow, Leipzig yellow, Paris yellow, crocoite

Chemical Name	Common Names
lead chromate oxide	basic lead chromate, red lead chromate, chrome red, Persian Red, Austrian cinnabar
lead citrate	
lead cyanide	
lead dioxide	lead oxide brown, lead superoxide, lead peroxide, plumbic acid anhydride, plattnerite
lead fluoride	
lead hexafluorosilicate	lead fluorosilicate, lead silicofluoride
lead hydroxide	basic lead hydroxide, lead hydrate, hydrated lead oxide
lead iodide	
lead metaborate	
lead metasilicate	alamosite
lead molybdate	wulfenite
lead monoxide	lead oxide yellow, plumbus oxide, litharge, massicot, lead protoxide
lead nitrate	
lead nitrite	
lead phosphate	pyromorphite
lead phosphite	
lead sesquioxide	lead trioxide, plumbus plumbate
lead sulfate	anglisite
lead sulfide	galena, plumbous sulfide
lead tartrate	
lead telluride	altaite
lead tetrafluoride	plumbing fluoride

Chemical Name	Common Names
lead tetraoxide	lead oxide red, red lead, minimum, lead orthoplumbate, mineral orange, mineral red, Paris red, Saturn red
lead thiocyanate	lead sulfocyanate
lead thiosulfate	lead hyposulfate
lead tungstate	raspite, scheelite, stolzite, lead wulframate
lead vanadate	lead metavanadate, vanadinite

Some Occupations with Potential Lead Exposures

Acid finishers
Actors
Babbiters
Battery makers
Blacksmiths
Bookbinders
Bottle cap makers
Brass founders
Brass polishers
Braziers
Brick burners
Brick makers
Bronzers
Brushmakers
Cable makers
Cable splicers
Canners
Cartridge makers
Chemical equipment makers
Chlorinated Paraffin makers
Chippers
Cigar makers
Crop dusters
Cutlery makers
Decorators (pottery)
Demolition workers
Dental technicians
Diamond polishers
Dye makers
Dyers
Electronic device makers
Electroplaters
Electrotypers
Embroidery workers
Emery wheel makers
Enamel burners
Enamelers
Enamel makers
Explosives makers
Farmers
File cutters
Firemen
Flower makers (artificial)
Foundry workers
Galvanizers
Garage mechanic
Glass makers
Glass polishers
Glost kiln workers
Gold refiners
Gun barrel browners
Incandescent lamp makers
Ink makers
Insecticide makers
Insecticide users
Japan makers
Japanners
Jewellers
Junk metal refiners
Labelers (paint can)

Occupations with Potential Lead Exposures (cont.)

Lacquer makers	Policemen
Lead burners	Pottery glaze mixers
Lead counterweight makers	Pottery glaze dippers
Lead flooring makers	Pottery workers
Lead foil makers	Putty makers
Lead Mill Workers	Pyroxylin-plastics workers
Lead miners	Riveters
Lead pipe makers	Roofers
Lead salt makers	Rubber buffers
Lead shield makers	Rubber makers
Lead smelters	Rubber reclaimers
Lead stearate makers	Scrap metal workers
Lead workers	Semiconductor workers
Linoleum makers	Service station attendants
Linotypers	Sheet metal workers
Linseed oil boilers	Shellac makers
Lithographers	Ship dismantlers
Lithotransfer workers	Shoe stainers
Match makers	Shot makers
Metal burners	Silk weighters
Metal cutters	Slushers (porcelain enameling)
Metal grinders	Solderers
Metal polishers	Solder makers
Metal refiners	Steel engravers
Metal refinishers	Stereotypers
Metallizers	Tannery workers
Mirror silverers	Television picture tube makers
Musical instrument makers	Temperers
Nitric acid workers	Textile makers
Nitroglycerin makers	Tile makers
Painters	Tin foil makers
Paint makers	Tinners
Paint pigment makers	Type founders
Paper hangers	Type setters
Patent leather makers	Vanadium compound makers
Pearl makers (imitation)	Varnish makers
Pharmaceutical makers	Vehicle tunnel attendants
Photography workers	Wallpaper printers
Pipe fitters	Welders
Plastic workers	Wood stainers
Plumbers	Zinc mill workers
Printers	Zinc smelter chargers

Medical Evaluation

(See also Decision-Making Process)

In the personal history, consider the following:

- 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, but it is not occupational. Common nonoccupational lead exposures include:

- ceramics, pottery and related hobbies
- electronics and related hobbies involving extensive soldering
- firing ranges
- hunting (especially those who cast their own bullets)
- eating or drinking from improperly fired lead-glazed ceramic tableware
- eating lead-bearing paint (especially children)
- burning battery casings
- consuming illicitly distilled whiskey
- extensive auto driving (especially in cities)
- extensive work with motor fuels
- painting with lead-containing paints
- home plumbing repairs (lead pipe systems)
- exterminating

Signs and Symptoms

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

- malaise
- fatigue
- sleep disturbance
- irritability
- aching muscles and bones

Signs and Symptoms (Cont.)

- headache
- nausea and vomiting
- loss of appetite
- constipation
- abdominal cramps

In more advanced cases of lead poisoning, the above signs and symptoms progress and frequently involve the gastro-intestinal and neuro-muscular systems (both nerves and muscles), and the kidneys (Fanconi Syndrome).

Gastro-intestinal signs and symptoms are:

- severe abdominal pain (lead colic)
- constipation (never diarrhea)
- marked loss of appetite (anorexia) leading to weight loss
- characteristic lead line of the gums may be present, usually with pyorrhea

Neuro-muscular and neuro-behavioral symptoms are:

- generalized tenderness or pain in the muscles (myalgia)
- muscular weakness, especially of the most frequently used muscles
- tremors or palsy may be present
- decreased hand grip strength
- characteristic "wrist drop" (wrist flexed and cannot be extended because of nerve involvement)
- the peripheral nerves of the upper extremities are involved; rarely those of the lower extremities

Reproductive:

- decreased fertility in men and spontaneous abortion in women have been reported

A most severe form of lead poisoning is lead encephalopathy, impairment of the brain due to lead poisoning. Lead or Saturnine encephalopathy is rarely seen today because of improved techniques of handling lead. It is a vague term and includes coma, delirium, psychosis, convulsions; muscles affecting speech, eyes, and face are often involved. It can result in blindness and death.

