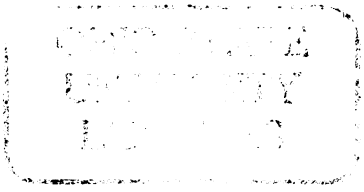
Occupational Diseases

A Guide To Their Recognition

U. S. DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
Public Health Service



OCCUPATIONAL DISEASES

A Guide

To Their Recognition

Rewritten and Enlarged Edition of

Occupation Hazards and Diagnostic Signs

W. M. GAFAFER, D. Sc., Editor

Division of Occupational Health

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE

Public Health Service

PUBLIC HEALTH SERVICE PUBLICATION NO. 1097

Reprinted June 1966

U.S. Government Printing Office, Washington : 1966

For sale by the Superintendent of Documents, U.S. Government Printing Office
Washington, D.C., 20402 - Price $1.25 (paper cover)

• foreword

The tremendous technologic achievements of the past few decades have greatly intensified the problem of occupational disease detection and control. Indeed, few, if any, of the vast array of new products and processes created by a dynamic technology are without their potential health hazards. As part of the effort to produce new tools to facilitate the discovery and prevention of job-related illness, this guide is offered to those responsible for meeting the growing challenges of occupational health.

Harry Heimann

HARRY HEIMANN, M.D.
Chief, Division of Occupational Health

• preface

In 1918 the Metropolitan Life Insurance Company issued a small pamphlet entitled *Occupation Hazards and Diagnostic Signs*. The pamphlet was prepared as a guide for medical examiners and pointed out impairments to be looked for in hazardous occupations. The company revised the pamphlet in 1921, and subsequently the U.S. Department of Labor assumed the responsibility of publishing further revisions prepared by the company. The pamphlet was last revised in 1942 and the final reprinting published in 1951.

Because of the popularity of the pamphlet, the U.S. Department of Labor recognized the need for further revision when the supply of the final reprinting became exhausted. This need, as well as the medical nature of the work, prompted the Department to request the Division of Occupational Health, Public Health Service, to undertake the preparation of a revision. This has been done.

A comparison of the present work with its progenitor will reveal a number of changes in material and method of presentation suggested by the passage of almost a half century. These changes, in turn, suggested the selection of a new title which it is believed reflects more adequately the nature of the contents of the book. It is hoped that the current edition will be found even more helpful to physicians and members of allied professions engaged in efforts to prevent and control the potential diseases of the occupational environment, thus leading to the fulfillment of the primary objective of optimal health for the working population.

• contributors

(*Except when otherwise indicated contributors are associated with the Occupational Health Research and Training Facility, Division of Occupational Health, Public Health Service, Cincinnati, Ohio*)

DONALD J. BIRMINGHAM, M.D., *Chief*, Dermatology Section

ALEXANDER COHEN, PH. D., Research Psychologist, Physiology Section

ROY L. GIBSON, M.D., Occupational Medicine Section. Present Address: Regional Medical Director, Gulf Oil Corporation, Pittsburgh, Pa.

DUNCAN A. HOLADAY, M.A., Chief, Occupational Health Field Station, Division of Occupational Health, Salt Lake City, Utah

MARCUS M. KEY, M.D., Assistant Chief, Dermatology Section

THOMAS H. MILBY, M.D., Occupational Medicine Section. Present Address: California Department of Public Health, Berkeley

HERBERT E. STOKINGER, PH. D., Chief, Toxicology Section

• contents

SECTION VI

SECTION VII

SECTION VIII

SECTION IX

SECTION X

SECTION XI

SECTION XII

SECTION XIII

• section I

INTRODUCTION

That occupation is recognized as an important factor in determining the cause of disability and even death is reflected in the continued revision of workmen's compensation laws to include increasing numbers of occupational diseases.

Numerous occupations involve exposures to chemical, physical, and biologic agents and there is little question that the dynamic growth of the environmental spectrum is increasing the complexity of this situation. This dynamic growth involves essentially the introduction into the environment of new processes and materials which augment or replace those previously in use. Furthermore, the size of the occupational hazards problem is increased by the fact that only a small percentage of the American working population has access to in-plant industrial and hygienic services.

Occupational History in Diagnosis

Physicians are regularly consulted by patients with signs and symptoms of definite as well as indefinite character. In some instances, presenting complaints may stem from the occupation. When a physician's knowledge and interest lead him to suspect the occupation as a possible causative factor, many obscure cases can be diagnosed which previously had puzzled competent clinicians.

In perplexing cases which appear to defy diagnosis, the clinician must exert great care in determining whether any of the usual diagnostic signs known to be inherent in an occupational disease are in evidence in his patient. Of no less importance are the physical signs and symptoms of vague character which could easily be passed over unless the occupational background of the patient has been explored.

There are also situations where exposures to certain chemicals and other environmental hazards are only occasionally experienced by the worker. In such instances, there is not only an increase in risk because the danger is not suspected but also an increase in the difficulty of arriving at an acceptable diagnosis of the illness.

The examiner must, therefore, be wholly mindful not only of the present occupation, but of former ones as well, since a patient suffering from certain ailments may no longer be engaged in the occupation responsible for his present condition.

By continued vigilance regarding the occupational history and the hazards encountered, the medical profession can use the occupational findings more effectively in forming judgments concerning disabilities as well as in the diagnosis and treatment of disease. In this way, the physician may not only add to the knowledge of occupational diseases and disabilities but also to the understanding of the possible part played by work factors in the development or aggravation of the diseases and disabilities not usually associated with the work environment. Thus, heart disease is becoming prominent among the claims submitted to workmen's compensation agencies. In such instances, the difficult problem of causal relation and disability evaluation confronts the physician.

Nonoccupational History

It must be pointed out that in evaluating signs and symptoms it is essential that the physician consider also the possible part played by the nonoccupational environment. Thus, the patient may have taken a medicament, in some instances momentarily forgotten, which might account for the illness. On the other hand, the patient may have chosen a hobby which he engages in after work hours in the home or the garden and involves the handling of an injurious agent which might be the offending one.

Moreover, the physician in his study of the nonoccupational environment of his patient may find a factor possibly synergistic, or potentiating, in its effect on the hazards presented by the occupation of his patient. Questions concerning the nonoccupational environment should be routinely raised; in some cases, the information elicited will be helpful in establishing a diagnosis.

Bases for a Diagnosis

In any case, regardless of whether the environment concerned is occupational or nonoccupational the diagnosis must be based on (1) a meticulously taken history, (2) knowledge of the nature and severity of the exposure, (3) signs and symptoms furnishing corroborative evidence as to its accuracy, and (4) supporting clinical and analytical laboratory tests indicating the extent of the exposure.

Purpose and Origin

This handbook has been prepared to assist physicians in general practice, consultants, industrial hygienists, and allied professional personnel who come into close contact with those engaged in industrial and agricultural pursuits.

Industry may find the material useful in developing its preventive health work; labor may discover much of value in its efforts to promote healthful

work environments; and those responsible for the administration of workmen's compensation should gain information helpful in the settling of claims.

The book is an outgrowth of the pamphlet, *Occupation Hazards and Diagnostic Signs*—see preface—which for many years has enjoyed great popularity and proved to be a useful reference source, as well as a teaching aid, in clinical medicine in general and occupational diseases in particular.

Contents

The current work entailed the development of three major categories of hazard: chemical, physical, and biologic. Numerous publications, and the files of the Division of Occupational Health, served as reference sources.

The special diagnostic tests suggested under the various chemical hazards are intended as an aid to the reader with the hope that they will stimulate the use of more detailed textbook material dealing with the test or disease in question. Recommended threshold limit values are included if they have been published. It must be kept in mind, however, that these values are only suggested guidelines subject to change as evidenced by their annual publication.

Occupations associated with different environmental agents appear in various sections under the heading *Potential Occupational Exposures.* The word potential is used because it is not to be assumed that the mere presence of an injurious agent will lead to an occupational disease or disability. Much depends upon such factors as severity and duration of exposure, individual susceptibility, and the health protection practices adopted by management. Occupation information was derived principally from material made available by the Bureau of Labor Standards, U. S. Department of Labor; the records of the Division of Occupational Health; and the following books: *The Condensed Chemical Dictionary, Encyclopedia of Chemical Technology* and *The Merck Index of Chemicals and Drugs.* When similar activities were performed in the same or different industries efforts were made to use the same name for the occupation. In general, the term worker includes both maker and user.

Basic material appears in the sections on *Means of Contact of Toxic Agents* and *Mode of Action of Toxic Substances.* Because of their importance in occupational health, separate sections have been prepared on skin irritants and sensitizers, pneumoconioses, and pesticides. Plastics and synthetic resins are also given separately not only because this method facilitates presentation but because of the relatively numerous chemicals and hazards involved in their production. Plant and wood hazards appear separately.

A section is included which lists sources of consultation on matters pertaining to industrial hygiene and occupational health.

A list of general references useful to those interested in occupational health comprises a section. Specific references are subjoined to various sections, subsections and the different chemical hazards. It is hoped that the reader

will find this source material helpful in the further study of particular problems that may come to his attention.

Exclusions

Material on treatment has not been included since such information is readily available elsewhere. The prevention and control of health hazards has been given only minor attention because it was felt that this field was adequately covered in an earlier publication, *Occupational Health Hazards, their Evaluation and Control.* The important area of workmen's compensation laws is not dealt with since material on the subject appears in periodically revised bulletins prepared by the U. S. Department of Labor under the title, *State Workmen's Compensation Laws.*

Much thought was given to the inclusion of a section on mental illness in industry since it is well known that mental disorders can complicate occupational disease, accidents, prolonged disability, work efficiency, and job stability. An example in this category is the syndrome labelled traumatic neurosis, a condition representing a post-traumatic episode which is a sequel of severe physical injury or threat of such injury, or of illness.

Mental illnesses are less easily recognized by the practitioner and allied personnel in the field of occupational health. The causes are generally obscure and the diagnosis and management of the disorder are frequently dependent upon one skilled in psychiatry. Thus, it was not possible to present the subject material in a manner paralleling the other more readily defined entities covered, except in the specific instances where acute or chronic mental disease is part of a pathologic response to an environmental factor. Omitting such a section, therefore, is not because of a lack of importance. Rather, it is felt that the subject merits far more detail than can be given in a work of this type.

Misuse of Information

Since this publication has been prepared primarily as a reference source for professional personnel interested in the prevention, diagnosis, and management of occupational diseases, it is probable that some readers will encounter areas of little interest and will prefer to exercise their prerogative of judicious skipping. Yet, it is felt necessary to conclude with a word of caution lest there be made a gross misuse of the clinical data as presented. Nonprofessional interpretation of the clinical material must never become a substitute for competent medical consultation.

References

Text

DIAMOND, N. M.: State workmen's compensation laws. Bureau of Labor Standards, U.S. Department of Labor, Bull. No. 161. Revised May 1960. U.S. Government Printing Office, Washington, D.C., 1960. Supplement dated December 1961; reviews major state workmen's compensation legislation enacted in United States, 1961.

DUBLIN, L. I. AND VANE, R. J.: Occupation hazards and diagnostic signs, a guide to impairments to be looked for in hazardous occupations. Division of Labor Standards, U.S. Department of Labor, Bull. No. 41. 4th ed. U.S. Government Printing Office, Washington, D.C., 1941. Revised 1942. Reprinted 1951. The first edition was published anonymously in 1918 in New York by the Metropolitan Life Insurance Company as a 15-page, 4- x 6.5-inch, pamphlet with the title, *Occupation hazards and diagnostic signs, a guide for medical examiners regarding impairments to be looked for in hazardous occupations.* A second edition of 30 pages with no change in format was published by the Company in 1921. An enlarged second edition, carrying the title of Bulletin No. 41, and L. I. Dublin and P. Leiboff as authors, appeared in 1922 as Bulletin No. 306 of the U.S. Bureau of Labor Statistics. The third edition by L. I. Dublin and R. J. Vane was a revision of Bulletin No. 306 and issued in 1933 as Bulletin No. 582 of the same Bureau.

GRIFFIN, W. G. AND HOSEY, A. D.: Occupational health hazards, their evaluation and control. Bureau of Labor Standards, U.S. Department of Labor, Bull. No. 198. U.S. Government Printing Office, Washington, D.C., 1958.

KIRK, R. E. AND OTHMER, D. F. (EDITORS): *Encyclopedia of Chemical Technology.* 15 vols. and 2 supplement vols. Interscience Publishers, New York, 1947–1960.

ROSE, A. AND ROSE, E. (EDITORS): *The Condensed Chemical Dictionary.* 6th ed. Reinhold Publishing Corp., New York, 1962.

STECHER, P. G.; FINKEL, M. J.; SIEGMUND, O. H., AND SZAFRANSKI, B. M.: *The Merck Index of Chemicals and Drugs, an Encyclopedia for Chemists, Pharmacists, Physicians, and Members of Allied Professions.* 7th ed. Merck & Co., Rahway, N.J., 1960.

Additional References

COLLINS, R. T.: *A Manual of Neurology and Psychiatry in Occupational Medicine.* Grune & Stratton, New York, 1961.

EDITORIAL: Heart disease and workmen's compensation. *New Eng. J. Med.* 268: 680, 1963. Refers to the work of the American Heart Association's Committee on the Effect of Strain and Trauma on the Heart and Great Vessels. Reference is made to the committee's published recommendation that heart disease be removed from workmen's compensation laws and covered rather by voluntary health and life insurance plans sponsored by industry. This recommendation, it is pointed out, could as well include most other degenerative diseases.

GOLDWATER, L. J.: Occupational exposures and heart disease. *Trauma* 3: 29, (December) 1961.

MEDICAL COMMITTEE: The medical aspects of proof of causal relationship in occupational diseases cases. *In* Workmen's compensation problems. *Proceedings, 45th annual convention, International Association of Industrial Accident Boards and Commissions, 1959.* Bureau of Labor Standards, U.S. Department of Labor, Bull. No. 213. U.S. Government Printing Office, Washington, D.C., 1960.

ROSENBAUM, F. F. AND BELKNAP, E. L. (EDITORS): *Work and the Heart.* Paul B. Hoeber, New York, 1959.

ROSS, W. D.: *Practical Psychiatry for Industrial Physicians.* Charles C. Thomas, Springfield, Ill., 1956.

SAGALL, E. L.: Heart disease, workmen's compensation and the practicing physician. *New Eng. J. Med.* 264: 699, 1961.

• section II

MEANS OF CONTACT AND ENTRY OF TOXIC AGENTS

HERBERT E. STOKINGER, PH. D.

Of the various means of body exposure, skin contact is of first importance in the number of affections occupationally related. Intake by inhalation ranks second, while oral intake is generally of minor importance except as it becomes a part of the intake by inhalation or when an exceptionally toxic agent is involved. For some materials, as might be inferred, there are multiple routes of entry.

Skin Contact

Upon contact of an industrial agent with the skin, four actions are possible: (1) The skin and its associated film of lipid and sweat may act as an effective barrier which the agent cannot disturb, injure or penetrate; (2) the agent may react with the skin surfaces and cause primary irritation; (3) the agent may penetrate the skin, conjugate with tissue protein and effect skin sensitization; and (4) the agent may penetrate the skin through the folliculo-sebaceous route, enter the blood stream and act as a systemic poison.

The skin, however, is normally an effective barrier for protection of underlying body tissues, and relatively few substances are absorbed through this barrier in dangerous amounts. Yet serious and even fatal poisonings can occur from short exposures of skin areas, not necessarily large, to strong concentrations of extremely toxic substances such as parathion and related organic phosphates, tetraethyl lead, aniline and hydrocyanic acid. Moreover, the skin as a means of contact may also be important when an extremely toxic agent penetrates body surfaces from flying objects or through skin lacerations or open wounds.

Inhalation

The respiratory tract is by far the most important means by which injurious substances enter the body. The great majority of occupational poisonings that affect the internal structures of the body result from breathing air-borne substances. These substances lodging in the lungs or other parts of the

respiratory tract may affect this system, or pass from the lungs to other organ systems by way of the blood, lymph, or phagocytic cells. The type and severity of the action of toxic substances depend on the nature of the substance, the amounts absorbed, the rate of absorption, individual susceptibility, and many other factors.

The relatively enormous lung-surface area (90 square meters total surface, 70 square meters alveolar surface), together with the capillary network surface (140 square meters) with its continuous blood flow, presents to toxic substances an extraordinary leaching action that makes for an extremely rapid rate of absorption of many substances from the lungs. Despite this action, there are several industrially important substances that resist solubilization by the blood or phagocytic removal by combining firmly with the components of lung tissue. Such substances include silica, beryllium, thorium, and tolylene diisocyanate. In instances of resistance to solubilization or removal, irritation, inflammation, fibrosis, malignant change, and allergic sensitization may result.

Reference is made in the following material to various air-borne substances and to some of their biologic aspects.

Particulate Matter: Dust, Fume, Mist, and Fog

Dust is composed of solid particulates generated by grinding, crushing, impact, detonation, decrepitation, or other forms of energy resulting in attrition of organic or inorganic materials such as rock, metal, coal, wood, and grain. Dusts do not tend to flocculate except under electrostatic forces; they do not diffuse in air but settle under the influence of gravity. Examples are silica dust and coal dust.

Fume is composed of solid particles generated by condensation from the gaseous state, as from volatilization from molten metals, and often accompanied by oxidation. A fume tends to aggregate and coalesce into chains or clumps. The particle diameter is less than 1 micron. Examples are lead vapor on cooling in the atmosphere; and uranium hexafluoride (UF_6) which sublimes as a vapor, hydrolyzes, and oxidizes to produce a fume of uranium oxyfluoride (UO_2F_2).

Mist is composed of suspended liquid droplets generated by condensation from the gaseous to the liquid state as by atomizing, foaming, or splashing. Examples are oil mists, chromic acid mist, and sprayed paint.

Fog is composed of liquid particles of condensates whose particle size is larger than mists, usually greater than 10 microns. An example is supersaturation of water vapor in air.

Gas and Vapor

A *gas* is a formless fluid occupying completely the space of an enclosure and which can be changed to the liquid or solid state by the combined effect of increased pressure and decreased temperature. Examples are carbon mon-

oxide and hydrogen sulfide. An *aerosol* is a dispersion of a particulate in a gaseous medium while *smoke* is a gaseous product of combustion, rendered visible by the presence of particulate carbonaceous matter.

A *vapor* is the gaseous form of a substance which is normally in the liquid or solid state and which can be transformed to these states either by increasing the pressure or decreasing the temperature. Examples are carbon disulfide, gasoline, naphthalene, and iodine.

Biologic Aspects of Particulate Matter

Size and surface area of particulate matter play an important role in occupational lung disease, especially the pneumoconioses. The particle diameter associated with the most injurious response is believed to be less than 1 micron; larger particles either do not remain suspended in the air sufficiently long to be inhaled or, if inhaled, cannot negotiate the tortuous passages of the upper respiratory tract. Smaller particles, moreover, tend to be more injurious than larger particles for other reasons. Upon inhalation, a larger percentage (possibly as much as 10-fold) of the exposure concentration is deposited in the lungs from small particles than from larger particles. In addition, smaller particles appear to be less readily removed from the lungs. This additional dosage and residence time act to increase the injurious effect of a particle.

The density of the particle also influences the amount of deposition and retention of particulate matter in the lungs upon inhalation. Particles of high density behave as larger particles of smaller density on passage down the respiratory tract by virtue of the fact that their greater mass and consequent inertia tend to impact them on the walls of the upper respiratory tract. Thus, a uranium oxide particle of a density of 11, and 1 micron in diameter will behave in the respiratory tract as a particle of several microns in diameter, and thus its pulmonary deposition will be less than that of a low density particle of the same measured size.

Other factors affecting the toxicity of inhaled particulates are the rate and depth of breathing and the amount of physical activity occurring during breathing. Slow, deep respirations will tend to result in larger amounts of particulates deposited in the lungs. High physical activity will act in the same direction not only because of greater number and depth of respirations but also because of increased circulation rate, which transports the toxic agent at a greater rate to critical tissues, and the presence of increased amounts of certain hormones that act adversely on substances injurious to the lung. Environmental temperature also modifies the toxic response of inhaled materials. High temperatures in general tend to worsen the effect, as do temperatures below normal, but the magnitude of the effect is less for the latter.

218-695 O-66-2

Biologic Aspects of Gases and Vapors

The absorption and retention of inhaled gases and vapors by the body are governed by certain factors different from those that apply to particulates. Here solubility of the gas in the aqueous environment of the respiratory tract governs the depth which a gas will penetrate in the respiratory tract. Thus, very little, if any, of inhaled, highly soluble, ammonia or sulfur dioxide will reach the pulmonary alveoli, depending on concentration, whereas relatively little of insoluble ozone and carbon disulfide will be absorbed in the upper respiratory tract.

Following inhalation of a gas or vapor, the amount that is absorbed into the blood stream depends not only on the nature of the substance but more particularly on the concentration in the inhaled air, and the rate of elimination by the body. For a given gas, a limiting concentration in the blood is attained that is never exceeded no matter how long it is inhaled, providing the concentration of the inhaled gas in the air remains constant. For example, 100 parts per million of carbon monoxide inhaled from the air will reach an equilibrium concentration in the blood corresponding to about 13 percent of carboxyhemoglobin in 4 to 6 hours. No additional amount of breathing carbon monoxide will increase the blood carbon monoxide level. Upon raising the amount of carbon monoxide in the air, however, a new equilibrium level will eventually be reached.

Ingestion

Poisoning by this route in industry is far less common than by inhalation for the reason that the frequency and degree of contact with toxic agents from material on the hands, food, and cigarettes are far less than by inhalation. Because of this, only the most highly toxic substances are of concern by ingestion. Examples are lead, arsenic, and mercury.

The ingestion route, however, passively contributes to the intake of toxic substances by inhalation. That portion of the inhaled material that lodges in the upper respiratory tract is swept up the tract by ciliary action and is subsequently swallowed, thereby contributing to the body intake.

The absorption of a toxic substance from the gastrointestinal tract into the blood is commonly far from complete, despite the fact that, in passing through the stomach, substances are subjected to relatively high acidity and, on passing through the intestine, are subjected to alkaline media.

On the other hand, favoring low absorption are observations such as the following: (1) Food and liquid mixed with the toxic substance not only provide dilution but also reduce absorption because of the formation of insoluble material resulting from the combinatory action of substances

commonly contained in such food and liquid; (2) there is a certain selectivity in absorption through the intestine that tends to prevent absorption of "unnatural" substances or to limit the amount absorbed; and (3) following absorption into the blood stream, the toxic material goes directly to the liver, which metabolically alters, degrades, and detoxifies most substances.

• section III

MODE OF ACTION OF TOXIC SUBSTANCES

HERBERT E. STOKINGER, PH. D.

Toxic substances exert their effects by physical, or by chemical or physiologic (enzymatic) means, or by a combination of both.

The classification as presented here of the toxic mechanisms in the mammalian host has no precedent or any accepted basis other than that it appears to be inclusive, reasonable, and practicable. The classification has been developed to delineate two basic actions: the action of the toxic substance on the host, and the action of the host on the toxic substance. For it is the interplay of these two actions, together with the rate at which the body excretes the toxic substance, that determines what is called the toxicity of a substance.

Thus, the full toxic potential of most substances is not usually asserted, because of destructive actions by the body and its mechanisms of elimination by urine, sweat, feces, and exhalations, or because of sequestration in inactive forms at certain tissue sites such as bone, skin, hair, and nails. If this were not so, synergistic or enhanced toxicities would never be manifest. Synergistic or enhanced toxicities arise from the development of unusual or enhanced concentrations of the toxic substance. This occurs when one or more of the usual means of elimination or reduction of the toxic substance are blocked.

It is to be recognized that the following classification on toxic mechanisms must necessarily be based on prevailing knowledge, which varies greatly from discipline to discipline. In enzymology, for example, the state of knowledge is at the molecular, and in some instances, at the submolecular level. Such a situation obviously permits more exact definition of the governing mechanisms than is afforded by a discipline in which knowledge is at a cellular or organ level. Thus, a mechanism regarded at present as physical might be later labelled chemical or enzymatic to reflect the acquisition of new knowledge at a more intimate level. Indeed, when all mechanisms can be explained at the submolecular level, an entirely different classification will result. It is hoped that the classification, believed appropriately designated

within the limits of present knowledge, may not only provide greater insight into how chemicals act in the body but also point to possibly unsuspected relationships among the actions of diverse chemicals.

Physical Modes of Action

Harmful substances that have a solvent or emulsifying action can produce, after prolonged or repeated contact, a dry, scaly, and fissured dermatitis. This effect is commonly attributed to the physical removal of surface lipid, but may also be caused by denaturation of the keratin or injury to the water barrier layer of the skin. Acidic or alkaline soluble gases, vapors, and liquids, may dissolve in the aqueous protective film of the eye and mucous membranes of the nose and throat, and in sweat, causing irritation at these sites. Moreover, such insults may erode teeth and produce changes in hair structure.

On the inner surfaces of the body, the lungs and gastrointestinal tract, physical contact of unphysiologic amounts of substances causes irritation. This may lead to inflammation, or produce contraction, as in the reflex constriction of the respiratory passages upon inhalation of an irritant gas with resultant coughing, choking, or asphyxiation. In the upper gastrointestinal tract, the effect may include vomiting and, further down in the tract, the irritation may result in peristalsis and defecation.

Inert gases can exert serious and often fatal effects simply by physical displacement of oxygen, leading to asphyxia. Under pressure, inert gases such as nitrogen can produce compressed air illness by dissolving in unphysiologic amounts in the blood, lymph, and intercellular spaces, or may rupture delicate membranes such as the eardrum. Sudden, or too rapid, decrease in pressure results in decompression sickness. Less inert gases such as carbon dioxide and oxygen under greater than atmospheric pressure can lead to narcosis and other more serious effects, such as nerve and brain damage.

Physical adsorption of gases or vapors on solid or liquid particulates (aerosols), may, upon inhalation, lead to physiologic effects out of proportion to that anticipated from their inhaled concentration prior to adsorption. The action is known as synergism when the effect of gas and particulate exceeds the sum of the effects expected from either alone, or antagonism when the effect is less than expected. A physical theory has been developed to explain these abnormal actions. It is based on molecular properties of gases, and accounts for the synergism, by postulating "adsorbed" layers of the gas on the particulate that, upon inhalation, carry to the sensitive lung tissue enormously increased concentrations of the gas that become localized point sources of contact. Synergism results when a rapid rate of desorption of the gas from particulate to the tissue occurs; antagonism, when the desorption rate is very slow or nonexistent.

An example of synergism is the inhalation of a mixture of sulfur dioxide and sodium chloride crystals in which the effects on broncho-constriction are greater than that from the same concentration of inhaled gas. Sodium chloride inhaled alone is inert. An example of antagonism is the inhalation of welding fumes of nitrogen oxides and iron oxides; reduction of effect in this case is explained on the basis of a firmly *combined* layer of nitrogen oxides on the iron oxide particles.

Radioactive particles cause dislocation and breaking of chromosomal linkages apparently from local energy release.

Chemical or Physiologic Modes of Action

Substances that act chemically to produce injurious effects on the organs and tissues of the body do so by two basic means, either by depression or stimulation of normally functioning pathways of metabolism. These two effects are brought about by a variety of mechanisms that are known in only a general way for most toxic substances. But there are a few important substances such as carbon monoxide, cyanide, arsenic, and uranium, for which detailed mechanisms are known. In other cases, mechanisms are but partially known.

It is possible also that a single substance may have more than one pathway of action, or act by stimulation of an enzyme system at a low concentration of the substance and by depression at a higher concentration. This is a characteristic response of many, if not all, toxic substances, better known examples of which are arsenic, cobalt, vanadium, chloroform, and benzene.

It is convenient to consider chemical mechanisms under the following categories: (1) Primary mechanisms of injury which involve interactions of the toxic substance at the enzymatic level; (2) nonenzymatic interactions which involve more or less direct chemical combination or replacement of the toxic substances with a body constitutent without enzyme intervention; and (3) secondary mechanisms of injury that may involve both enzymatic and nonenzymatic actions resulting in injury only indirectly as a consequence of the presence of the toxic substance.

Primary Enzymatic Mechanisms

Most of the metabolic activity of the body is a result of the activity of enzymes, biologic *catalysts* formed by living cells throughout the body. Consequently, it is reasonable that the bulk of all toxic mechanisms should involve interference in some way with normal enzyme activity.

Enzymatic actions occur throughout the body without restriction to any particular organ site, although the liver cells perform a major proportion of the metabolic activity of the body. Equally active, however, but less diversified, are all other tissues in the body, the lung, kidney, intestine, brain and nervous tissue, and bone as well. From this it may be inferred that

enzymatic mechanisms may occur with the enzyme situated at nerve endings, within the nerve cell itself, or at cell surfaces.

It may be observed that two groups of enzymes, phosphatases and dehydrogenases, are commonly involved in a large variety of toxic mechanisms. The reason for this is that the two groups are included in a large number of important enzyme systems in the body.

In "metabolizing" a toxic substance, it is important to observe that the enzyme is merely performing a function that it normally performs in metabolizing natural foodstuffs; no special enzymes exist to metabolize toxic substances.

Although substances are toxic for a variety of causes, one of the causes is the frequent inability of enzymes to metabolize completely, and thus to destroy, the toxic substance. The reason for this is attributable to the rather high specificity, or selectivity, of the enzyme for the substance it is attacking, the *substrate.*

Enzymes are proteins, highly complex interlocking chains of amino acids, possessing to a marked degree specific, spatial orientation of the chemical constituents. The orientation of the enzyme is such that it fits, much like the key to the lock, the substrate with which it combines prior to modifying it.

It is now recognized that certain enzymes heretofore considered homogeneous in composition and in action may consist of several distinct components, each still acting, however, on the same substrate; these components are called *isoenzymes.*

The substrates which enzymes act on with highest efficiency are those with chemical structures and configurations of natural foodstuffs. Foreign, toxic substances do not possess these precise spatial requirements. It is thus apparent why enzymes only incompletely metabolize toxic agents.

Many enzymes have additional specificity requirements, in that they require a metal or a vitamin, or both, as *cofactor*(*s*) or *activator*(*s*). For example, the enzyme cocarboxylase, that splits carbon dioxide from certain organic acids, requires vitamin B_1 and magnesium ions as necessary constituents before it can function.

Because enzymes are proteins, they exhibit the physical and chemical properties of proteins. They undergo *denaturation* (1) by heat, as in burns, (2) by marked changes in acidity or alkalinity as effected, for example, by contact with corrosive agents, or (3) by chemical denaturing agents, such as urea in high concentrations. These agents alike cause structural and configurational changes in the protein, and the characteristic specificity is lost, and with it the catalytic activity of the enzyme.

Enzymes may become inactivated to varying degrees by less drastic means, however. Among those enzymes requiring a specific metal as activator, any agent that will displace or render inactive this metal will render the enzyme

inactive to the degree that the metal was rendered inert. Certain metals with similar spatial requirements for the specific metal required by the enzyme may do this. Certain poisonous metals such as beryllium are believed to act in this way. Cyanide may combine with the iron of an iron-dependent enzyme and inactivate or *inhibit* the enzyme.

Another common way an enzyme may become inhibited is from competition with a substance whose structure is sufficiently similar to the natural substrate but does not quite fulfill the spatial requirements of the enzyme. This is probably the most common way in which toxic substances exert their effect on enzymes.

A third way by which enzyme activity is inhibited is by accumulation of the product of the enzyme's activity. This is one of the natural ways by which body enzyme activity is regulated.

Like other catalysts, enzymes theoretically undergo no net change during the reactions they catalyze. Within a minute, one molecule of an enzyme can alter many thousand molecules of the substrate (*turn-over-rate*). In no case does the enzyme contribute to the net energy requirements of the reaction and only those reactions that are energetically possible without an enzyme can occur in its presence. Enzymes merely accelerate a chemical reaction. They catalyze the backward as well as the forward direction of the reaction.

The fundamental aspects of enzyme activity with respect to toxicity may be summarized as follows. Enzymes combine with the toxic substance. This combination may be inhibited partly or completely by the toxic substance, or the enzyme may act on the toxic substance more or less incompletely, possibly with the production of even more toxic substances. If the enzyme whose activity is blocked is a critical one, the slowing down may occur of some vital function resulting in alteration of cellular constituents in amount or type, or even in cell death.

A discussion follows of presently known enzymatic mechanisms.

Direct combination—The simplest way by which a toxic substance can alter enzyme action is by direct combination of the substance with active groups on the enzyme structure. Such is believed to occur with certain metals as mercury and arsenic which combine so tightly with the active group of the enzyme that further action is blocked. If the enzyme or enzymes represent critical systems for which there is no shunt mechanism, then cells may die or function subnormally resulting ultimately in injury to the cell, the organ, and the host. Similarly, nonmetallic substances such as cyanide can combine with and block the action of heavy metal-bearing enzymes because of the production of an inactive metal-cyanide enzyme. The blocking of this enzyme system to a significant degree results in the well-known fatal cyanide poisoning.

Another mechanism of poisoning by direct combination is illustrated by substances such as ozone and nitrogen dioxide, and possibly iodine and

fluorine, that destroy enzymes by oxidation of their functioning groups. In these cases, specific chemical groups such as -SH and -SS- on the enzyme are believed to be converted by oxidation to nonfunctioning groups; or the oxidants may break chemical bonds in the enzyme leading to denaturation and inactivation.

One of the more commonly encountered enzyme inhibition mechanisms in occupational exposures is that of the inhibition of the action of cholinesterase (acetylcholine esterase), an enzyme that regulates nerve-muscle action by destroying the muscle excitor acetylcholine. This muscle excitor is a powerful pharmacologic substance which if not destroyed when it is free can act as a poison. The destruction is accomplished by the hydrolysis of the potential poison into its components, an acetyl group and choline. A large number of pesticides, chiefly organic phosphates, act in the body by blocking this enzyme action, thus allowing excessive amounts of the muscle stimulator to accumulate. The excessive stimulation results in paralysis and prostration.

Competitive inhibition—A second, and one of the more usual toxic mechanisms involving enzymes, is that of competition of the toxic substance with normal metabolites, or the cofactor(s) essential for enzyme action, for the site of action on the enzyme. This form of competition is highly effective, and thus injurious, only when the chemical structure of the competing toxic substance resembles that of the constituent normally used by the enzyme; the closer the structural similarity, the more effective the competition.

The successful competition of an unnatural or foreign toxic substance for the enzyme sites of action blocks normal action by not permitting either significant amounts of normal substances to be metabolized, or by preventing combination of a cofactor necessary for enzyme action. The cofactor can be a metal or a highly complex specific organic substance such as a vitamin.

Competitive inhibition, first shown to be the action of sulfanilamide by reason of its close similarity to the B vitamin, para-aminobenzoic acid, has been demonstrated to function similarly in many other drug actions; it is also the basis of the mechanism of action of a number of anticancer drugs, many of which are appreciably toxic, for example, the fluoropyrimidines.

Toxic mechanisms may operate also by metal-to-metal competition. For example, it is believed that the poisonous action of beryllium results from its capacity to compete effectively for the sites of combination of magnesium and manganese on critical body enzymes, by which action the enzyme is no longer able to function at its normal rate or may be inactivated completely. This competitive inhibition of foreign metals is a very general way by which metals exert their toxic action.

A highly interesting example of a competitive mechanism is that recently found to explain the increased toxicity sustained following simultaneous exposure to two structurally similar economic poisons, malathion and EPN. Alhough EPN is highly toxic, malathion has a far lower order of toxicity.

When the two substances are present in the body together, however, malathion has a toxicity equalling that of EPN, and the summated toxicities of both is far beyond expectation.

The explanatory mechanism is found to reside in the fact that, inasmuch as both substances have chemically similar structures, EPN effectively competes for the same enzyme that hydrolyzes and thus would otherwise reduce the toxicity of malathion. By inhibiting this enzyme action, the concentration of the toxic form of malathion is maintained at a high level in the body, and consequently the toxicity is enhanced.

This is not an isolated instance of such a competitive mechanism. A number of other combinations of economic poisons are believed to produce enhanced toxicities by similar mechanisms, for example, the combinations malathion and dipterex^{R}, and guthion^{R} and dipterex^{R}. Other similar examples, but not involving the organic thiophosphates, undoubtedly will be found.

"Lethal synthesis"—Another means by which enzymes are involved in toxic mechanisms concerns the synthesis of a new toxic product by enzyme action on the toxic substance originally taken into the body. The newly synthesized product then exerts its toxic effect by interfering with normal metabolic processes.

A striking example of a substance involved in this type of mechanism is the rat poison 1080, sodium fluoroacetate. Following its absorption into the body, an enzyme transfers the fluorine atom in fluoroacetate to citric acid, an important intermediate in the cycle of terminal metabolism. The converted fluorocitrate, unable to function to a significant degree in this important metabolic cycle, breaks the metabolic chain of activity, with the result that tissue respiration ceases, and death ensues.

Toxic enzymes—A rather unusual type of toxic mechanism results when the toxic substance itself is an enzyme. A number of these instances are known. They are associated with the introduction into the body of such substances as snake and bee venoms and bacterial toxins. Although these substances exhibit a variety of toxic manifestations, the mechanisms of some of which are as yet unknown, the venoms of bees and certain snakes possess enzymes (phosphatidases) that lyse red blood cells destroying the oxygen-carrying power of the blood, as well as enzymes (proteolytic) that destroy cells and inhibit blood coagulation. In addition, bee venom contains a substance that inhibits dehydrogenases, enzymes important in the metabolism of many body functions. Snake bites are currently an occupational hazard in certain areas of the United States.

Inducible enzymes—Thus far all of the mechanisms discussed have been depressant in action. As indicated previously, response of toxic substances may under certain conditions act as stimulants to metabolic activity. In this category may be placed inducible (adaptive) enzymes, by which is meant the physiologic synthesis of additional amounts of an enzyme in response to the

presence of an inducing agent. In this instance, the inducing agent is a toxic substance.

Because inducible enzymes are difficult to demonstrate in the mammalian host (although a number have been so demonstrated in bacteria and yeasts), only one instance of industrial health interest is presently known in sufficient detail; undoubtedly others will be found. High sucrose diets fortified with vitamins fed for 3 weeks to rats stimulate the enzymatic production of additional amounts of protein sulfhydryl groups in the kidney, which enables the rats to withstand otherwise lethal doses of mercury. The newly-formed sulfhydryl binds the mercury firmly, thus effectively reducing its toxic potential.

A mechanism exemplifying stimulation, probably mediated through inducible enzymes, is the increased production of serum alpha globulins by cobalt when absorbed into the body at relatively low levels of intake. At slightly higher levels of intake, cobalt stimulates the production of increased amounts of red blood cells (polycythemia production); associated with the polycythemia is increased production of hemoglobin. The exact mechanism of this stimulation is not known, but a new hormone, erythropoietin, whose production is stimulated by cobalt, is believed involved. It appears also that the action of erythropoietin is not entirely restricted to stimulating bone marrow to increased production of red cells but may include stimulation of other centers as well.

Nonenzymatic Mechanisms

There are a number of industrially important types of poisoning which proceed through mechanisms that do not involve the intervention of enzyme action but for which the energy is supplied, so far as is known, by chemical action.

Direct chemical combination—Among the best known and understood mechanisms of poisoning is that of direct chemical combination of the toxic substance and a body constituent, as illustrated by carbon monoxide poisoning. In this instance, the gas combines rapidly and rather firmly with hemoglobin forming a new compound, carboxyhemoglobin, that cannot perform the usual function of hemoglobin, which is the transport of oxygen to the tissues.