Laboratory

Signs pertaining to lead's effect on the blood forming organs (hematopoietic 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.

Abnormal laboratory values that may be found in lead poisoning:

- decreased red blood count
- decreased hemoglobin and hematocrit
- decreased motor nerve conduction velocity
- increased urinary delta-aminolevulinic acid
- increased free erythrocyte protoporphyrin (FEP) and zinc protoporphyrin (ZP or ZPP)
- increased lead in blood
- increased lead in urine

NOTE: Results of blood and urine laboratory analyses for lead are subject to a 10-15 percent 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.

Epidemiologic Data

There is vast and detailed scientific literature providing evidence of lead poisoning in workers with significant exposure. Neuropathies, nephropathy and blood changes are well documented. Lead absorption, however, does not necessarily indicate poisoning.

Lane[18] reported a study of nine lead workers in a storage battery industry who had been exposed to lead concentrations in air around 0.5 milligrams per cubic meter of air for over 20 years. All died from hypertension and renal failure between the ages of 42 and 52.

Williams, King and Walford[19] report the following data taken in table form from the Criteria Document on lead, published by the National Institute for Occupational Safety and Health.

AIR LEAD CONC. IN MILLIGRAMS PER CUBIC METER	BLOOD LEAD MILLIGRAMS PER 100 MILLILITERS	URINE LEAD MILLIGRAMS PER LITER	URINE COPRO-PORPHYRIN (DONATH)	URINE ALA* MILLIGRAMS PER 100 MILLILITERS
0.20	0.070 (0.048-0.092)	0.143 (0.056-0.230)	4.2 (2.4-6.0)	1.8 (0.3-3.3)
0.15	0.060 (0.038-0.082)	0.118 (0.031-0.205)	3.6 (1.8-5.4)	1.4 (0.1-2.9)

*ALA values were determined by a method which probably gives higher values than do other methods, thus a high "normal" value.

The figures in the top lines indicate mean values.

(It is recommended[20] that blood lead levels greater than 0.060 milligrams lead per 100 grams whole blood is indicative of unacceptable lead absorption and that urine lead levels of 0.20 milligrams lead per liter of urine or greater is indicative of unacceptable lead absorption.)

Elkins 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 or urine would, averaging, correspond to an air concentration of 0.2 milligrams lead per cubic meter of air.

Hartogenesis andZielhuis[21] report blood changes in workers exposed to lead chromate dust in concentrations greater than 0.2 milligrams per cubic meter of air as lead. They further report doubtful changes in blood at exposures to atmospheric concentrations between 0.1 and 0.2 milligrams per cubic meter as lead.

The following data relating average blood lead content with exposure and duration of employment has been adapted from Dreessen et al[22] Committee on Biologic Effects of Atmospheric Pollutants,[23] and the National Institute of Occupational Safety and Health.[20]

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 MORE
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

DURATION OF LEAD EXPOSURE	AIR LEAD CONTENT MILLIGRAMS PER CUBIC METER (Continued)			
YEARS 5-9				
Number	10	13	40	20
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

Evidence of Exposure

Air Sampling and Analysis

There are three commonly accepted methods of lead air sampling:

1. impingement
2. electrostatic precipitation
3. mechanical filtration

There are three commonly accepted methods to analyze the samples for presence of lead:

1. atomic absorption spectrophotometry
2. colorimetrically using the dithizone method
3. polarographically

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

The Occupational Safety and Health Adm.(OSHA) limits exposure to lead and its inorganic compounds (except lead arsenate) to 0.2 milligrams per cubic meter of air based on an eight hour time-weighted average exposure.

The American Conference of Governmental Industrial Hygienists threshold limit value for lead and its inorganic compounds (except lead arsenate) is 0.15 milligrams per cubic meter of air based on an eight hour time-weighted average exposure.

Conclusion

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

1. confirmed history of occupational exposure to lead

2. findings compatible with lead poisoning

3. increased lead in blood and/or urine

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

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, 1000 and 2000 Hz. (Sataloff, J.; and Michael, P. 1973. Hearing Conservation 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 and the visual, presbyopia. 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 prexence 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 of 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
Chemical products manufacture
Construction
Corrugated paper manufacture
Demolition
Earth moving equipment operators
Electrical equipment manufacture
Engine rooms
Fabricated metal product manufacture
Farm equipment operators
Food processing
Foundries
Furniture manufacture
Glass manufacture
Lumbering
Metal forming
Metal machining
Metal working
Mining - Open pit
Mining - Underground
Ordinance manufacturing
Paper manufacture
Paper products manufacture
Petroleum refining
Plastics manufacture
Plastic products manufacture
Power plant operators
Printing
Primary metal processing
Quarrying
Rubber manufacture
Rubber products manufacture
Shipbuilding

Occupations with Potential Exposures to Noise (Cont.)

Steel making
Stone products industries (cement mills)
Stone workers
Textile manufacture
Transportation equipment operators
Trucking
Tunneling
Wood products manufacture

Medical Evaluation

(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
   - 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
- operating motorcycles, snowmobiles or boats

Clinical Evaluation

Other Tests

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

- external ear examination for scars or malfunctions
- otoscopic examination 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
- 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.

Laboratory

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
- exposure to intense noise within 18 hours or less prior to the test

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
- changes occur in routine audiometric examination

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

Epidemiological data

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 NIOSH.[24] 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 Knight[25] 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 Jones[26] 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."

Schneider[27] 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 Zlamal[28] reported a study of noise in the cabs of heavy trucks ranging from 90-110 dB. Examinations made on 51 truck drivers and in each case a loss of hearing was determined."

Mancini and Stancari[29] 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." Chadwick[30] 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 2000-4000 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."

Filin[31] reported a study of drivers of self-propelled jumbos in underground ore mining. "Noise levels of 127 dB at frequencies between 1000 Hz and 8000 Hz. Hearing loss in 91 of 135 miners examined; after 10 years' work, 28 dB loss at 4000 Hz."

Weston[32] 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."

Cohen[33] 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."

Burns[34] 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."

Stone[35] reported a study of "3116 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 standarized 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 Act (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 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 C1/T1 + C2/T2 + ... Cn/Tn exceeds unity, then the mixed exposure should be considered to exceed the limit value. Cn indicates that the total time of exposure at a specified noise level, and Tn 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

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.