Hydrogen sulfide likewise unites with hemoglobin to convert it to sulfhemoglobin, a nonoxygen carrying pigment, although this mechanism is not important in hydrogen sulfide poisoning.

Release of body constituents—A less well understood mechanism of injury, but on which there is nevertheless an enormous amount of indirect evidence, is the release by toxic substances of natural body constituents in abnormal amounts that lead to injury and even death. Instances of this mechanism are numerous and involve the intake into the body of such common substances as "hay-fever" allergens or other allergenic materials, for example, tolylene diisocyanate.

Intake of these substances results in release of histamine or histamine-like substances in local large amounts with the characteristic development of inflammation, edema, and other evidences of injury. A large number of amines are capable of histamine release; in these instances the mechanism involved is believed to be one of displacement, whereby the tissue-bound histamine is displaced and liberated by the unnatural amine. Similarly, any type of simple cellular damage results in the liberation of histamine-like substances.

There is accumulating evidence also that release of hormones from nerves may be the common mechanism by which a number of chemical substances exert their toxic action. The example that follows not only illustrates an action that releases body constituents, but also illustrates a highly indirect toxic action formerly believed to be a direct effect on a substance on an end organ.

Carbon tetrachloride has been shown to cause the massive discharge of epinephrine and related neurohumors from central sympathetic nerves. This discharge possibly mediated by enzyme action results in the stimulation of the nerve supply to the blood vessels of the liver to produce (1) restriction of the liver's blood flow leading to reduced oxygen transport and, ultimately, the characteristic centri-lobular necrosis of the liver and (2) release of un-esterified fatty acids from fat depots and their deposition in the liver to produce the well known "fatty" liver of carbon tetrachloride poisoning.

Chelation—A toxic mechanism that is increasingly being recognized to be one of the more common pathways of toxic action is chelation. Chelation is the term applied to the chemical combination of an organic structure and a metal whereby the metal is very firmly bound to the organic substance by both nonionic (organic) and ionic bonding. For example the therapeutic agent EDTA binds metals by chelation. Many drugs and antibiotics are now believed to act by chelation. By so acting, these substances exert their effects in a number of ways:

(1) By removal of biologically active metals that are normally bound in the cell or its components with resulting inactivation and cell damage. For example, treatment of lead poisoning with EDTA may in addition remove other metals such as zinc, that is required for important functions in certain kidney enzymes (carbonic anhydrase).

(2) By reacting with fixed intracellular metals.

(3) By chelating firmly with a fixed tissue constituent. This is believed to be the mechanism by which boron, as borate, exerts its toxic action. Borate is known to chelate with adjoining carbon atoms containing hydroxyl groups. If the structure prior to chelation happens to be a critical one in a metabolic chain, ordinary function ceases and injury occurs as a result of the altered chelated structure.

(4) By increasing the absorption of a toxic agent. Instances are being recognized of toxicity resulting from abnormally increased amounts of absorption into the blood stream by a chelating compound. Iron, normally nontoxic when absorbed by the usual regulatory mechanism, may under unusual circumstances be absorbed in toxic amounts by the mechanism of chelation to form a soluble, easily absorbed substance.

Stimulation of immune mechanisms—A mechanism whose toxic significance remains to be fully evaluated, but which nevertheless has been recognized for many years, is the stimulation of immune mechanisms as a result of the production of a new antigenic structure from the combination of a toxic substance with body constituents, usually protein. This mechanism is thought to be the basis of skin sensitivity resulting from contact with certain reactive organic substances, for example, the chloronitrobenzenes.

Another substance that illustrates this mechanism strikingly is tolylene diisocyanate and related aromatic isocyanates. These substances, upon inhalation, have unusual avidity for combining with body protein with resultant allergic sensitization of the respiratory tract.

Secondary Toxic Mechanisms

In this category are grouped those pathways of metabolism and mechanisms of injury that are not effected by the direct action of the toxic substance but develop either (1) as a result of metabolic alteration of the toxic substance following its entrance into the body, or (2) as a consequence of an accumulation of toxic by-products from the initial, direct action of the toxic substance. In the second instance, further injury occurs at a site in the body different from that of the original toxic action. Most, if not all, of the mechanisms considered here are performed by enzymes.

Detoxication (metabolic) mechanisms—Mechanisms grouped here comprise all those metabolic activities that the *body* performs on a toxic substance in contradistinction to the actions that the *toxic substance* performs on the body. The latter actions were considered under Primary Enzymatic Mechanisms, and Nonenzymatic Mechanisms. Broadly, the so-called "detoxication" mechanisms are those performed by the body in the process of attempting to eliminate the toxic substance, namely, oxidation, reduction, and synthesis. A few examples of each of these mechanisms will be given for well-known industrial substances of a toxic nature.

It will become apparent that the body does not always act to its own advantage when handling a foreign, and generally atoxic, substance. These peculiarly disadvantageous reactions result, however, merely because the body is equipped with certain definitive pathways of metabolism derived from past utilization of food components. These are its only resources when confronted with nonfood substances, and accordingly these mechanisms are used insofar as they can act to a degree on foreign substances bearing chemical structures similar in some respects to food substances. Whether

this indiscriminate action by the body's enzymes results in an outcome favorable or unfavorable to the body depends only on the nature of the resultant modified foreign substance and not on any selective or guided action of enzymes. Some examples of *oxidation, reduction,* and *synthesis* follow.

(1) *Oxidation* is one of the most general metabolic activities of the body against foreign substances. It includes the oxidation of alcohols to aldehydes, aldehydes to acids, oxidation of hydrocarbon rings to phenols and quinones, alkyl groups to alcohols and acids, oxidative removal of ammonia from amines, oxidation of organic sulfur compounds, oxidative splitting of carbon ring compounds, removal of halogens from halogenated hydrocarbons, and a variety of other reactions including the oxidation of certain metallic ions.

A well-known example in which secondary oxidative mechanisms are believed to play a dominant role in the toxicity of an alcohol is that of methyl alcohol. Oxidation to formaldehyde, which subsequently interferes with oxidative enzyme synthesis, is believed to be the pathway by which methyl alcohol exerts its injurious effect on the optic nerve leading to blindness. Ethyl alcohol, and presumably other alcohols, proceed through this metabolic pathway of oxidation to the corresponding aldehyde, which is responsible, in part at least, for the toxic effects.

Perhaps one of the more important and interesting examples in which oxidative mechanisms play a decisive role in the ultimate toxic response is the oxidation of the cancerigenic hydrocarbon, 3,4-benzpyrene. Current theories of cancerigenesis consider some oxidized product, not the original hydrocarbon, to be a step in the process leading to tumor development. Several oxidized products of 3,4-benzpyrene have been identified following its entry into the body including phenolic products and several quinones.

Similarly, the serious effect of the hydrocarbon, benzene, is believed to be the result of increasing oxidation of the benzene nucleus, first to phenol (monohydroxybenzene), then to dihydroxy- and trihydroxy-phenol, which are considered responsible for the toxicity of benzene. Further oxidation to quinone may be involved, followed by further oxidative cleavage of the benzene ring to form the relatively nontoxic mucic acid.

In this connection, it should be recognized that by no means do all metabolic alterations in the structure of toxic organic substances result in toxic by-products. A sizeable number of the metabolic products are detoxified in the process, as is reasonable.

An important and striking example of the role of oxidative mechanisms in developing the toxicity of an organic substance is parathion. This substance, containing sulfur in its molecule, is relatively nontoxic until oxygen replaces the sulfur forming paraoxon which is extremely toxic, inhibiting completely an important enzyme of nerve function, cholinesterase.

An example of oxidation among inorganic toxic substances is that of uranium. The tetravalent form is unstable to the body's oxidation-reduction potential, and is oxidized to the more toxic hexavalent form. The hexavalent form then combines with active sites (phosphate groups) on the surface of cells, blocking normal metabolic processes necessary for cell survival.

Much, if not all, of the toxicity of the long-recognized poisoning action of aniline arises not from aniline itself, but from its various oxidation products formed in the body. The more important of these are para-aminophenol and, by further oxidation, the quinoneimine which is believed responsible for the methemoglobinemia that develops when aniline, or other aromatic amines, are absorbed into the body. The oxidized product of aniline oxidizes the ferrous iron of hemoglobin to the ferric form, resulting in methemoglobin, incapable of releasing oxygen.

(2) *Reduction* is far less common a body function than oxidation. Nevertheless several types of foreign organic substances are metabolized by this pathway to produce one or more substances that are more injurious than the parent substance. Among certain of the inorganic metal ions, reduction is also the pathway of metabolism. Organic nitro-groups are reduced by stages to amines. Some aldehydes are reduced to alcohols. Unsaturated double bonds of carbon compounds may add hydrogen and thus become reduced. These types are not an exhaustive listing.

In general, however, reduction, contrary to oxidation, tends to result in products that are less toxic than the original substance, for example, reduction of aldehydes to alcohols, and are thus of lesser interest here. On the other hand, metabolism of nitrobenzene results in a number of products, one of which, para-aminophenol, is from 50–80 times more acutely toxic than the parent nitrobenzene.

Among inorganic ions, pentavalent arsenic is relatively inactive in the body until reduced to the trivalent state. The physiologically active form of manganese is trivalent. If manganese is taken into the body in the form of pyrolusite in which the manganese is tetravalent, reduction to the active form must occur, at least to that portion which is absorbed into the blood stream and later incorporated into active tissue components.

(3) *Synthesis*, whereby the body contributes some tissue constituents in the conversion of the foreign substance to a new product, is one of the more common means the body has of disposing of the toxic agent. There are a dozen known synthetic mechanisms to accomplish this. Without listing them all, the addition of such substances as sulfate, sulfur, glucose, and protein derivatives to the toxic substance in general results in true detoxication and lessening of the injurious effects of the foreign substance.

The well-known synthesis of phenylsulfate, which was one of the earliest synthetic mechanisms to be discovered (1876), converts highly toxic phenol to a substance which is practically nontoxic. Cyanide, both inorganic and

organic forms, is synthesized to thiocyanate, a structure many times less toxic than cyanide. Certain toxic metal ions may react with sulfur of the body to be excreted as insoluble, and, thus nontoxic, metal sulfides.

It should be pointed out that these synthetic detoxifying mechanisms are not entirely free of injury to the body. In contributing some of its constituents, the body may deprive itself of vital amounts of these substances if synthesis is prolonged, and thus injure itself.

Secondary organ involvement—A secondary mechanism of very general nature, and of considerable toxicologic importance, involves the indirect action of either the toxic agent or its metabolic by-products, or both. Once having injured a primary site, the substance(s) causes either the production or accumulation of deleterious products that in turn affect a secondary site.

A striking example of this secondary mechanism is the action of hexavalent uranium, which first injures the kidney in such a way as to prevent normal elimination of waste products such as urea, ammonia, and other substances. These products accumulate in the blood stream and injure the liver, resulting in fatty degeneration of this organ.

Similar indirect injury occurs to the heart when the lung, through direct injury by some toxic substance, restricts blood flow thus placing undue stress on the heart.

There are numerous other examples; in fact, the function of the body is so organized that there are few alterations of significant magnitude in an organ or tissue site that do not have repercussions in some other organ even at a remote site. The interlocking activities of the endocrine glands, with their respective hormones and their dependence on vitamins and minerals for normal function, is the basis for this entire group of secondary mechanisms.

An interesting example of the involvement of these highly sensitive interlocking endocrine systems is the simple inhalation of nonlethal concentrations of ozone, which produces alterations in the activities of the adrenal glands and disturbs the normal uptake of iodine by the thyroid gland, which in turn alters the activity of the thyroid-stimulating hormone of the pituitary body.

Conclusion

Reference was made to different physical modes of action of harmful substances and the effects of such substances following contact with outer and inner body surfaces.

The discussion of toxic mechanisms attempted to present as simply and briefly as possible a unified and comprehensive view of the entire field which, as is seen, is highly complex and involves most of the vital functions of the body.

218-695 O-66-3

References

BOYER, P. D.: Mechanism of enzyme action. 191 references. *In* Luck, J. M.; Allen, F. W., and Mackinney, G. (editors): *Annual Review of Biochemistry.* Vol. 29. Annual Reviews, Palo Alto, Calif., 1960.

BRODY, T. M.: Mechanism of action of carbon tetrachloride. *Fed. Proc.* 18: 1017, 1959.

COHEN, A. E.; SCHEEL, L. D.; KOPP, J. F.; STOCKELL, F. R., JR.; KEENAN, R. G.; MOUNTAIN, J. T., AND PAULUS, H. J.: Biochemical mechanisms in chronic carbon disulfide poisoning. *Am. Indust. Hyg. Assoc. J.* 20: 303, 1959.

MCLEAN, R. A. AND BERRY, L. J.: Adrenergic inhibition and lethal effect of bacterial endotoxin. *Proc. Soc. Exper. Biol. & Med.* 105: 91, 1960.

MURPHY, S. D. AND DUBOIS, K. P.: Quantitative measurement of inhibition of the enzymatic detoxication of malathion by EPN. *Proc. Soc. Exper. Biol. & Med.* 96: 813, 1957.

SCHEEL, L. D.; DOBROGORSKI, O. J.; MOUNTAIN, J. T.; SVIRBELY, J. L., AND STOKINGER, H. E.: Physiologic, biochemical, immunologic and pathologic changes following ozone exposure. *J. Appl. Physiol.* 14: 67, 1959.

SEVEN, M. J. AND JOHNSON, L. A.: *Metal-Binding in Medicine.* J. B. Lippincott Co., Philadelphia, 1960.

STOKINGER, H. E.: Toxicologic interactions of mixtures of air pollutants; review of recent developments. *Int. J. Air Poll.* 2: 313, 1960.

STOKINGER, H. E. AND MOUNTAIN, J. T.: Test for hypersusceptibility to hemolytic chemicals. *Arch. Environ. Health* 6: 495, 1963.

WEINBERG, E. D.: The mutual effects of antimicrobial compounds and metallic ions. *Bact. Rev.* 21: 1, 1957.

WILLIAMS, R. T.: *Detoxication Mechanisms. The Metabolism and Detoxication of Drugs, Toxic Substances and Other Organic Compounds.* 2nd ed. Chapman & Hall, London, 1959.

• section IV

OCCUPATIONAL DERMATOSES

DONALD J. BIRMINGHAM, M.D. AND MARCUS M. KEY, M.D.

An occupational dermatosis is any cutaneous abnormality resulting directly from, or aggravated by, the occupational environment. Numerous indirect factors contribute to the development of an occupational dermatosis. Among them may be age, race, sex, season, the pre-existence of other skin diseases, type of skin, perspiration, and poor personal hygiene.

The term occupational dermatitis is a more restrictive one, signifying inflammation of the skin, or an eczematous process; excluded are neoplasms, infections, and certain pigmentary disturbances. Because contact is the usual means of acquiring an occupational dermatitis, the terms occupational dermatitis and contact dermatitis are used interchangeably in industry. The direct causes of contact dermatitis, chiefly chemicals and plants, may be divided into primary irritants and allergic sensitizers.

Because of their external location, the occupational dermatoses are readily accessible for diagnosis and treatment. The most important of these diseases is contact eczematous dermatitis which usually can be treated, and further attacks averted, simply by prevention of contact.

Physiologic Protective Attributes of Skin

Many of the skin's physiologic attributes participate in resisting the various insults that produce ocupational skin diseases. Keratin, the outermost layer of the epidermis, is resistant to short-term immersion in water and to weak acids, but is vulnerable to the action of alkalis, detergents, strong soaps, solvents, and to prolonged immersion. The lipid emulsion on the surface of the keratin is composed of sebum, degradation products of keratin, and sweat. To a limited extent, the emulsion impedes the entrance of water and water-soluble chemicals and resists changes in the pH of the skin surface, but it is removed by solvents, soaps, detergents, emulsions, and prolonged immersion in water.

The transitional layer between the cornified and noncornified epidermis is an important barrier which prevents the loss of water from the skin and the entrance of most water-soluble chemicals. This subcorneal barrier layer

may be damaged by maceration, mechanical or chemical trauma, and certain internal diseases. Lipid-soluble and nonionizable (organic) substances are able to bypass the barrier layer and enter by way of the transfollicular pathway. Gases, except for carbon monoxide, are readily exchanged through the epidermis.

Protection against actinic stimuli is afforded by increased melanin production and a concomitant thickening of the keratin layer. An antimicrobial action is attributed to certain fatty acids in the surface lipid film, and the subcorneal barrier layer also offers some protection against microorganisms.

Primary Irritants

Primary irritation causes most of the cases of contact dermatitis in industry. A primary irritant is an agent which will cause dermatitis by direct action on the normal skin at the site of contact if it is permitted to act in sufficient intensity or quantity for a sufficient length of time. Thus, the normal skin will almost always react to a primary irritant if the necessary conditions are present.

Irritants can act in several ways to injure the skin. Several examples have already been given, such as removal of lipid film, denaturation of keratin, and interference with the subcorneal barrier layer. Other examples of primary irritation include dehydration by inorganic acids, anhydrides, and alkalis; protein precipitation by heavy metal salts and tanning agents; and oxidation by bleaches, chlorine compounds, and per- salts.

Sensitizers

Ordinarily, no more than 20 percent of contact dermatitis in industry is caused by allergens. However, certain of the strong sensitizers may affect many workers. Almost any chemical can be a sensitizer, but the mode of action usually differs from that exhibited by primary irritants. The cutaneous sensitizer, therefore, does not necessarily cause demonstrable cutaneous change on first contact, but may effect such specific changes in the skin so that after 5 to 7 days or more, further contact on the same or other parts of the body may cause dermatitis.

The difference between the irritant and the sensitizer, therefore, is generally a matter of time as well as mode of action. The irritant will usually act within a matter of minutes to a few hours, whereas the sensitizer requires at least 5 to 7 days because sensitization must build up during the period of incubation. Low-grade irritants such as soap may require prolonged or repeated contact before a dermatitis appears, and this prolonged reaction time may be confused with allergic incubation.

Secondary Effects

After the cutaneous defenses have been broken down and a contact dermatitis is present, the oozing or fissured surface offers ideal conditions for the entrance and growth of bacteria. Thus, it is not unusual for a contact

dermatitis to be secondarily infected. Another secondary effect commonly seen in contact dermatitis is over-treatment by irritating or sensitizing medication.

Diagnosis

Diagnosing an occupational dermatosis is generally contingent upon satisfying certain criteria. The disease should look like a contact dermatitis or one of the other clinical types of occupational dermatoses. It should be located on sites of exposure, and the time of appearance of the eruption as well as periods of remisson and exacerbation should correlate with the history of work exposures. When the patient does not get well following complete withdrawal from the suspected contact agent, stimuli of nonoccupational origin should be reinvestigated. Multiple factors not associated with the occupation can perpetuate a chronic dermatosis.

Patch tests are helpful in differentiating between a primary irritation dermatitis and an allergic contact dermatitis. A nonirritating concentration of the suspected allergen is applied to the normal skin of a patient for 24 to 48 hours in an attempt to reproduce an eczematous dermatitis beneath the covering patch.

Classification of Lesions

The clinical lesions seen among occupational exposures are multiple, varying from the mildest erythema to lesions of neoplastic nature. However, occupational skin disease can be classified as follows:

(1) Acute contact eczematous dermatitis characterized by erythema, edema, papules, vesicles, or bullae, crusts and finally desquamation. These effects are generally the result of contact with either a primary irritant or a sensitizer, or with both.

(2) Chronic contact eczematous dermatitis characterized by erythema, lichenification, and fissuring of the skin, usually resulting from contact with dehydrators, fat solvents, soaps, and detergents.