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, violen t 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 maximym 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
Adhesive workers
Aircraft burners
Foundry workers (core making)
Insulation workers
Isocyanate resin workers
Lacquer workers
Mine tunnel coaters
Nylon makers
Organic chemical symthesizers
Plastic foam makers
Plasticizer workers
Polyurethane foam makers
Polyurethane foam users
Polyurethane sprayers
Ship burners

Occupations with Potential Exposures to Toluene Diisocyanate (Cont.)

Ship welders
Spray painters
Textile processors
TDI workers
Upholstery makers
Wire coating workers

Medical Evaluation

(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 estent and importance of respiratory symptoms, such as:

   - breathlessness
   - sputum production
   - chest pain
   - cough
   - 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.

The reactions encountered with inhalation of TDI vapor are:

1. primary irritation to which all exposed persons are susceptible to some degree

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)
- 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
- 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
- neurological (nervous) and psychiatric (mental) 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

- chronic bronchitis, emphysema and corpulmonale (right heart failure)

Laboratory

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 (FEV_1)

- forced vital capacity (FVC) is decreased

Chest X-ray--findings are nonspecific. Corresponding changes will be seen if there is a bronchopneumonia 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

Epidemiological Data

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,[36] and any further exposure may be extremely dangerous. This should be kept in mind when considering the following data.

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

PLANT	YEAR	AIR ANALYSIS: NUMBER OF TESTS	AIR ANALYSIS: AVERAGE TDI CONCENTRATION (ppm)	WORKERS EXPOSED	NUMBER OF CASES: ACCEPTED ESTABLISHED	NUMBER OF CASES: QUESTIONABLE OR DISPUTED
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	4	-	-
8	1961	5	0.001*than)	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.

There is a report of a study of 12 workers in an automobile plant making polyurethane foam crashpads.[36] 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 Virchow[37] 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.

Elkins[38] published a report on a 5-year study of TDI exposure in 15 plants. The author concluded that 0.01 ppm for TDI was "a not unreasonable limit." Elkin's data is summarized in the following table:

Glass and Thom[39] 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).

Williamson[40] 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.

Maxon[41] 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 hemoptysis. There was evidence that 4 workers had become sensitized to TDI.

Bruckner et al[42] 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 muscous 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.

Peters[43] 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 0.000-0.0120ppm. 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.

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
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 Act (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.

See References 95-103, Toxicological Data, Appendix D.

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
4. progressive increase in eosinophil count

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B. JOBS AND POTENTIAL EXPOSURES

OCCUPATION	AGENT(S)
Abrasive blasters	Silica
Abrasives makers	Silica
Abrasion resistant rubber makers	Tol. diisocyanate
Acetic acid makers	Carbon monoxide
Acetylene workers	Arsenic
Acetylene purifiers	Chromic acid
Acid finishers	Lead
Acid dippers	Arsenic nitrogen dioxide
Acoustical product makers	Asbestos
Acoustical Product installers	Asbestos
Actors	Lead
Adhesive workers	Tol. diisocyanate
Adhesive makers	Benzene
Air filter makers	Asbestos
Aircraft burners	Tol. diisocyanate
Airplane Dope makers	Benzene
Airplane pilots	Carbon monoxide
Alcohol workers	Benzene
Alkali-salt makers	Sulfur dioxide
Alloy makers	Arsenic
Aluminum anodizers	Chromic acid
Aluminum hard coaters	Chromic acid
Ammonia makers	Carbon monoxide
Aniline makers	Benzene Nitrogen dioxide
Aniline color makers	Arsenic
Aniline workers	Arsenic
Antimony Ore Smelters	Antimony
Antimony workers	Antimony
Arc welders	Carbon monoxide
Arsenic workers	Arsenic
Arsine workers	Arsenic
Art Glass workers	Benzene
Artificial flower makers	Arsenic
Artificial leather makers	Benzene
Artificial abrasive makers	Carbon monoxide
Artificial gas workers	Carbon monoxide
Asbestos-cement products makers	Asbestos
Asbestos-cement products makers	Asbestos
Asbestos-cement products users	Asbestos
Asbestos-coating makers	Asbestos
Asbestos-coating users	Asbestos
Asbestos-Grout makers	Asbestos
Asbestos-Grout users	Asbestos

OCCUPATION	AGENT(S)
Asbestos-millboard makers	Asbestos
Asbestos-millboard users	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
Asphalt mixers	Benzene
Auto garage workers	Asbestos Silica
Auto painters	Nitrogen dioxide
Automobile repair garage workers	Asbestos
Automobile Users	Carbon monoxide
Babbiters	Lead

OCCUPATION	AGENT(S)
Babbit metal workers	Antimony arsenic
Bakers	Carbon monoxide
Battery makers	Lead
Battery workers (storage)	Antimony
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
Beryllium alloy machiners	Beryllium
Beryllium alloy makers	Beryllium
Beryllium compound makers	Beryllium
Beryllium-copper founders	Beryllium
Beryllium-copper grinders	Beryllium
Beryllium-copper polishers	Beryllium
Beryllium extractors	Beryllium
Beryllium metal machiners	Beryllium
Beryllium mineral miners	Beryllium

OCCUPATION	AGENT(S)
Beryllium phosphor makers	Beryllium
Beryllium workers	Beryllium
Bisque-kiln workers	Carbon monoxide silica
Blacksmiths	Carbon monoxide lead
Blast furnace workers	Carbon monoxide sulphur dioxide
Blast furnace gas users	Carbon monoxide
Bleaching powder makers	Arsenic
Blockers (felt hat)	Carbon monoxide
Blueprints	Nitrogen dioxide
Boiler rooms	Noise
Boiler water treaters	Sulfur dioxide
Boiler operators	Arsenic
Boiler room workers	Carbon monoxide
Bone extractors	Sulfur dioxide
Book binders	Arsenic
Bookbinders	Lead
Bottle cap makers	Lead
Brake lining makers	Asbestos
Brakelining makers	Benzene
Brass founders	Antimony carbon monoxide
Brass polishers	Lead
Brass cleaners	Nitrogen dioxide

OCCUPATION	AGENT(S)
Brass makers	Arsenic
Braziers	Lead Nitrogen dioxide
Brewery workers	Sulfur dioxide
Brewers	Carbon monoxide
Brick burners	Carbon monoxide
Brick makers	Lead
Brick layers	Lead Silica
Brickmakers	Sulfur dioxide
Bright dip workers	Nitrogen dioxide
Britannia metal workers	Antimony
Bronzers	Antimony Arsenic Benzene Lead
Bronze cleaners	Nitrogen dioxide
Bronze makers	Arsenic
Broommakers	Sulfur dioxide
Brush makers	Lead
Buffers	Silica
Buhrstone workers	Silica
Building demolition workers	Asbestos
Burnishers	Antimony Benzene
Busdrivers	Carbon monoxide
Cable makers	Lead
Cable splicers	Antimony Carbon monoxide Lead

OCCUPATION	AGENT(S)
Cadium workers	Arsenic
Can makers	Benzene
Candle (colored) makers	Arsenic
Canners	Arsenic Lead
Carbide makers	Carbon monoxide
Carbolic acid makers	Benzene Sulfur dioxide
Carbon monoxide workers	Carbon monoxide
Carders (cotton mill)	Cotton dust
Carders (asbestos)	Asbestos
Carding machine operatirs (cotton mill)	Cotton dust
Carper makers	Arsenic
Carborundum makers	Silica
Carroters (felt hat)	Arsenic
Cartridge makers	Lead
Cast scrubbers (electroplating)	Benzene
Casting Cleaners (Foundry)	Silica
Cathode ray tube makers	Beryllium
Cattle dip workers	Arsenic
Caulking compound makers	Asbestos
Caulking compound users	Asbestos
Celluloid makers	Nitrogen dioxide
Cellulose makers	Sulfur dioxide
Cement makers	Carbon monoxide Silica
Cement mixers	Silica
Ceramic makers	Antimony Arsenic Beryllium Lead
Ceramic workers	Silica
Ceramic enamel workers	Arsenic
Charcoal burners	Carbon monoxide
Chauffers	Carbon monoxide
Chemical equipment makers	Lead
Chemical products manufacture	Noise
Chemical glass makers	Silica
Chimney masons	Carbon monoxide
Chimney sweepers	Carbon monoxide
Chippers	Lead Silica
Chlorinated paraffin makers	Lead
Chlorobenzene makers	Benzene
Chlorodiphenyl makers	Benzene
Chrome platers	Beryllium Chromic acid
Chromic acid makers	Chromic acid
Cigar makers	Lead

OCCUPATION	AGENT(S)
Cleaner operators (Cotton mill)	Cotton dust
Cleaners (Cotton mill)	Cotton dust
Clutch facing makers	Asbestos
Clutch Disc impregnators	Benzene
Coal miners	Silica
Coal tar refiners	Benzene
Coal tar workers	Benzene
Coal distillers	Carbon monoxide
Cobblers (Asbestos)	Asbestos
Cobblers	Benzene
Coke oven door cleaners-luterman	Coke oven emissions
Coke oven door machine operators	Coke oven emissions
Coke oven heater	Coke oven emissions
Coke oven larry car operators	Coke oven emissions
Coke oven lidmen-larrymen	Coke oven emissions
Coke oven maintenance men	Coke oven emissions
Coke oven patcher	Coke oven emissions
Coke oven pusher operators	Coke oven emissions
Coke oven quench car operators	Coke oven emissions
Coke oven tar chaser	Coke oven emissions
Coke oven workers	Benzene Carbon monoxide Sulfur dioxide
Colored glass maker	Chromic acid
Combing machine operators (cotton mill)	Cotton dust
Compositors	Antimony
Compressed air workers	Carbon monoxide
Computer parts makers	Beryllium
Construction	Noise
Construct workers	Asbestos Silica
Copper cleaners	Nitrogen dioxide
Copper Smelters	Arsenic Sulfur dioxide
Copper refiners	Antimony
Copper strippers	Chromic acid
Corrosion inhibitor workers	Chromic acid
Corrugated paper manufacture	Noise
Cosmetics makers	Silica
Cotton bleachers	Nitrogen dioxide
Crop dusters	Arsenic Lead
Crushers (asbestos)	Asbestos
Cupola workers	Carbon monoxide

OCCUPATION	AGENT(S)
Cutlery makers	Lead Silica
Cyclohexane makers	Benzene
DDT najers	Benzene
Decorators (Pottery)	Lead
Defoliant applicators	Arsenic
Defoliant makers	Arsenic
Degreasers	Benzene
Demolition workers	Lead
Demolition	Noise
Dental Technicians	Lead
Dental workers	Nitrogen dioxide
Detergent makers	Benzene
Diamond Polishers	Lead
Diatomaceous earth calciners	Silica
Dichlorobenzene makers	Benzene
Diesel Equipment Operators	Nitrogen dioxide
Diesel Engine Operators	Carbon monoxide Sulfur dioxide
Diesel Engine Repairmen	Sulfur dioxide

OCCUPATION	AGENT(S)
Dimethysulfate makers	Arsenic
Diphenyl makers	Benzene
Dippers (acid)	Chromic acid
Disinfectant makers	Arsenic Benzene Sulfur dioxide
Disinfectors	Sulfur dioxide
Divers	Carbon monoxide
Dock workers	Carbon monoxide
Drawing frame operators (cotton mill)	Cotton dust
Drier workers	Carbon monoxide
Drug makers	Arsenic Benzene
Dry cleaners	Benzene
Dryer operators (cotton mill)	Cotton dust
Dye makers	Antimony Arsenic Benzene Lead Nitrogen dioxide Sulfur dioxide
Dyers	Lead
Earth moving equipment operators	Noise
Electronic device makers	Lead
Electroplaters	Antimony Arsenic Benzene Chromic acid Lead Nitrogen dioxide

OCCUPATION	AGENT(S)
Electrotypers	Lead
Electric arc welders	Nitrogen dioxide
Electrical equipment manufacture	Noise
Electronic equipment makers	Silica
Electrolytic copper workers	Arsenic
Electric equipment makers	Beryllium
Embroidery workers	Lead
Emery Wheel makers	Lead
Enamel burners	Lead
Enamelers	Arsenic Benzene Carbon monoxide Lead
Enamel makers	Arsenic Lead
Enamellers	Silica
Engine rooms	Noise
Engravers	Benzene
Etchers	Arsenic Nitrogen dioxide
Ethylbenzene makers	Benzene
Explosives makers	Antimony Lead Nitrogen dioxide