(3) Folliculitis and acneform types, including chloracne, characterized by plugged sebaceous follicles and suppurative lesions. Chloracne also shows numerous straw-colored cystic lesions. These forms of occupational dermatoses are usually caused by contact with oils, tars, waxes, and certain chlorinated hydrocarbons.

(4) Neoplastic (benign and malignant) types, characterized by keratoses, papillomata and epitheliomata of the exposed areas, usually caused by petroleum products, coal tar and certain derivatives, sunlight, and ionizing radiation.

(5) Pigmentary disturbances characterized by increase or decrease of pigment in the epidermis. Increased pigmentation can result from contact with

coal tar compounds, certain petroleum oils, vegetables, and fruits. Decreased pigmentation may result from trauma, dermatitis, or from contact with monobenzyl ether of hydroquinone.

Potential Occupational Exposures

The following is a list of occupations each accompanied by certain agents frequently associated with that occupation and capable of producing a dermatosis. Additional agents for the occupations listed as well as additional occupations will be found in other sections, principally the one on chemical hazards.

ABRASIVE WHEEL MAKERS
- abrasive dusts
- resin glues

AGRICULTURAL WORKERS
- *See* Farmers

AIRCRAFT WORKERS
- bichromates
- chlorinated solvents
- chromates
- chromic acid
- cutting fluids
- cyanides
- glass fiber
- hydraulic fluids
- hydrofluoric acid
- lubricants
- nitric acid
- oils
- paints
- plastics
- resins
- rubber
- solvents
- ultraviolet light
- vibrating tools
- X-rays

ANIMAL HANDLERS
- bacteria
- fungi
- insecticides
- parasites
- pesticides
- viruses

AUTOMOBILE WORKERS
- asbestos
- bichromates
- brake fluids
- cutting fluids
- epoxy resins
- gasoline

AUTOMOBILE WORKERS—continued
- lead
- lubricants
- oils
- paints
- plastics
- polyester resins
- rubber
- solvents
- thinners

AVIATION MECHANICS
- chlorinated solvents
- fuels
- hydraulic fluids
- lubricants
- oils and zinc chromate
  - used as aluminum-oxidation inhibitors

BAKERS
- cinnamon
- dough
- dusts
- flour improvers
- fungi
- heat
- monilia
- sugar

BARBERS AND HAIRDRESSERS
- ammonium thioglycolate
- bacteria
- cosmetics
- depilatories
- detergents, synthetic
- dyes
- fungi
- hair tonics
- lacquer removers
- nail lacquers
- perfumes
- soaps

BARBERS AND HAIRDRESSERS—continued
- ultraviolet light
- wave solutions

BARREL WASHERS
- bleaches
- deodorants
- detergents, synthetic
- soaps
- sodium carbonate
- solvents
- trisodium phosphate

BARTENDERS
- citrus fruits
- detergents, synthetic
- monilia
- soaps

BASKET WEAVERS
- essential oils
- fungi

BATH ATTENDANTS
- fungi
- linaments
- oils
- tonics
- ultraviolet light

BATTERY MAKERS
- benzol
- carbolic acid
- glass fiber
- mercury
- pitch
- sulfuric acid
- tar
- zinc chloride

BLEACHERS
- chlorine compounds
- chromium compounds
- hydrochloric acid
- hydrofluoric acid
- hydrogen peroxide
- nitric acid
- oxalic acid
- per-salts
- potassium hydroxide
- sodium hydroxide
- sodium silicate
- sulfur compounds

BOOKBINDERS
- formalin
- glues
- inks

BOOKBINDERS—continued
- oxalic acid
- shellac
- solvents

BRICK MASONS
- cement
- cold
- epoxy resins
- lime
- moisture
- sunlight

BRIQUETTE MAKERS
- coal tar pitch

BRONZERS
- acetone
- ammonia
- ammonium sulfide
- amyl acetate
- antimony sulfide
- arsenic
- arsine
- benzine
- benzol
- cyanides
- heat
- hydrochloric acid
- lacquers
- mercury
- methyl alcohol
- petroleum hydrocarbons
- phosphorus
- resins
- sodium hydroxide
- sulfur dioxide
- turpentine
- varnishes

BROOM AND BRUSH MAKERS
- bacteria
- bleaches
- colophony resin
- dust, vegetable
- dyes
- fungi
- glues
- parasites
- pitch
- plastics
- rubber
- shellac
- solvents
- tar

BROOM AND BRUSH MAKERS—continued
varnish
woods

BUTCHERS
antibiotics
bacteria
detergents, synthetic
fungi
moisture

BUTTON MAKERS
bacteria
dusts
dyes
hydrogen peroxide
plastics

CABINET MAKERS AND CARPENTERS
bleaches
glues
oils
rosin
shellac
solvents
stains
synthetic resins
varnish
woods
See also Woodworkers

CABLE WORKERS AND SPLICERS
chlorodiphenyls
chloronaphthalenes
dyes
epoxy resins
solvents

CANDLE MAKERS
ammonium chloride
ammonium phosphate
ammonium sulfate
borax
boric acid
chlorine
chromates
hydrochloric acid
potassium nitrate
sodium hydroxide
stearic acid
waxes

CANDY MAKERS
chocolate
citric acid
essential oils
pineapple juice

CANDY MAKERS—continued
spices
sugar
tartaric acid

CANNERS
bacteria
citrus oil
dyes
fruit acids
fungi
moisture
monilia
parasites
resins
salt
vegetable juices

CAP LOADERS, PERCUSSION
mercury compounds

CARPENTERS
See Cabinet makers and carpenters

CARPET MAKERS
alizarine
aniline dyes
anthrax bacillus
bleaches
chlorine
fungicides
glues
insecticides
loom oils
solvents

CARROTERS, FELT HAT
acids
mercury compounds, if used

CARTRIDGE DIPPERS
acids
soaps

CASE HARDENERS
heat
oils
sodium carbonate
sodium cyanide
sodium dichromate
sodium nitrite

CELLULOSE WORKERS
carbon disulfide
finishing oils

CEMENTERS, RUBBER SHOE
benzol
carbon disulfide

CEMENTERS, RUBBER SHOE—continued
coal tar products
methyl alcohol
naphtha

CEMENT WORKERS
cement
chromates
cobalt
epoxy resins
lime
moisture
pitch
resins

CHEMICAL WORKERS
See Chemical Hazards section

CHROME PLATERS
chromium compounds
degreasing solvents
sulfuric acid

CLERKS
adhesives
carbon paper
copy paper
duplicating fluid removers
duplicating materials
indelible pencils
ink removers
inks
rubber
solvents
type cleaners
typewriter ribbons

CLOTH PREPARERS
acids
alkalis
amino resins
detergents, synthetic
dyes
formaldehyde
fungicides
moisture
potassium salts
soaps
sodium metasilicate
sodium salts
sodium silicate

COAL TAR WORKERS
anthracene oil
benzol
coal tar
creosote

COAL TAR WORKERS—continued
cresol
naphtha
pitch
sunlight

COMPOSITORS
alkalis
inks
solvents

CONSTRUCTION WORKERS
adhesives
cement
cold
creosote
gasoline
glass fiber
oils
paints
pitch
solvents
sunlight
ultraviolet light

COOKS
fruit acids
heat
moisture
monilia
spices
sugar
vegetable juices

COTTON SIZERS
acids
aluminum salts
arsenic salts
calcium salts
carbolic acid
dicyanodiamide formaldehyde
fungicides
magnesium salts
melamine formaldehyde
sodium hydroxide
starch
urea formaldehyde
zinc chloride

DAIRY WORKERS
antibiotics
bacteria
detergents, synthetic
fungi
mites
viruses

DEGREASERS
- solvents

DEMOLITION WORKERS
- bacteria
- chemicals
- cold
- fungi
- moisture
- sunlight
- TNT
- ultraviolet light

DENTISTS
- anesthetics, local
- antibiotics
- disinfectants
- eugenol
- ionizing radiation
- mercury amalgams
- oil of clove
- phosphoric acid
- plastics
- soaps

DETONATOR CLEANERS, FILLERS AND PACKERS
- mercury compounds

DISHWASHERS
- bacteria
- detergents, synthetic
- grease
- moisture
- monilia
- soaps
- water softeners

DISINFECTANT MAKERS
- carbolic acid
- chloride of lime
- chlorine
- cresol
- formaldehyde
- iodine
- mercury compounds
- surfactants
- zinc chloride

DOCK WORKERS
- bacteria
- castor bean pomace
- chemicals
- cold
- fumigants
- fungi
- grains

DOCK WORKERS—continued
- heat
- insects
- irritating or infected cargoes
- mites
- moisture
- petroleum
- sunlight
- tar

DRUGGISTS
- acids
- alkalis
- antibiotics
- bleaching powder
- detergents, synthetic
- drugs
- iodoform
- soaps
- sodium salts
- sugar

DRY CLEANERS
- acetic acid
- ammonia
- amyl acetate
- benzine
- carbon tetrachloride
- chlorobenzene
- dusts
- methanol
- nitrobenzene
- perchloroethylene
- sizing compounds
- Stoddard solvent
- trichloroethylene
- turpentine
- waterproofing compounds

DYE MAKERS
- acids
- alkalis
- antimony compounds
- benzine
- calcium salts
- carbolic acid
- coal tar products
- cresol
- dextrins
- dye intermediates
- ferrocyanides
- formaldehyde
- gums
- hydroquinone

DYE MAKERS—continued
- lead salts
- potassium chlorate

DYERS
- acids
- alkalis
- bleaches
- detergents, synthetic
- dyes
- mercurial salts
- moisture
- solvents
- zinc chloride

ELECTRIC APPARATUS MAKERS
- acids
- asbestos
- enamels
- epoxy resins
- ionizing radiation
- phenolic resins
- pitch
- rubber
- solder fluxes
- solvents
- synthetic waxes
- varnishes

ELECTRICIANS
- chlorinated diphenyls
- chlorinated naphthalenes
- electricity
- epoxy resins
- solder fluxes
- solvents
- waxes, synthetic

ELECTROPLATERS
- acids
- alkalis
- benzine
- chromic acid
- heat
- lime
- moisture
- nickel
- potassium cyanide
- soaps
- waxes, synthetic
- zinc chloride
- zinc cyanide

EMBALMERS
- bacteria
- carbolic acid

EMBALMERS—continued
- formaldehyde
- fungi
- ionizing radiation
- mercury
- oil of cinnamon
- oil of clove
- thymol
- zinc chloride

ENAMELERS
- acids
- alkalis
- arsenic
- chromium
- cobalt
- nickel

ENGRAVERS
- acids
- alkalis
- ferric chloride
- potassium cyanide
- solvents
- tropical woods

ETCHERS
- acids
- alkalis

EXPLOSIVE WORKERS
- ammonium salts
- mercury compounds
- nitroglycerin
- PETN
- picric acid
- tetryl
- TNT

FARMERS
- antibiotics
- bacteria
- cold
- detergents, synthetic
- feeds
- fertilizers
- fruits
- fungi
- heat
- lubricants
- oils
- parasites
- pesticides
- poison ivy
- poison oak
- poison sumac

FARMERS—continued
ragweed
solvents
sunlight
vegetables

FELT HAT MAKERS
acids
bacteria
dyes
Glauber's salt
hydrogen peroxide
mercuric nitrate, if used
sodium carbonate

FERTILIZER MAKERS
acids
ammonium compounds
calcium cyanamide
castor bean pomace
fluorides
lime
manure
nitrates
pesticides
phosphates
potassium salts

FISH DRESSERS
bacteria
brine
cold
moisture
redfeed
sunlight
trauma

FLAX WORKERS
brine
lime

FLOUR MILL WORKERS
dust
fungi
parasites
pesticides

FOOD PRESERVERS
bleaches
brine
ionizing radiation
moisture
monilia
resins
spices
sugar

FOOD PRESERVERS—continued
vinegar
waxes

FOUNDRY WORKERS
acids
heat
lime
resins
ultraviolet light

FURNACE WORKERS
heat
ultraviolet light

FURNITURE POLISHERS
alkalis
benzine
methyl alcohol
naphtha
pyridine
rosin
soaps
turpentine
waxes

FUR PROCESSORS
acids
alkalis
alum
bacteria
bleaches
chromates
dyes
formaldehyde
fungi
lime
oils
salt

GALVANIZERS
acids
ammonium chloride
zinc chloride

GARAGE WORKERS
antifreeze solutions
detergents, synthetic
epoxy resins
gasoline
gasoline additives
glass fiber
greases
moisture
oils
paint removers
paints

GARAGE WORKERS—continued
- polyester resins
- solvents

GARDENERS
- fertilizers
- fungi
- fungicides
- herbicides
- insecticides
- insects
- plants
- poison ivy
- poison oak
- sunlight

GAS MANTLE MAKERS
- thorium compounds

GLASS WORKERS
- arsenic
- borax
- boric acid
- glass fiber
- glass wool
- heat
- hydrofluoric acid
- lead compounds
- lime
- metallic oxides
- petroleum oils
- resins
- soda ash
- ultraviolet light

HAIRDRESSERS
- *See* Barbers and hairdressers

HIGHWAY WORKERS
- *See* Road workers

HISTOLOGY TECHNICIANS
- alcohol
- benzol
- formaldehyde
- mercury bichloride
- osmium tetroxide
- potassium dichromate
- stains
- toluene
- waxes
- xylene

INK MAKERS
- anti-skinning agents
- chromates
- cobalt compounds

INK MAKERS—continued
- detergents, synthetic
- dyes
- ethyl acetate
- ethyl alcohol
- mercuric chloride
- soaps
- solvents
- turpentine
- varnish

INSECTICIDE MAKERS
- aldrin
- allethrin
- arsenic trioxide
- calcium arsenate
- chlordane
- DDT
- dieldrin
- lindane
- malathion
- methoxychlor
- parathion
- piperonyl compounds
- pyrethrin
- strobane
- *See also* Pesticides section

JANITORS
- bacteria
- detergents, synthetic
- disinfectants
- polishes
- soaps
- solvents
- waxes

JEWELERS
- acids
- chromates
- cyanides
- mercury
- mercury solvents
- nickel
- solder fluxes

LABORATORY WORKERS, CHEMICAL
- acids
- alkalis
- chromates
- detergents, synthetic
- moisture
- organic solvents
- soaps

LAUNDRY WORKERS
- alkalis
- bactericides
- bleaches
- chemical dusts
- detergents, synthetic
- heat
- soaps

LINOLEUM MAKERS
- asphalt
- dyes
- oils
- pigments
- resins

LONGSHOREMEN
- *See* Dock workers

MACHINISTS
- chlorinated cutting oils
- chromates
- cutting fluids
- germicides
- lubricating oils
- rust inhibitors
- solvents

MASONS
- *See* Brick masons

MATCH FACTORY WORKERS
- ammonium phosphate
- chromates
- dextrins
- dyes
- formaldehyde
- glues
- gums
- phosphorus sesquisulfide
- potassium chlorate
- red phosphorus
- waxes

MEAT PACKERS
- *See* Butchers

MECHANICS
- *See* Aviation mechanics, and Garage workers

MERCERIZERS
- acids
- alkalis

METAL POLISHERS
- abrasives
- acids
- alkalis

METAL POLISHERS—continued
- ammonia
- naphtha
- pine oil
- potassium cyanide
- soluble oils
- soaps
- solvents
- triethanolamine
- waxes

MIRROR MAKERS
- ammonia
- cyanides
- formaldehyde
- lacquers
- silver nitrate
- solvents
- tartaric acid
- varnishes

MORDANTERS
- acids
- alkalis
- aluminum salts
- antimony compounds
- arsenates
- chromates
- copper salts
- iron salts
- lead salts
- phosphates
- silicates
- tin salts
- zinc chloride

NICKEL PLATERS
- detergents, synthetic
- heat
- moisture
- nickel sulfate
- zinc chloride

NITROGLYCERIN MAKERS
- ethylene glycol dinitrate
- nitric acid
- nitroglycerin
- sodium carbonate
- sulfuric acid

NURSES
- anesthetics, local
- antibiotics
- antiseptics
- bacteria
- detergents, synthetic

NURSES—continued
- disinfectants
- drugs
- fungi
- ionizing radiation
- moisture
- soaps
- tranquilizers
- viruses

OIL FIELD WORKERS
- alkalis
- brine
- crude petroleum
- ionizing radiation
- lubricating oils
- sunlight

OPTICAL WORKERS
- alkalis
- grinding fluids
- oils
- turpentine

PACKING-HOUSE WORKERS
- *See* Slaughter- and packing-house workers

PAINTERS
- acetone
- acids
- alkalis
- benzine
- chromates
- drying agents
- paint removers
- paints
- pigments
- resins
- solvents
- sunlight
- thinners
- turpentine

PAINT MAKERS
- anti-mildew agents
- chromates
- coal tar distillates
- drying agents
- fish oils
- latex
- oil, vegetable
- petroleum solvents
- pigments
- resins
- thinners

PAINT MAKERS—continued
- turpentine
- zinc chloride

PAPER BOX MAKERS
- dyes
- glues
- plastics
- resins
- waxes

PAPER MAKERS
- alkalis
- aluminum sulfate
- calcium bisulfite
- calcium chloride
- chromates
- glues
- heat
- moisture
- resins
- rosin
- sodium hydroxide
- sodium sulfate
- sodium sulfide
- sulfur dioxide

PARAFFIN WORKERS
- paraffin
- paraffin distillates
- solvents

PARCHMENT MAKERS
- zinc chloride

PENCIL MAKERS
- aniline dyes
- chromium pigments
- glues
- gums
- lacquer
- lacquer thinners
- methyl violet
- pyridine
- red cedar wood
- resins
- solvents
- waxes

PETROLEUM REFINERY WORKERS
- acids
- alkalis
- aluminum chloride
- arsenic
- gas oil
- gasoline
- hydrofluoric acid

PETROLEUM REFINERY WORKERS—con.
- kerosine
- paraffin
- paraffin distillates
- petroleum
- petroleum solvents
- tar
- waxes

PHOTOENGRAVERS
- ammonium bichromate
- etching acids
- inks
- photographic developers
- solvents
- ultraviolet light

PHOTOGRAPHERS
- acids
- alkalis
- chromates
- hydroquinone
- methyl-para-aminophenol sulfate
- para-aminophenol
- paraformaldehyde
- paraphenylenediamines
- photographic developers
- pyrogallic acid
- sodium hypochlorite
- sodium sulfide
- turpentine

PHYSICIANS
- anesthetics, local
- antibiotics
- antiseptics
- bacteria
- detergents, synthetic
- drugs
- fungi
- ionizing radiation
- rubber gloves
- soaps
- tranquilizers
- viruses

PITCH WORKERS
- heat
- pitch
- solvents
- sunlight
- tar

PLASTERERS
- lime
- moisture

PLASTIC AND RESIN MAKERS
- *See* Plastics and Synthetic Resins section

PLUMBERS
- cement
- cold
- hydrochloric acid
- parasites
- solvents
- tar
- zinc chloride

PRINTERS
- alkalis
- aniline
- chromates
- glues
- gum arabic
- inks
- solvents

RAILROAD SHOP WORKERS
- alkalis
- antiseptics
- chromates
- cutting fluids
- detergents, synthetic
- dichlorobenzene
- diesel fuel oil
- greases
- insecticides
- lacquers
- lubricating oils
- magnaflux
- paint
- paint strippers
- paint thinners
- solvents
- ultraviolet light

RAILROAD TRACK WORKERS
- cold
- creosote
- fungicides
- herbicides
- pitch
- poison ivy
- poison oak
- poison sumac
- ragweed
- sunlight
- tar

RAYON WORKERS
- acetic anhydride
- acids
- alkalis
- ammonium sulfide
- bleaches
- calcium bisulfite
- carbon disulfide
- coning oils
- sodium cyanide
- sodium sulfide
- sodium sulfite
- solvents