OCCUPATION	AGENT(S)
Explosive users	Nitrogen dioxide
Explosive makers	Benzene
Exterminators	Arsenic Sulfur dioxide
Fabricated metal product manufacture	Noise
Farm equipment operators	Noise
Farmers	Arsenic Lead
Feather workers	Arsenic Benzene Sulfur dioxide
Ferrosilicon workers	Arsenic
Fertilizer makers	Arsenic Nitrogen dioxide Silica Sulfur dioxide
Fettlers	Silica
Fiberizers (Asbestos)	Asbestos
File Cutters	Lead
Firemen	Asbestos Lead
Fireman	Carbon monoxide
Fireproofers	Asbestos
Fireworks makers	Antimony Arsenic
Fischer-Tropsch Process workers	Carbon monoxide
Flameproofers	Antimony
Flint workers	Silica

OCCUPATION	AGENT(S)
Flour bleachers	Nitrogen dioxide Sulfur dioxide
Flower makers (artificial)	Lead
Flue cleaners	Sulfur dioxide
Fluorescent screen makers	Beryllium
Flypaper	Arsenic
Food processing	Noise
Food bleachers	Sulfur dioxide
Formaldehyde makers	Carbon monoxide
Foundry workers	Antimony Carbon monoxide Lead Silica Sulfur dioxide
Foundries	Noise
Foundry workers (core making)	Tol. Diisocyanate
Fruit bleachers	Sulfur dioxide
Fruit preserves	Sulfur dioxide
Fumigant makers	Benzene Sulfur dioxide
Fumigators	Sulfur dioxide
Fungicide makers	Benzene

OCCUPATION	AGENT(S)
Fumigators	Sulfur dioxide
Fungicide makers	Benzene
Furnace liners	Silica
Furnace operators	Sulfur dioxide
Furnace filter makers	Asbestos
Furnace starters	Carbon monoxide
Furnace workers	Carbon monoxide
Furniture manufacture	Noise
Furniture finishers	Benzene
Fused quartz workers	Silica
Galvanizers	Arsenic Lead Sulfur dioxide
Garage mechanics	Carbon monoxide Lead
Gas mantle makers	Beryllium
Gas shrinking operators	Nitrogen dioxide
Gas Station attendants	Carbon monoxide
Gas workers (Illumination)	Benzene Carbon monoxide
Gasket makers	Asbestos
Gasoline engine testers	Carbon monoxide
Gelatin bleachers	Sulfur dioxide
Gin stand operators (cotton mill	Cotton dust

OCCUPATION	AGENT(S)
Ginners	Cotton dust
Glass makers	Antimony Arsenic Beryllium Lead Silica Sulfur dioxide
Glass polishers	Lead
Glass manufacture	Noise
Glaze Mixers (Pottery)	Silica
Glaze dippers (Pottery)	Antimony
Glost-kiln workers	Lead
Glue bleachers	Sulfur dioxide
Glue makers	Benzene
Gold refiners	Antimony Lead
Gold extractors	Arsenic
Gold refiners	Arsenic
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
Gyroscope makers	Beryllium

OCCUPATION	AGENT(S)
Hair remover makers	Arsenic
Hairdressers	Benzene
Handpickers (cotton)	Cotton dust
Hard rock miners	Silica
Heat treaters	Nitrogen dioxide
Heat treaters (magnesium	Sulfur dioxide
Heat resistant clothing makers	Asbestos
Heat treaters	Carbon monoxide
Herbicide makers	Arsenic Benzene
Hide preservers	Arsenic
Histology technicians	Benzene
Hydrochloric acid workers	Benzene
Ice makers	Arsenic
Illuminating gas workers	Arsenic
Incandescent lamp makers	Lead
Inert filter media workers	Asbestos
Ink makers	Arsenic Benzene Chromic Acid Lead
Insecticide makers	Antimony Arsenic Benzene Lead Silica Sulfur dioxide

OCCUPATION	AGENT(S)
Insecticide users	Lead
Insulators	Silica
Insulation workers	Asbestos Tol. Diisocyanate
Insulators (Wire)	Antimony
Internal guidance system makers	Beryllium
Iron workers	Carbon monoxide
Ironing board cover makers	Asbestos
Isocyanate resin workers	Tol. diisocyanate
Japan makers	Lead
Japanners	Lead
Japan makers	Arsenic
Japanners	Arsenic
Jet fuel makers	Nitrogen dioxide
Jewelers	Arsenic Lead Silica
Junk metal refiners	Lead
Jute workers	Silica
Kiln liners	Silica
Kraft recovery furnace workers	Carbon monoxide
Labelers (paint can)	Lead
Laboratory hood installers	Asbestos

OCCUPATION	AGENT(S)
Lacquer makers	Benzene Lead Nitrogen dioxide
Lacquer workers	Tol. diisocyanate
Laggers	Asbestos
Lake color makers	Antimony
Laundry workers	Carbon monoxide
Lead burners	Lead
Lead counterweight makers	Lead
Lead flooring makers	Lead
Lead foil makers	Lead
Lead mill workers	Lead
Lead miners	Lead
Lead pipe makers	Lead
Lead salt makers	Lead
Lead shield makers	Lead
Lead smelters	Lead
Lead stearate makers	Lead
Lead workers	Lead
Lead smelters	Sulfur Dioxide
Lead burners	Antimony
Lead hardeners	Antimony
Lead shot workers	Antimony
Lead burners	Arsenic
Lead shot makers	Arsenic

OCCUPATION	AGENT(S)
Lead smelters	Arsenic
Leather mordants	Antimony
Leather workers	Arsenic
Leather makers	Benzene
Lift truck operators (propane and gasoline fueled)	Carbon monoxide
Lime burners	Arsenic
Lime kiln workers	Carbon monoxide
Linoleum makers	Benzene Lead
Linotypers	Antimony Lead
Linseed oil boilers	Lead
Lint cleaner operators (cotton mill)	Cotton dust
Lithographers	Lead
Lithotransfer workers	Lead
Lithographers	Benzene Silica
Lumbering	Noise
Magnesium foundry workers	Sulfur dioxide
Maleic acid makers	Benzene
Masons	Silica
Match makers	Antimony Lead
Meat preservers	Sulfur dioxide
Medical technicians	Nitrogen dioxide