REFRIGERATION WORKERS
- ammonia
- brine
- cold
- dry ice
- ethyl bromide
- ethyl chloride
- glass fiber
- methyl chloride
- sulfur dioxide

ROAD WORKERS
- asphalt
- cement
- cold
- epoxy resins
- herbicides
- paint
- parasites
- pitch
- poison ivy
- poison oak
- poison sumac
- ragweed
- sunlight
- tar

ROCKET FUEL HANDLERS
- aniline
- boron hydrides
- chlorine trifluoride
- dimethylhydrazine
- ethyl oxide
- fuming nitric acid
- gasoline
- hydrazine
- hydrogen fluoride
- hydrogen peroxide
- kerosine
- liquid oxygen

218-695 O-66-4

ROPE MAKERS
- alkalis
- bleaches
- dusts
- dyes
- oils
- pitch
- soaps
- tar

RUBBER WORKERS
- accelerators
- acids
- activators
- adhesive removers
- alkalis
- antioxidants
- benzol
- chloroprene dimers
- chromium pigments
- curing agents
- formaldehyde
- heat
- oils
- plasticizers
- resins
- retarders
- soaps
- solvents
- tar
- turpentine
- zinc chloride

SHIPYARD WORKERS
- asbestos
- chlorinated diphenyls
- chlorinated naphthalenes
- chromates
- cold
- fungicides
- glass fiber
- paint removers
- paints
- paint thinners
- resins
- solvents
- tar
- ultraviolet light
- wood preservatives

SHOEMAKERS (MANUFACTURERS)
- adhesives
- ammonia
- amyl acetate
- amyl alcohol

SHOEMAKERS (MANUFACTURERS)—con.
- aniline dyes
- benzine
- benzol
- coal tar products
- methyl alcohol
- naphtha
- plastics
- rubber
- shoe polishes
- waxes

SILK PROCESSORS
- acids
- alkalis
- dyes
- xylene

SLAUGHTER- AND PACKING-HOUSE WORKERS
- antibiotics
- bacteria
- brine
- cold
- detergents, synthetic
- enzymes
- fungi
- parasites
- spices

SOAP MAKERS
- alkalis
- bacteriostats
- detergents, synthetic
- oil, vegetable
- perfumes
- sodium silicate

SOLDERERS
- acids
- cyanides
- fluxes
- heat
- hydrazine salts
- rosin
- zinc chloride

STEVEDORES
- *See* Dock workers

STOCKYARD WORKERS
- bacteria
- fungi
- insecticides
- parasites

STONE WORKERS
- cement

STONE WORKERS—continued
- dusts
- lime
- vibrating tools

SUGAR REFINERS
- acid
- burlap
- heat
- jute
- lime
- monilia
- sugar

TANNERY WORKERS
- acetic acid
- acids
- alum
- ammonium chloride
- arsenic salts
- bacteria
- benzol
- brine
- calcium hydrosulfide
- chromates
- dimethylamine
- dyes, mineral
- dyes, vegetable
- formaldehyde
- lime
- oils
- pancreatic extract
- sodium hydroxide
- sodium sulfide
- solvents
- tannin

TAR WORKERS
- heat
- pitch
- solvents
- sunlight
- tar

TAXIDERMISTS
- anthrax bacillus
- arsenic
- bacteria
- calcined alum
- fungi
- mercuric chloride
- parasites
- solvents
- tannin
- zinc chloride

TEMPERERS
- oils
- sodium carbonate
- sodium cyanide
- sodium dichromate
- sodium nitrite

TINNERS
- pitch
- sunlight
- zinc chloride

TOBACCO WORKERS
- dust, vegetable
- glues
- glycerine
- insecticides
- oil, vegetable

TYPISTS
- *See* Clerks

UNDERTAKERS
- *See* Embalmers

UPHOLSTERERS
- bacteria
- fungi
- glues
- lacquer
- lacquer solvents
- methyl alcohol
- parasites

VETERINARIANS
- anesthetics, local
- antibiotics
- bacteria
- carbon disulfide
- drugs
- fungi
- mercuric chloride
- parasites
- pesticides
- viruses

WATCHMAKERS
- acids
- chromates
- nickel salts
- potassium cyanide
- solvents

WATERPROOFERS
- alum
- Japan wax
- melamine formaldehyde resins
- oils
- paraffin
- pitch
- rubber
- solvents
- waxes

WELDERS
- fluxes
- heat
- ultraviolet light

WIRE DRAWERS
- drawing oils
- lime
- soaps
- sulfuric acid

WOOD PRESERVERS
- chlorophenols
- chromates
- copper compounds
- creosote
- cresols
- mercuric chloride
- phenyl mercuric compounds
- resins
- tar
- zinc chloride
- zinc sulfate

WOODWORKERS
- acid bleaches
- amino resin glues
- fillers
- formaldehyde
- glues
- lacquers
- mercuric chloride
- oil stains
- paints
- phenolic resin glues
- rosin
- solvents
- varnishes
- woods
- *See also* Cabinet makers and carpenters

References

COMMITTEE ON OCCUPATIONAL DERMATOSES, COUNCIL ON INDUSTRIAL HEALTH (D. J. BIRMINGHAM, CHAIRMAN): *Occupational Dermatoses.* American Medical Association, Chicago, 1959. A collection of five reprints from the Journal of the American Medical Association.

ROTHMAN, S.: *Physiology and Biochemistry of the Skin.* University of Chicago Press, Chicago, 1954.

SCHWARTZ, L.; TULIPAN, L., AND BIRMINGHAM, D. J.: *Occupational Diseases of the Skin.* 3d ed. Lea & Febiger, Philadelphia, 1957.

SUSKIND, R. R.: *Occupational skin problems.* 1, Mechanisms of dermatologic response. 2, Methods of evaluation for cutaneous hazards. *J. Occup. Med.* 1: 39 and 119, 1959.

• section V

PNEUMOCONIOSES

THOMAS H. MILBY, M.D.

The word pneumoconiosis literally means dust retained in the lung, with no implication as to whether disease is or is not present. In more common usage, however, the word has become a general term for any of the dust diseases of the lung and is used here with that meaning.

The development of a pneumoconiosis depends upon a number of factors related to the worker and to the dust. Worker factors include duration of exposure to the dust and susceptibility; factors related to the dust include its chemical composition, particle size, and concentration.

There have been suggested many different classifications for the pneumoconioses. They have been classified according to etiology, pathology, and physiology. The offending dusts themselves have been classified according to origin, and chemical, physical, and noxious properties. Notwithstanding the numerous efforts to systematize the information available concerning the pneumoconioses, no single classification has been widely accepted.

In this presentation, there has been neither an attempt to classify these dust diseases nor to discuss all of those described in the available medical literature. Included are only the pneumoconioses which are best understood, most commonly seen, and most widely accepted as definite clinical entities. These include:

(1) Silicosis
(2) Coal Workers' Pneumoconiosis
(3) Asbestosis
(4) Diatomite Pneumoconiosis
(5) Shaver's Disease
(6) Talcosis
(7) Pulmonary Siderosis
(8) Byssinosis
(9) Bagassosis
(10) Farmer's Lung

The following dusts and their effects when inhaled have not been included even though they have been reported as having caused pneumoconiosis: mica, kaolin, feldspar, cement, gypsum, fluorspar, sepiolite, sulfur, jute, moura seed, and grain.

Since the discussions that follow apply generally to dusts containing a single pathogenic substance, it should be recognized that where exposure occurs to dusts containing several harmful substances, the entire response may be markedly altered and result in the production of many bizarre findings.

References

DAVIES, C. N. (EDITOR): *Inhaled Particles and Vapors.* Permagon Press, London and New York, 1961.

FELSON, B.: *Fundamentals of Chest Roentgenology.* W. B. Saunders Co., Philadelphia, 1960.

GROSS, P.: Current concepts of pneumoconiosis. Pathological aspects. *J. Am. Med. Assoc.* 172: 546, 1960.

HEPPLESTON, A. G. AND LEOPOLD, J. G.: Chronic pulmonary emphysema. Anatomy and pathogenesis. *Am. J. Med.* 31: 279, 1961.

INTERNATIONAL LABOUR OFFICE: International classification of radiographs of the pneumoconioses. *Occup. Safety & Health* (extract) vol. 9, (April–June) 1959. 8 pp.

KING, E. J. AND FLETCHER, C. M. (EDITORS): *Industrial Pulmonary Diseases.* Little, Brown and Co., Boston, 1960.

LIEBEN, J.; PENDERGRASS, E., AND MCBRIDE, W. W.: Pneumoconiosis study in central Pennsylvania coal mines. 1, Medical phase. *J. Occup. Med.* 3: 493, 1961. Editorial: Let's take another look; p. 544.

NAGELSCHMIDT, G.: The relation between lung dust and lung pathology in pneumoconiosis. *Brit. J. Indust. Med.* 17: 247, 1960.

ORENSTEIN, A J. (EDITOR): *Proceedings, Pneumoconiosis Conference, Johannesburg, 1959.* Little, Brown and Co., Boston, 1960.

SANDER, O. A.: Current concepts of pneumoconioses. Clinical aspects. *J. Am. Med. Assoc.* 172: 1587, 1960.

WRIGHT, G. W.: Emphysema; terminology and classification. *A.M.A. Arch. Indust. Health* 13: 140, 1956.

(1) Silicosis

grinders' rot, miners' consumption, miners' phthisis, potters' asthma, stone masons' phthisis

Silicosis is a pneumoconiosis caused by the inhalation of finely divided silicon dioxide (silica) in the free state, which may be in a crystalline form such as in quartz, cristobalite and tridymite, or in a noncrystalline or amorphous form such as in opal. It has been shown that the crystal structure of pure silica has an important influence upon tissue reaction. Thus, in the production of a fibrous tissue response, tridymite is intensely fibrogenic, cristobalite and quartz, are somewhat less fibrogenic, and finally, amorphous silica is only slightly fibrogenic.

Silica in the nonfree or combined state, namely a silicate, refers to silica in chemical combination. Thus, the feldspars are aluminum silicates with potassium, sodium, calcium, or barium. Other silicates include kaolin, mica, serpentine, shale, slate, and talc. A pneumoconiosis associated with the inhalation of the dust of a silicate is termed a silicatosis.

Silica and silicates, composing almost entirely the crust of the earth, constitute the major portion of all rocks and their products such as soils, sands, and clays.

It is generally accepted that the size of the offending silica particle is of extreme importance in determining the degree of tissue reaction that will occur in the lung following the inhalation of siliceous dust. The size of the particle directly influences the concentration of particles that may be suspended in the air; it also determines the depth to which these particles penetrate into the lung and in what amounts they may become deposited and retained. While experimental silicosis has been produced with particles as large as 8 to 10 microns in diameter, it has been reported that the optimum size for alveolar retention of silica dust is about 1 micron. Recent evidence suggests, however, that particles below 1 micron in size may be the most dangerous since they penetrate deep into the alveolar spaces and are deposited there in very high concentrations. The lower limit of particle size which will produce a fibrogenic reaction is unknown, but may be close to 0.1 micron.

Silicosis may be either of an acute or of a chronic nature. The former is referred to as rapidly-developing silicosis rather than as acute silicosis.

The etiology, symptomatology, and pathology of rapidly-developing silicosis are not well understood. The disease has been most often reported in manufacturers and packers of abrasive soap powders, in sand-blasters working in enclosed tanks, and in high-power drillers of tunnel rock. It was suggested in 1939 at the Fourth Saranac Laboratory Symposium that one or more factors may have important etiologic significance. Such factors are exposure to very finely divided crystalline silica dust; exposure to massive amounts of free crystalline silica; synergistic action of other ions; differences in individual susceptibility; and presence of concomitant infection, especially tuberculosis.

The time of exposure to silica dust was relatively short in the reported cases of rapidly-developing silicosis, varying from 8 to 18 months from the time of the first exposure to the time of the onset of symptoms. After development of symptoms, the survival time is likely to be very short. The clinical picture of this type of silicosis is characterized by pulmonary insufficiency, with dyspnea, tachypnea, and cyanosis leading to the development of cor pulmonale. Many cases have been complicated by pulmonary tuberculosis. The chest roentgenogram in rapidly-developing silicosis shows diffuse fibrosis with no visible typical nodulation. Roentgenographic evidence of pulmonary tuberculosis is often present.

Chronic pulmonary silicosis, the type usually encountered in industry, is produced, as a rule, only after years of silica dust inhalation. The disease is reported to occur most commonly in the mining industries but is also seen in numerous other industries such as potteries, foundries, stone cutting and finishing, tile and clay producing, and glass manufacturing.

Although silicosis may be identified in a relatively early stage with the aid of a satisfactory chest roentgenogram, the uncomplicated disease may progress to an advanced stage while producing only symptoms of moderate dyspnea. The shortness of breath is noted first on moderate exertion, but as the disease progresses, the dyspnea occurs with less and less exertion.

Clinically, silicosis may follow one of several courses. The simple, uncomplicated form, frequently called simple discrete nodular silicosis, often does not progress beyond the stage where the nodules comprise a relatively small amount of the total lung tissue. This form of silicosis may present itself symptomatically only as a slowly increasing, non-disabling, exertional dyspnea. The chest roentgenogram usually reveals, in this form of the disease, uniformly distributed, discrete densities up to 10 mm in diameter. There is also very often seen enlargement of the shadows cast by the tracheobronchial lymph nodes.

In some silicotic patients, there is seen to develop in the upper portions of both lungs, large irregular masses of dense fibrous tissue. When these conglomerate masses appear on the chest roentgenogram, the disease may be categorized as conglomerate silicosis. In this form of the disease, the presence of advanced fibrosis and diffuse, obstructive emphysema may lead to severe respiratory crippling due to a decrease in the maximum breathing capacity and an increase in the residual lung volume. At this stage, the clinical symptoms, in addition to dyspnea on exertion, may include a productive cough, chest pain, and marked weakness. Cor pulmonale, probably caused by the increase in pressure required to force blood through a damaged pulmonary capillary bed, is a late and frequently fatal complication.

Tuberculosis is considered a common complication of silicosis. This combination is frequently manifested by the appearance of coalescent or conglomerate shadows on a chest roentgenogram which previously had demonstrated only shadows suggestive of simple discrete nodular silicosis.

References

CHAPMAN, E. M.: Acute silicosis. *J. Am. Med. Assoc.* 98: 1439, 1932.

DAUTREBANDE, L.; BECKMANN, H., AND WALKENHORST, W.: Lung deposition of fine dust particles. *A.M.A. Arch. Indust. Health* 16: 179, 1957.

DRINKER, P. AND HATCH, T. F.: *Industrial Dust; Hygienic Significance, Measurement and Control.* 2nd ed. McGraw-Hill Book Co., New York, 1954.

GARDNER, L. U.: Rapidly developing silicosis. *In* Kuechle, B. E. (editor): *Fourth Saranac Laboratory Symposium on Silicosis, 1939.* Employers Mutual Liability Insurance Co., Wausau, Wis., 1939.

GROSS, P.; WESTRICK, M. L., AND MCNERNEY, J. M.: Tuberculosilicosis, a study of its synergistic mechanisms. *J. Occup. Med.* 2: 571, 1960.

MOREY, G. W.: Silica and the silicate minerals. *In* Kirk, R. E. and Othmer, D. F. (editors): *Encyclopedia of Chemical Technology.* Vol. 12. Interscience Publishers, New York, 1954.

WELLS, A. F.: *Structural Inorganic Chemistry,* 2nd ed. Clarendon Press, Oxford, 1950. Ch. 19, Silicon. 3rd ed., 1962.

(2) Coal Workers' Pneumoconiosis

The dust to which a coal worker may be exposed is complex in nature. Besides the dust arising from the disintegration of coal, which itself is always intimately associated with other minerals, siliceous dusts of various types are derived from the rock strata above and below the coal seam. The relative importance of the coal and the mineral dust in the production of coal workers' pneumoconiosis is a question that is continually debated. At present, however, there is growing agreement as to the terminology, namely, coal workers' pneumoconiosis, applicable to the pathologic condition of the lung resulting from exposure to coal dust.

The term anthracosis refers to a blackish pigmentation of the lungs caused by deposition of carbon particles and may be observed with no evident pathologic change at autopsy. The condition is frequently observed in the lungs of city dwellers who have had no industrial exposure. Since no apparent disease or disability is associated with this deposition of pigment, anthracosis will not be considered further here.

The term anthracosilicosis generally means a modified form of classical silicosis resulting from prolonged exposure to coal dust and to rock dust containing significant amounts of free silica. The pathologic condition observed in this form of pulmonary disease is essentially the deposition of coal dust in the lungs accompanied by extensive fibrosis, both diffuse and nodular, with associated functional changes.

The term coal workers' pneumoconiosis, as described by a number of authors, is a different disease, however, since there is little or no evidence of classical silicosis or of significant silica exposure. The disease, apparently caused by coal dust itself, is an established entity and is pathologically distinct from silicosis. In the simple or uncomplicated form of coal workers' pneumoconiosis, the lungs contain large quantities of coal dust which is aggregated into foci surrounding the respiratory bronchioles, frequently causing them to dilate, a condition known as focal emphysema. The fibrosis produced is strikingly sparse, the coal dust being held in a fine mesh of reticulin fibrils, stellate in appearance, and contrasting markedly with the rounded, whorled nodule of silicosis.

The advanced or complicated form of coal workers' pneumoconiosis starts within a few coal foci as a collagen fibrosis and subsequently enlarges and coalesces to form a dense mass of fibrous tissue. This fibrosis may occupy much of a lobe or even a whole lung, and is thought usually to be due to tuberculosis superimposed upon a lung heavily laden with coal dust. Because of the nature of this condition, it is often referred to as progressive massive fibrosis (PMF) and carries with it the implications of a grave prognosis, death frequently resulting from tuberculosis or

pulmonary insufficiency or from cor pulmonale secondary to obliteration of the pulmonary vascular bed by fibrous tissue invasion.

The roentgenographic characteristics of simple coal workers' pneumoconiosis include discrete opacities up to 10 mm in diameter, which may be arranged in groups or spread diffusely throughout the lung fields.

In the complicated form of the disease, the earliest roentgenographic evidence of PMF is the presence, usually on a background of simple pneumoconiosis, of larger, less well defined opacities, often resembling reinfection-type tuberculosis in both position and appearance. These large shadows tend to increase in size and to coalesce. They later may contract with resultant severe distortion of the lung architecture.

References

COCHRANE, A. L.: The attack rate of progressive massive fibrosis. *Brit. J. Indust. Med.* 19: 52, 1962.

DOYLE, H. N.; FLINN, R. H., AND DREESSEN, W. C.: A review of the pneumoconiosis problem in the United States. *Am. Indust. Hyg. Assoc. J.* 19: 317, 1958.

FLETCHER, C. M.: Classification of roentgenograms in pneumoconiosis. *A.M.A. Arch. Indust. Health* 11: 17, 1955.

FLINN, R. H.; SEIFERT, H. E.; BRINTON, H. P.; JONES, J. L., AND FRANKS, R. W.: Soft coal miners health and working environment. Pub. Health Bull. No. 270. U.S. Government Printing Office, Washington, D.C., 1941.

GILSON, J. C.: Pathology, radiology, and epidemiology of coal workers' pneumoconiosis in Wales. *A.M.A. Arch. Indust. Health* 15: 468, 1957.

GILSON, J. C. AND HUGH-JONES, P.: Lung function in coal workers' pneumoconiosis. Medical Research Council, Special Report Series No. 290. Her Majesty's Stationery Office, London, 1955.

GOUGH, J.: Pneumoconiosis in coal workers in Wales. *Occup. Med.* 4: 86, 1947.

HEPPLESTON, A. G.: Coal workers' pneumoconiosis. Pathological and etiological considerations. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 270, 1951.

HOLT, P. F.: *Pneumoconiosis; Industrial Disease of the Lung Caused by Dust.* Edward Arnold, London, 1957.