OCCUPATION	AGENT(S)
Mercury smelters	Carbon monoxide Sulfur dioxide
Metal burners	Lead
Metal cutters	Lead
Metal grinders	Lead
Metal miners	Lead
Metal polishers	Lead
Metal refiners	Lead
Metallizers	Lead
Metal forming	Noise
Metal machining	Noise
Metal working	Noise
Metal buffers	Silica
Metal burnishers	Silica
Metal polishers	Silica
Metal refiners	Arsenic Sulfur dioxide
Metal bronzers	Antimony
Metal cleaners	Arsenic
Metallurgists	Beryllium
Metal oxide reducers	Carbon monoxide
Metal refiners	Carbon monoxide
Methanol makers	Carbon monoxide
Microscopical preparation workers	Chromic acid
Millinery workers	Benzene
Mine workers	Nitrogen dioxide

OCCUPATION	AGENT(S)
Mine-tunnel coaters	Tol. diisocyanate
Miners	Antimony Arsenic Carbon monoxide Silica
Mining open pit	Noise
Mining underground	Noise
Mirror silverers	Benzene Lead
Missile technicians	Beryllium
Mold makers (plastic)	Beryllium
Mond process workers	Carbon monoxide
Monotypers	Antimony Carbon monoxide
Mordanters	Antimony Arsenic Benzene
Mortar makers	Silica
Motorman	Silica
Musical instrument makers	Lead
Neon sign workers	Beryllium
Neon tube makers	Beryllium
Nickel refiners	carbon monoxide
Nickel smelters	Carbon monoxide
Nitrate workers	Nitrogen dioxide

OCCUPATION	AGENT(S)
Nitric acid workers	Lead Nitrogen dioxide
Nitroglycerin makers	Lead
Nitrogen dioxide workers	Nitrogen dioxide
Nitrocellulose makers	Arsenic
Nitrobenzene makers	Benzene
Nitrocellulose makers	Arsenic
Nitrobenzene makers	Benzene
Nitrocellulose workers	Benzene
Nonsparking tool makers	Beryllium
Nuclear physicists	Beryllium
Nuclear reactor workers	Beryllium
Nurses	Nitrogen dioxide
Nylon & makers	Tol. diisocyanate
Oil bleachers	Sulfur dioxide
Oil purifiers	Silica
Oil processors	Benzene Sulfur dioxide
Oil purifiers	Chromic acid
Oilcloth makers	Benzene
Oilstone workers	Silica
Openers (cotton mill)	Cotton dust

OCCUPATION	AGENT(S)
Optical equipment makers	Silica
Ordnance manufacturing	Noise
Ore smelting workers	Sulfur dioxide
Ore smelters	Arsenic
Organic chemical synthesizers	Antimony Arsenic Benzene Carbon monoxide Chromic acid Nitrogen dioxide Tol. diisocyanate
Organic sulfonate makers	Sulfur dioxide
Oxalic acid makers	Carbon monoxide
Oxidized cellulose compound makers	Nitrogen dioxide
Painters	Lead
Paint makers	Antimony Lead
Paint pigment makers	Lead
Paint mixers	Silica
Painters	Antimony Arsenic
Paint makers	Arsenic Asbestos
Painters	Benzene
Paint makers	Benzene
Paper hangers	Arsenic Lead

OCCUPATION	AGENT(S)
Paper manufacture	Noise
Paper products manufacture	Noise
Paper makers	Arsenic Sulfur dioxide
Paraffin processors	Benzene
Patent leather makers	Carbon monoxide Lead
Pearl makers (imitation)	Lead
Pencil makers	Benzene
Perfume makers	Antimony Benzene
Petroleum refinery workers	Benzene
Petroleum refining	Noise
Petroleum refinery workers	Arsenic Sulfur dioxide
Petrochemical workers	Benzene
Pewter workers	Antimony
Pharmaceutical makers	Lead
Pharmaceutical workers	Antimony Arsenic Benzene
Phenol makers	Benzene
Phosphor makers	Antimony
Phosphate coating strippers	Chromic acid

OCCUPATION	AGENT(S)
Photography workers	Lead
Photoengravers	Nitrogen dioxide
Photographic Chemical makers	Benzene
Photography workers	Chromic acid
Physicians	Nitrogen dioxide
Pickers (cotton mill)	Cotton dust
Picklers	Chromic acid Nitrogen dioxide
Picric acid makers	Benzene
Pigment makers	Antimony Arsenic
Pipe fitters	Lead Nitrogen dioxide
Pipe insulators	Asbestos
Plasma torch operators	Nitrogen dioxide
Plastic workers	Lead
Plastics manufacture	Noise
Plastic products manufacture	Noise
Plastic foam makers	Tol. diisocyanate
Plasticizer workers	Tol. diisocyanate
Plaster cast bronzers	Antimony
Plastic workers	Arsenic

OCCUPATION	AGENT(S)
Plastic makers	Abestos
Plastics workers	Beryllium
Plumbers	Arsenic Lead
Policemen	Lead
Police	Carbon monoxide
Polishing soap makers	Silica
Polish makers	Benzene
Polyurethane foam makers	Tol. diisocyanate
Polyurethane foam users	Tol. diisocyanate
Polyurethane sprayers	Tol. diisocyanate
Porcelain workers	Antimony Silica
Pottery glaze mixers	Lead
Pottery glaze dippers	Lead
Pottery workers	Antimony Lead Silica Sulfur dioxide
Pottery decorators	Benzene
Pottery kiln workers	Carbon monoxide
Pouncers (felt hat)	Silica
Power plant operators	Noise

OCCUPATION	AGENT(S)
Precision instrument makers	Beryllium
Preservative makers	Sulfur dioxide
Press box operators (cotton mill)	Cotton dust
Primary metal processing	Noise
Printers	Lead
Printing	Noise
Printers	Antimony
Printing ink workers	Arsenic
Printers	Benzene
Producer gas workers	Carbon monoxide
Protein makers (industrial)	Sulfur dioxide
Protein makers (food)	Sulfur dioxide
Pulpstone workers	Silica
Pump packing makers	Asbestos
Putty makers	Benzene Lead
Pyrites burners	Sulfur dioxide
Pyrotechnics workers	Antimony Arsenic
Pyroxylin-plastics workers	Lead

OCCUPATION	AGENT(S)
Quarrying	Noise
Quarry workers	Silica
Quartz workers	Silica
Raw silk bleachers	Nitrogen dioxide
Rayon makers	Arsenic
Reclaimers (rubber)	Benzene
Refractory Makers	Chromic acid Silica
Refractory material Makers	Beryllium
Refrigeration workers	Sulfur Dioxide
Resin 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	Asbestos Lead
Roofing materials Makers	Asbestos
Rotogravure printers	Benzene