KERR, L. E.: Coal workers' pneumoconiosis. *Indust. Med. & Surg.* 25: 355, 1956.

MARTIN, J. E.: Coal miners' pneumoconiosis. *Am. J. Pub. Health* 44: 581, 1954.

SAYERS, R. R.; BLOOMFIELD, J. J.; DALLAVALLE, J. M.; JONES, R. R.; DREESSEN, W. C.; BRUNDAGE, D. K., AND BRITTEN, R. H.: Anthraco-silicosis among hard coal miners. Pub. Health Bull. No. 221. U.S. Government Printing Office, Washington, D.C., 1936.

(3) Asbestosis

Asbestos is a general term used to describe several fibrous mineral silicates which differ in their chemical composition and physical properties The most important types of asbestos are chrysotile, a simple magnesium silicate; amosite and anthophyllite, which are complex magnesium iron silicates; and crocidolite, a complex sodium iron silicate. About 95 percent of the world's asbestos production is derived from chrysotile. Deposits of this mineral are found in many countries, but the largest mines are located in Canada.

Prolonged inhalation of asbestos fibers between 20 and 50 microns long may result in the production of a typical pulmonary fibrosis which may be accompanied by severe respiratory disability. On the basis of experimental studies of asbestosis, it was reported in 1951 that this fibrosis is due to the mechanical action of the asbestos fiber. The fibers, upon being deposited in the terminal bronchioles, initiate a tissue response which results in the coating of the fiber with the ultimate production of what is known as the asbestos or asbestosis body. This response appears to be a defense mechanism of the lung. If large quantities of the fibers are inhaled over a prolonged period of time, characteristically 10 to 20 years, the tissue reaction progresses until a generalized, diffuse fibrosis becomes evident. This fibrosis is seen first in the lower lobes of the lungs but eventually, if exposure continues, appears in the other lobes as well. Respiratory insufficiency and cardiac failure may supervene. It is of considerable interest and significance that asbestos fibers smaller than about 20 microns in length are thought to be incapable of initiating a fibrogenic response.

The roentgenogram of the chest with pulmonary fibrosis resulting from prolonged inhalation of asbestos fibers discloses a typical pattern. In the early or first stages of the disease, the shadows are fine, diffuse and homogeneous and appear characteristically at the base of both lungs. The typical nodular pattern of silicosis is not seen in asbestosis; rather, the affected lung fields present a ground glass appearance.

In moderately advanced or second-stage asbestosis, the infiltration is more in evidence but remains generally confined to the lower lobes. The heart borders may become indistinct or shaggy, a condition which has been referred to as porcupine heart.

In far advanced or third-stage asbestosis, the infiltrate can be seen throughout the middle and upper lung fields; however, the apices generally remain clear. There is almost complete obliteration of the cardiac outline, the domes of the diaphragm and the costophrenic sulci.

It should be emphasized that the chest roentgenogram cannot accurately be used to estimate the presence or extent of impaired pulmonary function or disability in lung diseases in general, and in asbestosis in particular, since many individuals with radiographic evidence of third-stage asbestosis have been able to carry on their usual work and live fairly comfortable lives for several years. On the other hand, definite disability due to asbestosis has rarely been reported in the absence of a typical radiographic pattern.

There is no typical clinical picture for asbestosis. The disease is insidious in its onset and is slowly progressive so long as inhalation of the fiber continues. There is a gradual increase in cough and expectoration, anorexia, and weight loss, all combined with slowly increasing dyspnea. Cyanosis and clubbing of the fingers are rare findings. When an acute pneumonitis

develops in the presence of established asbestosis with fibrosis, recovery is often delayed because healing is slow and relapses are frequent.

The primary functional abnormality in pulmonary asbestosis is one of impaired oxygen transfer across the alveolar membrane rather than impairment of ventilatory capacity. This condition is referred to as an alveolar-capillary block.

Conflicting opinions and differences in reports make it difficult to confirm or deny conclusively a causal relationship between asbestosis and cancer of the lung or extrapulmonary tissues. However, there is increasing evidence to suggest that such a relationship exists.

With regard to the relationship between asbestosis and tuberculosis, it is fairly well established that asbestosis does not predispose to the development of tuberculosis, nor does it aggravate an apparently healed lesion.

References

ANDERSON, J. AND CAMPAGNA, F. A.: Asbestosis and carcinoma of the lung. Case report and review of the literature. *Arch. Environ. Health* 1: 27, 1960.

BADOLLET, M. S.: Asbestos. *In* Kirk, R. E. and Othmer, D. F. (editors): *Encyclopedia of Chemical Technology.* Vol. 2. Interscience Publishers, New York, 1948.

DOLL, R.: Mortality from lung cancer in asbestos workers. *Brit. J. Indust. Med.* 12: 81, 1955.

HURWITZ, M.: Roentgenologic aspects of asbestosis. *Am. J. Roentgenol.* 85: 256, 1961.

ISSELBACHER, K. J.; KLAUS, H., AND HARDY, H.: Asbestosis and bronchogenic carcinoma. *Am. J. Med.* 15: 721, 1953.

KEAL, E. E.: Asbestosis and abdominal neoplasms. *Lancet* 2: 1211, 1960.

LEATHART, G. L.: Clinical, bronchographic, radiological and physiological observations in ten cases of asbestosis. *Brit. J. Indust. Med.* 17: 213, 1960.

PEARL, R. M.: *Rocks and Minerals.* Barnes & Noble, New York, 1956.

SMITH, K. W.: Pulmonary disability in asbestos workers. *A.M.A. Arch. Indust. Health* 12: 198, 1955.

VORWALD, A. J.; DURKAN, T. M., AND PRATT, P. C.: Experimental studies of asbestosis. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 3: 1, 1951.

WAGNER, J. C.; SLEGGS, C. A., AND MARCHAND, P.: Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. *Brit. J. Indust. Med.* 17: 260, 1960.

WRIGHT, G. W.: Functional abnormalities of industrial pulmonary fibrosis. *A.M.A. Arch. Indust. Health* 11: 196, 1955.

(4) Diatomite Pneumoconiosis

Diatomite, frequently designated diatomaceous earth, diatomaceous silica, or kieselguhr, is composed of the siliceous skeltons of microscopic, unicellular, aquatic plants known as diatoms. Because of its remarkable properties, this nonmetallic mineral has found many industrial uses, such as in filters, insulators, absorbents, and polishes.

Crude diatomite is essentially amorphous silica and contains less than 5 percent of quartz and only traces of cristobalite and tridymite; however, after being processed by high-temperature calcining, the cristobalite content

may be as high as 60 percent. The particle size of finished diatomite powder products is predominantly under 10 microns.

As with most pneumoconiosis-causing dusts, the longer the exposure to diatomite dust, the more is the chance of developing demonstrable lung changes; however, it has been shown that exposure to this dust for as little as 1 to 3 years may produce definite roentgenographic evidence of pneumoconiosis. In addition, the extent and severity of diatomite pneumoconiosis correlate with the cristobalite content of the dust involved.

Radiographic changes resulting from exposure to diatomite dust can roughly be divided into two groups: (1) changes of a linear-nodular type, and (2) changes resulting in the production of coalescent opacities, usually superimposed on definite linear-nodular changes.

In this type of pneumoconiosis, pulmonary signs and respiratory symptoms correlate poorly with roentgenographic changes, except where massive confluent lesions are present, in which case pulmonary disability may be extreme.

When tuberculosis is superimposed on diatomaceous earth pneumoconiosis, the infection often pursues a benign course until cavitation supervenes; then the course is frequently one of slow deterioration despite modern treatment including collapse procedures and chemotherapy.

References

COOPER, W. CLARK; CRALLEY, L. J.; CLARK, W. H.; HUBBARD, B. R.; HURLEY, D. J., AND SULLIVAN, R. R.: Pneumoconiosis in diatomite mining and processing. Pub. Health Service Pub. No. 601. U.S. Government Printing Office, Washington, D.C., 1958.

OECHSLI, W. R.; JACOBSON, G., AND BRODEUR, A. E.: Diatomite pneumoconiosis; roentgen classification characteristics and classification. *Am. J. Roentgenol.* 85: 263, 1961.

SMART, R. H. AND ANDERSON, W. M.: Pneumoconiosis due to diatomaceous earth. Clinical and X-ray aspects. *Indust. Med. & Surg.* 21: 509, 1952.

(5) Shaver's Disease

corundum fume fibrosis, bauxite fume fibrosis

As reported in the American literature, Shaver's disease is a pneumoconiosis of occupational origin, resulting from the inhalation of fume emitted by electric furnaces used in the production of corundum. This fume is rich in alumina and silica, both of which are in the free state and are largely amorphous in structure. The fume is further characterized by its small particle size, generally smaller than 0.5 micron and extending down to about 0.02 micron. Although the noxious agent or agents within this fume have not been identified, both silica fume and finely divided aluminum are thought to be capable of causing lung damage if inhaled in significant amounts.

In contrast to classical silicosis, Shaver's disease may develop in a remarkably short time, the period between first exposure and onset of symptoms being as brief as 24 months in some cases.

The most outstanding symptom of this disease is shortness of breath, usually mild in the early stages of illness but worsening as the disease progresses. Sudden attacks of extreme breathlessness are not uncommon and may indicate the occurrence of spontaneous pneumothorax, a condition seen with disturbing frequency among those afflicted with this disease. Additional signs and symptoms include cough productive of frothy white sputum, chest tightness and pain, weakness and fatigue.

The chest roentgenogram characteristically reveals bilateral granular haziness, widened mediastinum, heavy fibrotic strands, distortion and elevation of the diaphragm, and radiographic evidence suggestive of emphysematous bullae.

There is no evidence to suggest that this disease predisposes to pulmonary tuberculosis.

References

MITCHELL, J.; MANNING, G. B.; MOLYNEUX, M., AND LANE, R. E.: Pulmonary fibrosis in workers exposed to finely powdered aluminum. *Brit. J. Indust. Med.* 18: 10, 1961.

POLICARD, A. AND COLLET, A.: Toxic and fibrosing action of submicroscopic particles of amorphous silica. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 9: 361, 1954.

RIDDELL, A. R.: Pulmonary changes encountered in employees engaged in the manufacture of alumina abrasives. Pathologic aspects. *Occup. Med.* 5: 710, 1948.

SHAVER, C. G.: Pulmonary changes encountered in employees engaged in the manufacture of alumina abrasives. Clinical and roentgenologic aspects. *Occup. Med.* 5: 718, 1948.

SHAVER, C. G. AND RIDDELL, A. R.: Lung changes associated with the manufacture of alumina abrasives. *J. Indust. Hyg. & Toxicol.* 29: 145, 1947.

VORWALD, A. J. (EDITOR): *Pneumoconiosis; Beryllium, Bauxite Fumes, Compensation.* Sixth Saranac Laboratory Symposium, 1947. Part 6, Shaver's disease. Paul B. Hoeber, New York, 1950.

(6) Talcosis

Pure talc is a hydrated magnesium silicate, similar in chemical composition to asbestos. It is a flaky mineral, but also occurs in a fibrous state. When crushed, it forms a smooth bland powder which is used for a wide variety of purposes. The word talc, as used in industry, usually refers to a product which meets certain physical requirements rather than to a substance of definite chemical composition. Commercial talc varies markedly in its composition, and the mineral talc itself is usually only a minor component present in combination with other minerals such as dolomite, tremolite, magnetite, serpentine, mica, and anthophyllite. Varying amounts of free silica may also be present.

For practical purposes, then, the word talc as used here will refer to a mixture of minerals rather than to the specific mineral talc, which is hydrated magnesium silicate.

Numerous investigators have shown that prolonged inhalation of talc will result in the production of significant lung damage *even though there is little or no free silica present.* Histopathologic examination of lung sections usually reveals the presence of mild to moderate peribronchial and perivascular fibrosis with dilatation of many small bronchi and bronchioles. In more advanced cases the fibrosis may be extensive. Roentgenographic evidence of emphysematous bullae and fibrosis is usually demonstrable.

Some reports indicate that tremolite may be the main pathogenic agent in producing this characteristic talc lung lesion. The similarities between the histopathologic changes present in the talc lesion and those seen in the asbestos-produced lesion have been pointed out and are of considerable interest in view of the fact that tremolite is recognized as an asbestiform mineral.

A striking feature very frequently noted upon histologic examination of affected lung tissue sections is the presence of many brilliantly birefringent, needle-shaped particles in the areas of fibrosis. X-ray diffraction studies have indicated that these particles are talc. Another commonly reported finding is the presence of asbestos-like bodies embedded in the fibrous tissue. These structures have been most frequently seen in specimens of lungs which have been found to contain appreciable quantities of tremolite. They are less commonly seen in specimens which contain only small amounts of this mineral.

When the characteristic talc lesion is modified by significant amounts of free crystalline silica in the inhaled dust, the entire clinical, pathologic and roentgenographic picture may be greatly changed. There may be a greater tendency toward the formation of massive lesions, fibrosis may be more intense, and damage to the pulmonary vascular bed may be extreme. True classical silicotic nodules are uncommon in such cases.

Potential occupational exposures include cosmetic workers, paint makers, paper makers, pottery makers, rubber cable coaters, rubber tire makers, talc millers, talc miners, and talcum powder makers.

References

HOGUE, W. L., JR., AND MALLETTE, F. S.: A study of workers exposed to talc and other dusting compounds in the rubber industry. *J. Indust. Hyg. & Toxicol.* 31: 359, 1949.

KIPLING, M. D. AND BECH, A. O.: Talc pneumoconiosis. *Trans. Assoc. Indust. Med. Officers* 10: 85, 1960. Case histories include 4 lead plate casters (storage batteries), extruder operator (rubber tires), and mold duster and dies caster (both, aluminum products).

MESSITE, J.; REDDIN, G., AND KLEINFELD, M.: Pulmonary talcosis, a clinical and environmental study. *A.M.A. Arch. Indust. Health* 20: 408, 1959.

SCHEPERS, G. W. H. AND DURKAN, T. M.: The effects of inhaled talc-mining dust on the human lung. *A.M.A. Arch. Indust. Health* 12: 182, 1955.

SEELER, A. O.; GRYBOSKI, J. S., AND MACMAHON, H. E.: Talc pneumoconiosis. *A.M.A. Arch. Indust. Health* 19: 392, 1959.

(7) Pulmonary Siderosis

Siderosis is a benign pneumoconiosis resulting from the deposition of inert iron dust in the lung. In general there is neither fibrosis nor emphysema associated with this condition unless, as often occurs, there is concomitant exposure to silica dust. Siderosis does not result in the production of disability nor does it show any predisposition to pulmonary tuberculosis or lung cancer.

The chest roentgenogram in siderosis closely resembles the picture seen in uncomplicated silicosis. There may be, in both conditions, discrete nodular densities evenly distributed throughout the lung fields. In siderosis when considered by itself, there is no emphysema and there is very little tendency toward the formation of the conglomerate masses which are often seen in silicosis.

The differential diagnosis between siderosis and silicosis is difficult, especially since they may occur together. The diagnosis can usually be made, however, on the basis of medical and occupational histories, physical examination, chest roentgenograms, pulmonary function studies, and an appraisal of the work environment.

References

HAMLIN, L. E.: Differential diagnosis of siderosis and silicosis. *Indust. Med. & Surg.* 21: 1, 1952.

HAMLIN, L. E. AND WEBER, H. J.: Siderosis, a benign pneumoconiosis due to the inhalation of iron dust. 1, A clinical, roentgenological and industrial hygiene study of foundry cleaning room employees. *Indust. Med. & Surg.* 19: 151, 1950.

VORWALD, A. J.; PRATT, P. C.; DURKAN, T. M.; DELAHANT, A. B., AND BAILEY, D. A.: Siderosis, a benign pneumoconiosis due to the inhalation of iron dust. 2, An experimental study of the pulmonary reaction following inhalation of dust generated by foundry cleaning room operations. *Indust. Med. & Surg.* 19: 170, 1950.

(8) Byssinosis

Byssinosis occurs in individuals who have experienced prolonged exposure to heavy air concentrations of cotton dust. Flax dust has also been incriminated. The exact mode of action of the cotton dust is unknown, but one or more of the following factors may be important in the pathogenesis of the disease: (1) Toxic action of microorganisms adherent to the inhaled fibers, (2) mechanical irritation from the fibers, and (3) allergic stimulation by the inhaled cotton fibers or adherent materials. There is no good evidence to suggest that pathogenic invasion by microorganisms plays a significant role in the etiology of byssinosis.

The earliest manifestations of byssinosis may become noticeable after several years of exposure to cotton dust. The worker at first develops slight dyspnea and tightness of the chest on reporting to work on Monday morn-

ings or on days immediately following holidays or absences. He usually recovers completely by the next day. During this early phase of the disease permanent removal from exposure to cotton dust generally results in permanent cessation of symptoms.

If the worker continues to be exposed to the dust, he may go for years without noticing a worsening of his Monday morning symptoms. In some cases, however, continued exposure to cotton dust over many years is attended by a slowly progressing increase in both duration and severity of symptoms as well as by the onset of cough, frequently productive in nature. Even if further exposure to cotton dust is terminated, workers who have progressed to this phase of byssinosis may experience a permanent reduction in exercise tolerance.

In the most advanced stages of the disease, cough, chest tightness, and dyspnea may be so severe that the worker is forced to leave the cotton industry. Although some relief may be experienced when exposure to cotton dust ceases, chronic bronchitis and generalized, nonspecific pulmonary emphysema usually remain to cause permanent disability. Cor pulmonale may develop in severe cases.

The diagnosis of byssinosis is based on (1) a history of exposure to cotton dust over a period of years and (2) the occurrence of dyspnea and chest tightness which appear when the cotton worker reports to work on Monday morning or on days following holidays or other absences.

Differentiation between byssinosis and unassociated chronic bronchitis is based on observation that patients with chronic bronchitis may experience chest tightness when exposed to any excessively dusty atmosphere and the worker with early byssinosis is affected only by cotton dust and is worse on returning to work after several days of absence, typically on Monday.

Pulmonary function studies performed on workers exposed to cotton dust have been reported to show significant decreases in ventilatory capacity as measured by tests of air way resistance and indirect maximum breathing capacity. These studies have been especially revealing when performed at the beginning and again at the end of the same work day. Inhalation of cotton dust does not initiate a fibrogenic response. There is no characteristic pattern identifiable on the chest roentgenogram.

References

LOGAN, J. S.: Flax dust byssinosis and chronic non-tuberculous lung disease in Belfast. *Ulster Med. J.* 28: 164, (Nov. 1) 1959.

MAIR, A.; SMITH, D. H.; WILSON, W. A., AND LOCKHART, W.: Dust diseases in Dundee textile workers; an investigation into chronic respiratory disease in jute and flax industries. *Brit. J. Indust. Med.* 17: 272, 1960.

MCKERROW, C. B. AND SCHILLING, R. S. F.: A pilot enquiry into byssinosis in two cotton mills in the United States. *J. Am. Med. Assoc.* 177: 850, 1961.

218-695 O-66-5

MCKERROW, C. B.; MCDERMOTT, M.; GILSON, J. C. AND SCHILLING, R. S. F.: Respiratory function during the day in cotton workers: A study in byssinosis. *Brit. J. Indust. Med.* 15: 75, 1958.

PERNIS, B.; VIGLIANI, E. C.; CAVAGNA, C., AND FINULLI, M.: The role of bacterial endotoxins in occupational diseases caused by inhaling vegetable dusts. *Brit. J. Indust. Med.* 18: 120, 1961.

SCHILLING, R. S. F.: Byssinosis in cotton and other textile workers. *Lancet* 2: 261 and 319, 1956.

SCHILLING, R. S. F.; HUGHES, J. P. W.; DINGWALL-FORDYCE, I., AND GILSON, J. C.: An epidemiological study of byssinosis among Lancashire cotton workers. *Brit. J. Indust. Med.* 12: 217, 1955.