OCCUPATION	AGENT(S)
Roving Frame Operators (cotton mill)	Cotton dust
Rubber Buffers	Lead
Rubber makers	Antimony Benzene Lead
Rubber Reclaimers	Lead
Rubber manu-facture	Noise
Rubber products manufacture	Noise
Rubber compound mixers	Silica
Rubber compounders	Asbestos
Rubber cementers	Benzene
Rubber gasket makers	Benzene
Sand cutters	Silica
Sand pulver-izers	Silica
Sandblasters	Silica
Sandpaper makers	Silica
Sandstone grinders	Silica
Sanitation workers	Carbon monoxide
Sawyers	Silica
Scouring soap workers	Silica
Scrap metal workers	Lead
Sealing wax makers	Arsenic

OCCUPATION	AGENT(S)
Semiconductor workers	Antimony Lead
Semiconductor com-pound makers	Arsenic
Service station attendants	Lead
Sewer workers	Carbon monoxide
Sheep dip workers	Arsenic
Sheet metal workers	Lead
Shellac makers	Benzene Lead
Shingle makers	Asbestos
Ship dismantlers	Lead
Ship burners	Tol. diisocyanate
Ship welders	Tol. diisocyanate
Ship builders	Asbestos
Ship demolition workers	Asbestos
Shipbuilding	Noise
Shoe stainers	Lead
Shoe factory workers	Benzene
Shoe finishers	Benzene
Shot makers	Lead
Silica brick workers	Silica
Silicon alloy makers	Silica
Silk weighters	Lead
Silo Fillers	Nitrogen dioxide
Silver polishers	Silica

OCCUPATION	AGENT(S)
Silver Refiners	Arsenic
Silver Platers	Beryllium
Slashing Operators (cotton mill)	Cotton dust
Slate workers	Silica
Slushers (porcelain enameling)	Lead
Smelters	Silica
Soap makers	Benzene
Soda makers	Arsenic
Sodium silicate makers	Silica
Sodium sulfite makers	Sulfur dioxide
Soil Sterilizer makers	Arsenic
Solderers	Arsenic Carbon monoxide Lead
Solder makers	Antimony Lead
Solid Rocket Fuel Makers	Beryllium
Solvent makers	Benzene
Spacecraft workers	Silica
Spindle pickers (cotton)	Cotton dust
Spinners (cotton mill)	Cotton dust
Spinners (asbestos)	Asbestos
Spooling operators (cotton mill)	Cotton dust

OCCUPATION	AGENT(S)
Spray painters	Tol. diisocyanate
Stainers	Benzene
Stain makers	Benzene
Steel engravers	Lead
Steel making	Noise
Steel makers	Carbon monoxide
Stereotypers	Antimony Lead
Stokers	Carbon monoxide
Stone products industries (cement mills)	Noise
Stone workers	Noise
Stone bedrubbers	Silica
Stone cutters	Silica
Stone planers	Silica
Storage battery chargers	Sulfur dioxide
Storage batter workers	Antimony
Straw bleachers	Sulfur dioxide
Street sweepers	Silica
Stripper operators (cotton)	Cotton dust
Stripper operators (cotton mill)	Cotton dust
Strippers	Chromic acid
Styrene makers	Benzene
Submarine workers	Arsenic

OCCUPATION	AGENT(S)
Subway construction workers	Silica
Sugar refiners	Sulfur dioxide
Sulfite makers	Sulfur dioxide
Sulfuric acid makers	Nitrogen dioxide Sulfur dioxide
Sulfur dioxide workers	Sulfur dioxide
Sulfurers (malt and hops)	Sulfur dioxide
Sulfuric acid workers	Arsenic
Synthetic fiber makers	Benzene
Talc miners	Asbestos
Talc workers	Asbestos
Tannery workers	Lead Sulfur dioxide
Tanners	Arsenic Chromic acid
Tar workers	Arsenic
Taxidermists	Arsenic
TDI workers	Tol. diisocyanate
Television picture tube makers	Lead
Temperers	Lead
Textile makers	Lead
Textile manufacture	Noise
Textile bleachers	Sulfur dioxide
Textile processors	Tol. diisocyanate

OCCUPATION	AGENT(S)
Textile dryers	Antimony
Textile flame-proofers	Antimony Asbestos
Textile printers	Antimony Arsenic
Textile workers	Asbestos
Textile mordants	Chromic acid
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
Toll collectors (highway)	Carbon monoxide
Tooth paste makers	Silica
Traffic controllers	Carbon monoxide
Transportation equipment operators	Noise
Tree Sprayers	Arsenic
Trinitrotoluol makers	Benzene
Trucking	Noise
Tube mill liners	Silica
Tumbling barrel workers	Silica

OCCUPATION	AGENT(S)
Tunnel workers	Carbon monoxide Nitrogen dioxide
Tunneling	Noise
Tunnel construction workers	Silica
Tunnel attendants	Carbon monoxide
Twisters (cotton mill)	Cotton dust
Type founders	Lead
Type setters	Lead
Type metal workers	Antimony Arsenic
Type cleaners	Benzene
Typesetters	Antimony
Undercoaters	Asbestos
Upholstery makers	Tol. diisocyanate
Vanadium compound makers	Lead
Varnish makers	Arsenic Benzene Lead
Vegetable preservers	Sulfur dioxide
Vehicle tunnel attendants	Lead
Vinyl-asbestos tile makers	Asbestos
Vinyl-asbestos tile installers	Asbestos
Vulcanizers	Antimony Benzene Sulfur dioxide
Wallpaper-printers	Arsenic Lead

OCCUPATION	AGENT(S)
War gas makers	Benzene
Warehouse workers	Carbon monoxide
Warfare gas makers	Arsenic
Water weed cantrollers	Arsenic
Water gas workers	Carbon monoxide
Wax makers	Benzene
Weavers (cotton mill)	Cotton dust
Weavers (asbestos)	Asbestos
Weed sprayers	Arsenic
Welders	Benzene Carbon monoxide Lead Nitrogen dioxide
Whetstone workers	Silica
Wicker ware bleachers	Sulfur dioxide
Window shade makers	Benzene
Wine makers	Sulfur dioxide
Wire coating workers	Tol. diisocyanate
Wire drawers	Arsenic
Wire insulators	Benzene
Wood stainers	Lead
Wood products manufacture	Noise
Wood filler workers	Silica
Wood bleachers	Sulfur dioxide
Wood pulp bleachers	Sulfur dioxide
Wood preservative makers	Arsenic

OCCUPATION	AGENT(S)	OCCUPATION	AGENT(S)
Wood preservers	Arsenic		
Wood distillers	Carbon monoxide		
X-ray tube makers	Beryllium		
Zinc mill workers	Lead		
Zinc smelter chargers	Lead		
Zinc Smelters	Sulfur dioxide		
Zinc refiners	Antimony		
Zinc chloride makers	Arsenic		
Zinc miners	Arsenic		
Zinc refiners	Arsenic		
Zinc white makers	Carbon monoxide		

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 phlegm with the first smoke or on first going out of doors. Exclude phlegm 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	
	NOW	BEFORE STOPPING
Cigarettes/day (Average including weekends)	___	________
Oz. tobacco/week (handrolled)	___	________
Cigars/week (large)	___	________
Cigars/week (small)	___	________

<u>OCCUPATION</u> (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 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 Toulene Diisocyanate, Health Advisory Committee, British Rubber Manufacturers' Association Ltd.

D. CASE HISTORIES

This section contains two detailed examples of investigation of occupationl 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 nonoccupational 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	

Epidemiological 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	CHROMIUM
OSHA ALLOWABLE LIMITS			0.5	1	0.2	0.5
Operator's Breathing Zone:						
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 ad 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 to 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 prominant, 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, inquinal rings firm. No clubbing of the fingers. Small varicosities on left lower leg. No ankle edema. Axillary and inquinal 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

Epidemiological Data

There is no evidence in the scientific literature to indicate that working in this environment is hazardous. Others in the work area also have occasional coughs--some with clear sputum production. These men all have negative clinical and laboratory findings. Epidemiological 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
Operator's Breathing Zone:			
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
Operator's Breathing Zone:			
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
Operator's Breathing Zone:			
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	10
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 epidemiological or toxicological evidence and the ambient work environment well below the recommended levels, this case must be considered non-occupational 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.

E. Glossary of Terms

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 hearing or vision.

Acute. Severe, usually crucial, often dangerous; in which relatively rapid changes are occurring. An acute exposure runs a comparatively short course.

Adhesion. A holding together by new tissue, 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.

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

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

Angina. Spasmodic, choking or suffocative pain.

Asphyxia. Suffociation from lack of oxygen.

Asthenia. Lack or loss of strength; debility.

Audiogram. A test and recording of hearing ability.

Audiometer. An instrument for measuring hearing ability.

Benign. Harmless.

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

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

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

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

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

Bone 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 part of the blood forming system.

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

Bronchiogenic. Pertaining to the bronchi.

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

Bronchitis. Inflammation of the bronchial tubes.

Bronchoscopy. Examination of the bronchi through a bronchoscope, an instrument used for visual examination of the interior of a bronchus.

Bronchospasm. Spasm of the bronchi or bronchioles.

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

Carcinogen. Substance which is capable of causing cancer.

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

Chemical Cartridge. The type of absorption unit used with a respirator for removal of low concentrations of solvent vapors and certain gases.

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

Chronic. Persistent, prolonged, repeated.

Chronic Obstructive Lung Disease. Interference with normal breathing.

Coalescence. Fusion of two or more parts.

Colorimetry. An analytical technique based on measuring color.

Coma. Prolonged unconsciousness.

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.

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.

Conjunctivitis. Inflammation of the mucous 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 atmosphere.

Cor Pulmonale. Hypertrophy (enlargement) or failure of the right side of the heart.

Cornea. The transparent part of the eye.

CPS. Cycles per second (Hertz); a measurement of frequency of sound.

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

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

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

Decibel (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 patient 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.

Edema. A swelling of body tissues.

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

Epithelioma. Carcinoma of the epithelial cells of the skin.

Erythema. Reddening of the skin.

Erythrocyte. The mature red blood corpuscle.

Etiology. The study of the causes of disease.

FEV 1. Forced expiratory volume in one second; a test of pulmonary function.

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

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

Hematrocrit. 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.

Hemoptysis. Spitting blood or blood-stained sputum.

Hemorrhage. Profuse Bleeding.

Hepatic Injury. Damage to the liver.

Hepatitis. Infection of the liver.

Hertz. Unit of frequency of sound.

Hyperemia. Congestion from an unusual amount of blood.

Hyperpigmentation. Increased coloration of the skin.

Hyperplastic. Excessive proliferation of cells.

Hypoplastic. Reduced or defective production of cells.

Inflammation. The reaction of body tissue to injury.

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

Inorganic. Term used to designate compounds that generally do not contain carbon.

Interstitial. Pertaining to the small spaces between cells.

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 over-production of white blood cells.

Leukocyte. A white blood cell.

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

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

Malignancy. A neoplasm or tumor that is cancerous.

Malignant. Virulent, harmful.

Mean Corpuscular Volume. A measurement of red blood cells expressed as the average volume.

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

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

Mists. Liquid droplets suspended in air.

Myalgia. Tenderness or pain in the muscles.

Narcotic. Producing stupor or sleep.

Neoplasm. Malignant (cancerous) tumor.

Nephritis. Inflammation in the kidneys.

NIOSH. National Institute for Occupational Safety and Health.

Node. A small round or oval mass of tissue.

Nodule. A small node.

Nuisance Dust. An innocuous dust.

Opacities. Opaque areas or spots.

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. Rapid heart beat of which a person is acutely aware.

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

Pathologicall Abnormal or diseased.

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

Pneumonitis. Inflammation of the lungs.

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.

Presbycusis. Hearing loss due to age.

Prognosis. Prediction of future course of a disease.

Purpura. Hemmorhage into the skin or mucous membrane.

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.

Serum. The clear fluid that separates from the blood when the blood clots.

Skin Absorption. Penetration of the unbroken skin by a material.

Subcutaneous. Beneath or to be introduced beneath the skin.

Substernal. Beneath the breastbone.

Syncope. Fainting.

Synergism. Cooperative action of substances, producing a total effect greater than the sum of their separate effects.

Systemic. Spread through the body.

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

Tinnitis. A ringing sound in the ears.

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

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

Trachea. The windpipe.

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

Trauma. An injury or a wound.

Tumor. A swelling or growth of useless cells.

VC (Vital Capacity). A test of lung function.

Vertigo. Dizziness.

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