SHAPTINI, E. A.: Byssinosis, a review. *Indust. Med. & Surg.* 30: 95, 1961.

(9) Bagassosis

Bagasse is the fibrous material remaining after the sugar-containing juice has been expressed from sugar cane. It is used in insulating and acoustic materials as well as in the manufacture of paper, fertilizer, explosives, animal feed, and refractory brick.

Chemically, bagasse consists of approximately 4 percent ash and 2 percent protein with the remaining portion being made up of cellulose and other complex plant carbohydrates and resins.

Bagassosis is a lung disease produced by the inhalation of dust attendant with the opening of bagasse bales which have been stored for several months or years and have subsequently become very dry. Bagasse which is moist from recent grinding, or which has been stored in an unbaled condition, is apparently incapable of producing bagassosis.

Although the specific etiologic mechanism involved in bagassosis is unknown, the following possibilities have been suggested: (1) The disease constitutes an allergic reaction to microorganisms released when the bales are opened, (2) the disease is primarily an infectious process, (3) inhalation of the fibrous bagasse causes irritation of the pulmonary tissues and resultant pathologic changes, (4) irritant products other than the bagasse fibers are released and inhaled with the observed effects, or (5) an interaction of two or more of the above mechanisms.

Clinically, bagassosis presents itself as an acute pneumonitis or bronchiolitis. In most instances, after exposure to the dust for a few weeks to a few months, symptoms begin to appear over a space of several days. Cough, exertional dyspnea, and low grade fever are usually the initial complaints. Hemoptysis of a mild degree is rather common, but true pulmonary hemorrhage is rare.

As the disease progresses, dyspnea becomes more and more severe, and soon the patient becomes incapacitated. Cyanosis is present in severe cases. Weakness, anorexia, and weight loss are common complaints.

Physical examination may reveal dyspnea, cyanosis, and crepitant rales. Examinations of the sputum generally are negative for pathogenic microorganisms, including the tubercle bacillus.

Roentgenograms of the chest often disclose the presence of miliary shadows symmetrically distributed throughout both lungs, which appear very similar to the shadows seen in typical miliary tuberculosis. Patchy areas of increased densities suggesting bronchopneumonic infiltration are also seen. Lesions are usually more in evidence in the hilar areas and at the lung bases while the apices are often spared. The cardiac shadow may be enlarged and the pulmonary artery segment may be very prominent.

Pulmonary function studies indicate that there occurs in this disease a disturbance of ventilatory function as shown by a lowered vital capacity and a diminished maximum breathing capacity. These findings are compatible with the presence of airway obstruction such as might be produced by bronchiolitis. In addition, there has been some evidence to indicate the presence of a disturbance of gas exchange at the alveolar capillary level.

The great majority of patients suffering from bagassosis tend to improve spontaneously when they are removed from contact with the offending agent. Symptoms gradually abate over a period of several weeks, and recovery usually takes place in 1 to 6 months. However, some impairment of pulmonary function may be detected for longer periods, and the question of whether there occurs permanent functional lung damage has not yet been answered.

The diagnosis of bagassosis is based on the occupational history and the characteristic, but not pathognomonic, clinical and roentgenographic picture.

References

BAYONET, N. AND LAVERGNE, R.: Respiratory disease of bagasse workers. A clinical analysis of 69 cases. *Indust. Med. & Surg.* 29: 519, 1960.

BUECHNER, H. A.: Bagassosis. Peculiarities of its geographical pattern and report of the first case from Peru and Puerto Rico. *J. Am. Med. Assoc.* 174: 1237, 1960.

BUECHNER, H. A.; PREVATT, A. L.; THOMPSON, J., AND BLITZ, O.: Bagassosis. A review, with further historical data, studies of pulmonary function, and results of adrenal steroid therapy. *Am. J. Med.* 25: 234, 1958.

(10) Farmer's Lung

Farmer's lung is the name given to the disease entity produced by the inhalation of dust arising from moldy silage. Characteristically, symptoms of marked dyspnea, chills, fever, and cough occur several hours following the initial exposure to the dust. The onset of these symptoms may, however, be delayed for several weeks. In addition, auscultation of the chest usually reveals the presence of diffusely scattered, crepitant rales. Wheezes and

ronchi are often present but not invariably so. Dyspnea may be so extreme as to be associated with cyanosis. Weight loss may be pronounced.

Chest roentgenograms taken during this acute phase of the disease generally show changes which parallel the severity of symptoms. Fine to coarse nodular densities may be seen scattered diffusely throughout both lung fields. Conglomeration of these nodules is not an infrequent occurrence. If additional exposure to the dust does not occur, symptoms clear in one or two weeks and recovery is generally complete.

With repeated exposures, dyspnea and cough may become progressively more severe and, if exposures continue, irreversible lung changes may result. These changes, which include interstitial fibrosis and diffuse obstructive emphysema, may produce extreme pulmonary insufficiency with attendant incapacitation. Pulmonary function studies frequently reveal a significant increase in residual volume and functional residual capacity, as well as a moderate decrease in vital capacity and maximum breathing capacity.

The pathogenesis of farmer's lung remains obscure. While fungi seem to play an important etiologic role, the disease is apparently not a true pulmonary mycosis. Available evidence indicates that the pulmonary reaction is due either to one or both of the following: a mechanical irritation produced by the action of dust on lung tissue, or the production of a hypersensitive state by the molds or their disintegration products.

Histologic sections of involved lungs often reveal the presence of acute granulomatous interstitial pneumonitis and, in some cases, interstitial fibrosis, bronchiectasis and emphysema.

References

BALDUS, W. P. AND PETER, J. B.: Farmer's lung. Report of two cases. *New Eng. J. Med.* 262: 700, 1960.

FRANK, R. C.: Farmer's lung, a form of pneumoconiosis due to organic dusts. *Am. J. Roentgenol.* 79: 189, 1958.

FULLER, C. J.: Farmer's lung. *Dis. Chest* 42: 176, 1962.

JACKSON, D. AND YOW, E.: Pulmonary infiltration with eosinophilia; report of two cases of farmer's lung. *New Eng. J. Med.* 264: 1271, 1961.

TOTTEN, R. S.; REID, D. H. S.; DAVIS, H. D., AND MORAN, T. J.: Farmer's lung. Report of two cases in which lung biopsies were performed. *Am. J. Med.* 25: 803, 1958.

Some Threshold Limit Values

The threshold limit values carried by the accompanying table were again adopted in 1963 by the American Conference of Governmental Industrial Hygienists. The percent of crystalline silica appearing in the formula, the percent SiO_2 increased by 5 divided into 250, is derived from an analysis of the air-borne dust.

Substance	*Millions of particles per cubic foot of air*
SILICA:	
Crystalline:	
Quartz	*250/(percent SiO_2+5).*
Cristobalite	*Do.*
Amorphous, including natural diatomaceous earth	*20.*
*SILICATES:**	
Asbestos	*5.*
Mica	*20.*
Soapstone	*20.*
Talc	*20.*
*MISCELLANEOUS**	*50.*

**Less than 1 percent crystalline silica.*

• section VI

CHEMICAL HAZARDS

THOMAS H. MILBY, M.D.; MARCUS M. KEY, M.D.; ROY L. GIBSON, M.D., AND HERBERT E. STOKINGER, PH. D.

Introduction

Raw materials from oil, gas and salt wells, mines, forests, the sea, air, and farms are converted by the chemical industry into thousands of chemicals such as acids, alkalis, salts, and organic compounds. These chemicals are used by the industry itself to produce cosmetics, detergents and soaps, drugs, dyes, pigments, explosives, fertilizers, petrochemicals, inks, paints, pesticides, plastic materials, synthetic fibers, synthetic rubber, and many other products. Other industries use the chemicals in the production of durable and nondurable goods. *Durable goods* include aircraft and equipment, building materials, electric equipment, hardware, machinery, metal products, motor vehicles and equipment, and other products of metal, glass, paper, and wood. *Nondurable goods* include beverages, food products, leather and leather products, packaging, paper and paper products, petroleum and coal products, rubber products, and textiles.

This section deals with the harmful effects of various substances according to their capacity to produce local and systemic effects; special diagnostic tests that may aid diagnosis, and identification of the agent; the recommended threshold limit of safe exposure (8 hours, daily) when this has been established; potential occupational exposures; and references.

Harmful Effects

Under the heading, *Harmful Effects*, are given only the chief or dominant effects that characterize the usual response to the toxic agent. Because of the lack of information on the mutagenic effects of chemicals, no consideration is given in specific instances to these effects. Such damage results from the injury of the genetic material of the cells that the chemicals enter. Most of the evidence concerning such effects has been derived from experimental work on microorganisms, plants, and insects. Chemicals in this category are said to be radiomimetic in that they mimic biologic effects usually associated

with ionizing radiation. Among the chemicals used in industry, ethylene oxide and ethylene imine have been referred to as being radiomimetic.

Local and *Systemic* under *Harmful Effects* are included in an effort to categorize the effects of the toxic agent. It was arbitrarily decided to limit local effects to the skin, eyes, and mucous membranes of the upper respiratory tract. Systemic effects include the manifestations elicited by the absorption of the toxic agent into the body and its distribution to the internal organs. In addition, systemic effects include the effects of the agent on the tissues of the lower respiratory and gastrointestinal tracts.

Route of Entry, when applicable, is intended to supply information on the method by which the toxic agent is most likely to gain entrance into the body when encountered in the industrial environment. Thus, the oral route of entry is listed only for very toxic chemicals such as lead which may be conveyed to the mouth from the hands or cigarettes of the worker, or swallowed from contamination of the nasopharyngeal secretions.

Special Diagnostic Tests

Ordinary tests such as complete blood counts, routine urinalyses, and chest roentgenograms are not included under the heading, *Special Diagnostic Tests*. Similarly, liver and kidney function tests and cutaneous patch tests have not been included, even though they may be of considerable diagnostic importance. It is felt that the reader-physician need not be reminded of the methods for determining abnormalities in the target organs which are mentioned under *Systemis Effects*.

It should be pointed out that many of these special diagnostic tests are difficult to carry out and should be performed only by qualified laboratories. In addition, the fact should be kept in mind that *normal* values may vary, somewhat, even from competent analytical laboratories.

Because of the absence of significant, interpretable information, no reference is made to behavioral patterns of response to toxic agents.

Recommended Threshold Limit

A great deal more is implied in the heading *Recommended Threshold Limit* than the specific assigned Threshold Limit Value appearing in the tentative list published annually by the American Conference of Governmental Industrial Hygienists. In their use, threshold limits are to be considered practical guides in the control of health hazards and should not be regarded as fine lines between safe and dangerous exposure levels. The threshold limit represents a level of exposure at which it is believed, on best available information, almost all workers may be repeatedly exposed day after day throughout their working lifetime without adverse effect on health, or without significant discomfort. In the establishment of threshold limit values, increasing attention is being given to possible long-term genetic

effects, carcinogenic potential, and to the capacity of chemical agents to produce allergic sensitization.

It should be observed that the definition and application of the threshold limit values depend upon the toxicoligic action of the substances. Three categories are recognized in the currently (1963) listed substances: (1) Substances whose primary action is rapid, such as irritants; for these substances the limiting value represents a *ceiling*, a limit not to be exceeded for any period however short. Listed substances in this category are preceded by a "C." (2) Substances whose action is prolonged or cumulative; for these substances the threshold value refers to a time-weighted concentration averaged throughout an 8-hour day. Substances in this category comprise the bulk of the list. (3) The third group of substances comprises a small number, mainly carcinogens, for which at present no contact by any route is to be permitted.

It should be observed that when the threshold limit value refers to the time-weighted concentration averaged throughout an 8-hour day, limited fluctuation is permitted *above* the specified value, provided at least an equivalent fluctuation *below* the value obtains. Excessive fluctuations above the value are to be considered indicative of the existence of a hazardous situation, and proper steps should be taken for its control.

It is most important to note that use of the threshold limits to make comparisons between the toxicity or hazard of two substances is improper. The reason for this is that the factors involved in the choice of a limit for one substance may differ from those used in setting the limit for another. For example, the threshold limit for one substance may be based on comfort while the value for another may be based on acute systemic toxicity. Another limit may have incorporated in it a substantial safety factor because of the highly injurious nature of the agent; another limit may have a relatively small safety factor when it is known that a substance can rarely be lethal. Thus, use of the threshold limits to make comparative toxicity or hazard ratings among toxic agents results in erroneous conclusions.

Similarly, it is incorrect to use the threshold limits either unmodified, or modified by the application of a factor, for community air pollution levels. The threshold limits apply to an 8-hour daily workweek of five days and to reasonably healthy adults; they are not applicable to continuous exposures of young and old, the indisposed, and the diseased.

Potential Occupational Exposures

The list of occupations appended to a particular chemical carries occupations in which the workers so engaged are potentially exposed to the toxic agent. Whether the exposure to the toxic agent constitutes a hazard depends upon such factors as concentration of the agent, how the agent is handled and used, duration of exposure, susceptibility of the worker to the agent,

and the health protection practices that might have been adopted by management. Thus, all hazardous situations imply an exposure, but all exposures are not hazardous.

Symptoms and exposure—When the problem is encountered of the relationship between the signs and symptoms presented by the worker and the potential toxic exposures in his occupation, the investigator armed with the knowledge of the major ways by which a toxic chemical enters the body, secures factual information on the physical and chemical characteristics of the work environment and the personal hygiene of the worker. At the same time, it is essential to recognize that (1) chemical formulas offer, at most, only rough guides to the prediction of toxic response and (2) the forms of acute and chronic toxicity are so often dissimilar that prediction cannot be made of the nature of chronic toxicity from acute manifestations.

The ordering of the various environmental and clinical observations into a logical causal chain involves all of the difficulties usually inherent in the determination of the cause and effect relationship. The investigation must be carefully and thoughtfully performed. Particular attention must be given all elements believed relevant, the listing of which can flow only from the experience and training of the investigator, a review of the pertinent literature, and, when indicated, consultation with others who may have experienced similar situations. Especially difficult are the separation of the occupational exposures from the nonoccupational ones and any retrospective study required by a long period of latency between exposure to the toxic agent and the recognition of the disease.

References to the Literature

Specific references to the literature will be found appended to most of the chemicals. A list of general references appears at the end of the section.

Organization and Selection of Chemicals

The various chemicals are alphabetically arranged. In a number of instances related substances are presented together, for example, carbonyls, cycloparaffins, and nitroparaffins. Pesticides appear in a separate section. Although plastics and synthetic resins also comprise a single section, most of the components that produce systemic effects are included in the list of chemical hazards.

Most of the known disease-producing chemicals are given. Moreover many materials, even though innocuous, are presented because the question of toxicity may arise during the course of handling or working with these materials. Other compounds, though controversial from the standpoint of toxicity, are included for this reason. Certain chemicals are excluded because of insufficient data.

References

ALEXANDER, P.: Radiation-imitating chemicals. *Sc. American* 202: 99, (January) 1960.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS: *Documentation of threshold limit values.* The Conference, 1014 Broadway, Cincinnati 2, Ohio, 1962.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS: *Threshold limit values for 1963.* Adopted at the 25th annual meeting of the Conference, Cincinnati, Ohio, May 6–10, 1963. The Conference, 1014 Broadway, Cincinnati 2, Ohio, 1963.

BROWNING, E.: An evaluation of the methods of assessment of the toxicity of chemical compounds. *Trans. Assoc. Indust. Med. Officers* 8: 138, 1959.

ECKARDT, R. E.: Clinical toxicology and the practicing physician. *J. Am. Med. Assoc.* 166: 1949, 1958.

EDSON, E. F. AND NOAKES, D. N.: The comparative toxicity of six organophosphorus insecticides in the rat. *Toxicol. Appl. Pharmacol.* 2: 523, 1960.

ELKINS, H. B.: Maximum acceptable concentrations. A comparison in Russia and the United States. 13 references. *Arch. Environ. Health* 2: 45, 1961.

FIELDNER, A. C.; KATZ, S. H., AND KINNEY, S. P.: Gas masks for gases in fighting fires. Bureau of Mines, Department of the Interior, Technical Paper 248. U.S. Government Printing Office, Washington, D.C., 1921. Appendix carries a "conversion table for gases: parts per million versus milligrams per liter" and examples explaining its use. The table has been reproduced in other occupational health texts.

HARRIS, H.: *Human Biochemical Genetics.* Cambridge University Press, London, 1959.

HUEPER, W. C.: Medicolegal considerations of occupational cancers. In *Lawyers' Medical Cyclopedia of Personal Injuries and Allied Specialties.* Vol. 5. Allen Smith Co., Indianpolis, 1960. Presents among other things, occupational groups exposed to various cancer hazards.

JOHNSTONE, R. T.: Importance of epidemiology in medicolegal controversy. *In* Workmen's compensation problems. *Proceedings, 47th annual convention, International Association of Industrial Accident Boards and Commissions, 1961.* Bureau of Labor Standards, U.S. Department of Labor, Bull. No. 242. U.S. Government Printing Office, Washington, D.C., 1962.

MANUFACTURING CHEMISTS' ASSOCIATION: *The Chemical Industry Facts Book.* 5th ed. The Association, Washington, D.C., 1962.

STOKINGER, H. E.: Standards for safeguarding the health of the industrial worker. *Pub. Health Rep.* 70: 1, 1955.

WALD, N.: Cytogenetics; a new clinical tool with potential environmental health applications. *Arch. Environ. Health* 5: 217, 1962.

WARSHAW, L. J.: Cardiovascular effects of toxic occupational exposures. *In* Warshaw, L. J. (editor): *The Heart in Industry.* Paul B. Hoeber, New York, 1960.

WETHERHOLD, J. M.; LINCH, A. L., AND CHARSHA, R. C.: Chemical cyanosis; causes, effects, and prevention. *Arch. Environ. Health* 1: 353, 1960.

The chemicals follow.

(1) Acetaldehyde

acetic aldehyde, aldehyde, ethanal, ethyl aldehyde

Harmful Effects

Local Liquid and concentrated vapor are irritating to eyes, skin, and mucous membranes of upper respiratory tract. Contact with liquid can sensitize skin.

Route of Entry Inhalation of vapor.

Systemic Acute effects are secondary to narcotic action and pulmonary irritation, and include drowsiness, unconsciousness, bronchitis, and pulmonary edema. Chronic poisoning has not been reported from inhalation of vapor.

Special Diagnostic Test

Determination of acetaldehyde in blood or urine. *See* Von Oettingen, 1958.

Recommended Threshold Limit

200 parts per million parts of air by volume or 360 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetaldehyde workers
Acetic acid makers
Acetic anhydride makers
Acrolein makers
Aldehyde pumpmen
Aldol makers
Butanol makers
Chloral makers
Disinfectant makers
Drug makers
Dye makers
2-Ethylhexanol makers
Explosive workers
Flavoring makers
Lacquer workers
2-Methyl-5-ethyl pyridine makers
Mirror silverers
Paraldehyde makers
Pentaerythritol makers
Perfume makers
Phenolic resin makers
Photographic chemical makers
Resin makers
Rubber makers
Urea resin makers
Varnishers
Varnish makers
Vinegar makers
Yeast makers

References

HENSON, E. V.: The toxicology of some aliphatic aldehydes. *J. Occup. Med.* 1: 457, 1959.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(2) Acetic Acid

ethanoic acid, pyroligneous acid

Vinegar contains 4–6 percent of acetic acid; commercial acetic acid varies in concentration from 6 to 99 percent. Glacial acetic acid contains about 99 percent of the acid.

Harmful Effects

Local High concentrations of acetic acid vapor produce conjunctivitis, lacrimation, nasal irritation, and dental erosion. On contact, glacial

acetic acid produces painful cutaneous burns which are slow to heal, corneal burns, conjunctivitis, and iritis. Repeated contact with dilute solutions can produce a hyperkeratotic and fissured dermatitis from primary irritation.

Route of Entry Inhalation of vapor.

Systemic Irritant effect of high vapor concentrations, if unheeded, can produce bronchitis and pulmonary edema.

Special Diagnostic Test

None.

Recommended Threshold Limit

10 parts per million parts of air by volume or 25 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetamide makers
Acetanilide makers
Acetate ester makers
Acetate fiber makers
Acetic acid workers
Acetic anhydride makers
Acetone makers
Acetyl chloride makers
Aspirin makers
Cellulose acetate makers
Drug makers
Dye makers
Ester makers
Ethyl alcohol makers
Food preservers
Insecticide makers
Ketene makers
Laundry workers
Methyl ethyl ketone makers
Paris green makers
Photographic chemical makers
Plastic makers
Rubber makers
Stain removers
Textile printers
Tint rinse makers
Vinegar makers
White lead makers

References

HENSON, E. V.: Toxicology of the fatty acids. *J. Occup. Med.* 1: 339, 1959.

VON OETTINGEN, W. F.: The aliphatic acids and their esters: toxicity and potential dangers; the saturated monobasic aliphatic acids and their esters. *A.M.A. Arch. Indust. Health* 21: 28, 1960.

(3) Acetic Anhydride

acetic oxide, acetyl oxide, ethanoic anhydride

Harmful Effects

Local Exposure to liquid or concentrated vapor can produce conjunctivitis, photophobia, lacrimation, irritation of nose and throat, and contact dermatitis due to primary irritation.

Route of Entry Inhalation of vapor.

Systemic Pulmonary irritation can occur but is usually avoided by heeding early warning symptoms resulting from irritation of upper respiratory tract. No chronic systemic effects have been reported.

Special Diagnostic Test

None.

Recommended Threshold Limit

5 parts per million parts of air by volume or 20 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetanilide makers
Acetate fiber makers
Acetic acid makers
Acetic anhydride workers
Acetyl chloride makers
Aspirin makers
Cellulose acetate fiber makers
Drug makers
Dye makers
Explosive makers
Flavoring makers
Peracetic acid makers
Perfume makers
Photographic film makers
Plastic makers
Textile makers

(4) Acetone. *See* Ketones

(5) Acetonitrile

methyl cyanide, cyanomethane, ethanenitrile

Harmful Effects

Local Contact dermatitis due to primary irritation of either liquid or concentrated vapor.

Route of Entry Inhalation of vapor.

Systemic Hydrolyzes to cyanide which is detoxified to thiocyanate. Late symptoms may be due to thiocyanate toxicity. Inhalation of high concentrations can produce headache, weakness, shortness of breath, nausea, diarrhea, chest and abdominal pain, gray color, bleeding from mucous membranes, convulsions, coma, and death. Liver and kidney damage may also occur.

Special Diagnostic Test

Determination of blood cyanide, serum and urinary thiocyanate. *See* Amdur, 1959.

Recommended Threshold Limit

40 parts per million parts of air by volume or 70 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetonitrile workers
Animal oil processors
Drug makers
Fiber makers
Organic chemical synthesizers
Perfume makers
Petroleum hydrocarbon purifiers
Tank coaters
Thiamine makers
Vegetable oil processors

References

AMDUR, M. L.: Accidental group exposure to acetonitrile; a clinical study. *J. Occup. Med.* 1: 627, 1959.

RIEDERS, F. AND BRIEGER, H.; LEWIS, C. E., AND AMDUR, M. L.: What is the mechanism of toxic action of organic cyanide? *J. Occup. Med.* 3: 482, 1961. Three answers to the question.

(6) Acetylene

ethine, ethyne, narcylene

Harmful Effects

Local None.

Route of Entry Inhalation of gas.

Systemic In low concentrations, acetylene acts as narcotic. In high concentrations, it decreases available oxygen, thus causing anoxia. Impurities in commercial acetylene, such as arsine, hydrogen sulfide, phosphine, carbon disulfide, and carbon monoxide, may also produce symptoms.

Initial symptoms are rapid respiration and air hunger. Mental alertness and muscular coordination are impaired. Other manifestations include cyanosis, weak and irregular pulse, nausea, vomiting, prostration, impairment of judgment and sensation, loss of consciousness, convulsions, and death.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Acetaldehyde makers
Acetic acid makers
Acetone makers
Acetylene black makers
Acetylene workers
Acrylonitrile makers
Alcohol makers
Braziers
Butadiene makers
Carbon black makers
Ceramic makers
Chloro- derivative makers
Copper purifiers
Descalers
Drug makers
Dye makers
Foundry workers
Gougers

Hardeners
Heat treaters
Lead burners
Metalizers
Metal refiners
Motor boat fuel makers
Organic chemical synthesizers
Oxyacetylene cutters
Oxyacetylene solderers
Oxyacetylene welders
Rubber makers
Scarfers
Tetrachloroethane makers
Vinyl derivative makers

Reference

JONES, A. T.: Fatal gassing in an acetylene manufacturing plant. *Arch. Environ. Health* 1: 417, 1960.

(7) Acridine

dibenzopyridine, 10-azaänthracene

Harmful Effects

Local Either solid or vapor can produce contact dermatitis from primary irritation. Photosensitization dermatitis, conjunctivitis, corneal damage, and sneezing have been reported.

Route of Entry Inhalation of vapor.

Systemic No serious industrial poisonings have been reported. This is probably due to early warning by intense irritation of nose and throat.

Special Diagnostic Test

Determination of acridine in blood and urine. *See* Von Oettingen, 1958.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Acridine makers
Acridine workers
Acriflavine makers
Asphalt workers
Coal tar workers
Coke makers
Disinfectant makers
Drug makers
Dye makers
Highway maintenance workers
Laboratory workers, chemical
Lacrimator makers
Methionine makers
Organic chemical synthesizers
Pipeline workers
Pitch workers
Proflavine makers
Quinacrine makers
Railroad track workers
Rim steel makers
Road builders
Roofers
Stack cleaners
Street repairers
Wood preservers

References

BALDI, G.: Occupational pathology from acridine. *Med. Lavoro* 44: 240, 1953.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(8) Acrolein

acraldehyde, acrylic aldehyde, allyl aldehyde, propenal

Harmful Effects

Local Either liquid or concentrated vapor produces intense irritation of eyes, nose, throat, and skin.

Route of Entry Inhalation of vapor.

Systemic Pulmonary edema and narcosis are possible but seldom occur because of warning properties of vapor.

Special Diagnostic Test

None.

Recommended Threshold Limit

0.1 part per million parts of air by volume or 0.25 milligram per cubic meter of air.

Potential Occupational Exposures

Acrolein workers
Alcohol denaturant workers
Allyl alcohol makers
Bookbinders
Coffee roasters
Cooks
Core makers
Diesel engine workers
Drug makers
Drying oil workers
Fat processors
Foundry workers
Galvanizers
Glycerine workers
Heat treaters
Lacrimator makers
Linoleum makers
Linseed oil workers
Methionine makers
Organic chemical synthesizers
Perfume makers
Renderers
Resin makers
Rubber makers
Soap makers
Textile resin makers
Tinsmiths
Tung oil workers
Varnish makers

Reference

HENSON, E. V.: The toxicology of some aliphatic aldehydes. *J. Occup. Med.* 1: 457, 1959.

218-695 O-66-6

(9) Acrylonitrile

vinyl cyanide, cyanoethylene, propene nitrile

Harmful Effects

Local Liquid and high concentrations of vapor are irritating to eyes and nose. Contact with the liquid is irritating to skin and results in blister formation.

Routes of Entry Inhalation of vapor and percutaneous absorption of liquid. May be absorbed from contaminated rubber.

Systemic In addition to a toxic action from the whole molecule, acrylonitrile may also exert a toxic action by partial in vivo conversion to cyanide. Toxic effects include headache, nausea, weakness, diarrhea, anemia, and jaundice.

Special Diagnostic Tests

Examination of serum and urine for thiocyanate. Spectrographic determination of acrylonitrile in blood. *See* Lawton et al., 1943; Wilson and McCormick, 1949; Brieger et al., 1952, and Elkins, 1959.

Recommended Threshold Limit

20 parts per million parts of air by volume or 45 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Acrylic resin makers
Acrylonitrile workers
Fumigant workers
Grain fumigators
Leather finish makers
Lubricating oil additive makers
Lucite makers
Organic chemical synthesizers
Paper makers
Plasticizer makers
Plexiglass makers
Polymethacrylate resin makers
Rubber makers
Safety glass makers
Synthetic fiber makers
Textile finish makers

References

BRIEGER, H.; RIEDERS, F., AND HODES, W. A.: Acrylonitrile; spectrophotometric determination, acute toxicity and mechanism of action. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 128, 1952.

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

LAWTON, A. H.; SWEENEY, T. R., AND DUDLEY, H. C.: Toxicology of acrylonitrile (vinyl cyanide). 3, Determination of thiocyanates in blood and urine. *J. Indust. Hyg. & Toxicol.* 25: 13, 1943.

WILSON, R. H. AND MCCORMICK, W. E.: Acrylonitrile, its physiology and toxicology. *Indust. Med.* 18: 243, 1949.

(10) Allyl Alcohol

vinyl carbinol, propenyl alcohol

Harmful Effects

Local Liquid and vapor are highly irritating to eyes and upper respiratory tract. Effects include lacrimation, photophobia, retrobulbar pain, blurring of vision, corneal ulceration, coryza, and headache. Eye irritation is usually delayed in onset and may be prolonged. Skin irritation and burns have occurred from contact with liquid and may also be prolonged.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Local muscle spasms occur at sites of percutaneous absorption. Pulmonary edema, liver and kidney damage, diarrhea, delirium, convulsions, and death have been observed in laboratory animals but have not been reported in man. Vapor has strong warning properties.

Special Diagnostic Test

None.

Recommended Threshold Limit

2 parts per million parts of air by volume or 5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Acrolein makers
Allyl alcohol workers
Drug makers
Glycerine makers
Herbicide makers
Organic chemical synthesizers
Plasticizer makers
Resin makers

Reference

DUNLAP, M. K.; KODAMA, J. K.; WELLINGTON, J. S.; ANDERSON, H. H., AND HINE, C. H.: The toxicity of allyl alcohol. 1, Acute and chronic toxicity. *A.M.A. Arch. Indust. Health* 18: 303, 1958.

(11) Aluminum and Compounds

Harmful Effects

Local Aluminum salts are astringent. Contact may harden and tan skin resulting in fissuring. Aluminum chloride may act as sensitizer and produce contact dermatitis.

Route of Entry Inhalation of dust or fume.

Systemic Pathogenicity of inhaled aluminum dust or fume is controversial. It is likely that effect on lungs is intimately associated with particle size and purity of material involved. *See* Shaver's Disease, Pneumoconioses section.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Alumina (aluminum oxide) abrasive makers
Aluminum alloy grinders
Aluminum extractors
Aluminum workers
Ammunition makers
Ceramic makers
Cosmetic workers
Dye makers
Electronic workers
Fireworks makers
Foundry workers
Gem makers
Glass makers
Ink makers
Laboratory workers, chemical
Lithographers
Lubricant makers
Paint makers
Paper makers
Petroleum refinery workers
Plastic makers
Pottery makers
Rubber makers
Tannery workers
Textile workers

References

DWORSKI, M.: Prophylaxis and treatment of experimental silicosis by means of aluminum. *A.M.A. Arch. Indust. Health* 12: 229, 1955.

MCLAUGHLIN, A. I. G.; KAZANTZIS, G.; KING, E.; TEARE, D.; PORTER, R. J., AND OWEN, R.: Pulmonary fibrosis and encephalopathy associated with the inhalation of aluminum dust. *Brit. J. Indust. Med.* 19: 253, 1962.

MITCHELL, J.; MANNING, G. B.; MOLYNEUX, M., AND LANE, R. E.: Pulmonary fibrosis in workers exposed to finely powdered aluminum. *Brit. J. Indust. Med.* 18: 10, 1961.

RIDDELL, A. R.: Pulmonary changes encountered in employees engaged in the manufacture of alumina abrasives. Pathologic aspects. *Occup. Med.* 5: 710, 1948.

SHAVER, C. G.: Pulmonary changes encountered in employees engaged in the manufacture of alumina abrasives. Clinical and roentgenologic aspects. *Occup. Med.* 5: 718, 1948.

VORWALD, A. J. (EDITOR): *Pneumoconiosis; Beryllium, Bauxite Fumes, Compensation.* Sixth Saranac Laboratory Symposium, 1947. Paul B. Hoeber, New York, 1950.

(12) Ammonia

Harmful Effects

Local Contact with anhydrous liquid ammonia or with aqueous solutions is intensely irritating to mucous membranes, eyes, and skin. Eye symptoms range from lacrimation, blepharospasm, and palpebral edema to corneal ulceration and blindness. There may be corrosive burns of skin or blister formation. Ammonia gas is also irritating to eyes and moist skin.

Route of Entry Inhalation of gas.

Systemic Mild to moderate exposure to gas can produce headache, salivation, burning of throat, anosmia, perspiration, nausea, vomiting, and substernal pain. Irritation of ammonia gas in eyes and nose is sufficiently intense to compel workers to flee. If escape is not possible, there is irritation of lower respiratory tract with production of cough, glottal edema, pulmonary edema, or respiratory arrest. Bronchitis or pneumonia may follow a severe exposure if patient survives. Urticaria is a rare allergic manifestation from inhalation of gas.

Special Diagnostic Test

None.

Recommended Threshold Limit

50 parts per million parts of air by volume or 35 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetylene workers
Aluminum workers
Amine makers
Ammonia workers
Ammonium salt makers
Aniline makers
Annealers
Boneblack makers
Braziers
Bronzers
Calcium carbide makers
Case hardeners
Coal tar workers
Coke makers
Color makers
Corn growers
Cyanide makers
Decorators
Diazotypy machine operators
Drug makers
Dye intermediate makers
Dye makers
Electroplaters
Electrotypers
Explosive makers
Farmers
Fertilizer workers
Galvanizers
Gas purifiers
Gas workers, illuminating
Glass cleaners
Glue makers
Ice cream makers
Ice makers
Ink makers
Laboratory workers, chemical
Lacquer makers
Latex workers
Manure handlers
Metal extractors
Metal powder processors
Mirror silverers
Nitric acid makers
Organic chemical synthesizers
Paper makers
Perfume makers
Pesticide makers
Petroleum refinery workers
Photoengravers
Photographic film makers
Plastic cement mixers
Pulp makers
Rayon makers
Refrigeration workers
Resin makers
Rocket fuel makers

Rubber cement mixers
Rubber workers
Salt extractors, coke oven byproduct
Sewer workers
Shellac makers
Shoe finishers
Soda ash makers
Solvay process workers
Stablemen
Steel makers
Sugar refiners
Sulfuric acid workers
Synthetic fiber makers
Tanners
Tannery workers
Urea makers
Varnish makers
Vulcanizers
Water base paint workers
Water treaters
Wool scourers

(13) Amyl Acetate

isoamyl acetate, pear oil, banana oil, amyl acetic ester

Harmful Effects

Local Vapor is irritating to eyes and respiratory tract, and has produced laryngitis and glottal edema. Prolonged contact with liquid produced dry, scaly, and fissured dermatitis.

Route of Entry Inhalation of vapor.

Systemic Vapor has a narcotic action, and prolonged inhalation can produce fatigue, headache, vertigo, tinnitus, mental confusion, and somnolence. Overexposure is usually prevented by irritant warning property.

Special Diagnostic Test

None.

Recommended Threshold Limit

100 parts per million parts of air by volume or 525 milligrams per cubic meter of air.

Potential Occupational Exposures

Airplane dope makers
Amyl acetate workers
Art glass workers
Bath sponge makers
Battery makers, storage
Bookbinders
Bronzers
Bronzing liquid makers
Camphor workers
Cutlery makers
Dry cleaners
Dyers
Enamelers
Enamel makers
Explosive workers
Fruit essence makers
Furniture polishers
Gilders
Hefner lamp users
Incandescent lamp makers

Incandescent lamp wirers
Jewelers
Lacquer removers
Lacquer workers
Leather makers
Leather mottlers
Linoleum makers
Motion picture film workers
Nitrocellulose workers
Painters
Paint makers
Paint removers
Paper makers, coated
Patent leather makers
Pearl makers
Penicillin makers
Perfume makers
Photoengravers
Photographic film makers
Plastic cement workers
Plastic makers
Plastic wood workers
Polish makers
Rayon makers
Rubber buffers
Rubber cement workers
Shellackers
Shellac makers
Shoe factory workers
Shoe finishers
Shoe heel coverers, wood
Shoe polish makers
Silk makers
Smokeless powder makers
Soap makers
Stain removers
Straw hat makers
Tannery workers
Textile finishers
Textile printers
Toy makers
Varnishers
Varnish makers
Waterproofing makers

Reference

NELSON, K. W.; EGE, J. F., JR.; ROSS, M.; WOODMAN, L. E., AND SILVERMAN, L.: Sensory response to certain industrial solvent vapors. *J. Indust. Hyg. & Toxical.* 25: 282, 1943.

(14) Amyl Alcohol

fusel oil, grain oil, potato spirit, potato oil

Depending on source and method of manufacture, one or more isomeric primary, secondary, or tertiary alcohols may be present. When amyl alcohol is prepared by distillation of fusel oil, the chief constituent is isoamyl alcohol.

Harmful Effects

Local Liquid and vapor are irritating to eyes, mucous membranes, and skin.

Route of Entry Inhalation of vapor.

Systemic Early effects are irritation of nose and throat, followed by nausea, vomiting, facial flushing, headache, double vision, dizziness, and muscular weakness. Prolonged exposures to high concentrations can cause delirium, loss of consciousness, and death.

Special Diagnostic Test

Determination of amyl alcohol content of blood. *See* Von Oettingen, 1958.

Recommended Threshold Limit

(Isoamyl alcohol) 100 parts per million parts of air by volume or 360 milligrams per cubic meter of air

Potential Occupational Exposures

Alcohol distillery workers
Amyl acetate makers
Amyl alcohol workers
Amyl nitrite makers
Antifreeze makers
Drug makers
Explosive makers
Fat processors
Flotation workers
Fruit essence makers
Laboratory workers, chemical
Lacquerers
Lacquer makers
Mordanters
Nitrocellulose workers
Oil processors
Ore upgraders
Painters
Paint makers
Perfume makers
Photographic chemical makers
Plastic makers
Rubber makers
Shoe finishers
Smokeless powder makers
Varnishers
Varnish makers
Wax processors

Reference

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(15) Aniline and Other Amino Compounds of Benzene and Its Homologues

aminobenzene, phenylamine, aniline oil, aminophen, arylamine

Harmful Effects

Local Liquid may occasionally cause allergic contact dermatitis.
Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.
Systemic Aniline converts hemoglobin to methemoglobin, which causes anoxia and depression of central nervous system. Aniline may also have a direct toxic action causing hypotension and cardiac arrhythmias. Cyanosis is an early sign and is first noticed on lips, fingertips, and ears. Later, there may be headache, nausea, weakness, generalized aching, tachycardia, visual disturbances, mental confusion, and coma. Prolonged or repeated attacks may lead to anemia. Death from a single exposure is due to respiratory paralysis from central nervous system depression. Jaundice, enlargement of liver and spleen, and urinary bladder irritation

have also been reported following severe poisonings. Aniline is not a bladder carcinogen, but several derivatives such as beta-naphthylamine, benzidine and para-aminobiphenyl produce potentially malignant papillomas of the urinary bladder after years of exposure.

Special Diagnostic Tests

Examination of blood for methemoglobin. Examination of erythrocytes for Heinz bodies. Determination of para-aminophenol and diazotizable metabolites in urine. *See* Hill, 1953; Von Oettingen, 1958, and Elkins, 1959.

Recommended Threshold Limit

(Aniline) 5 parts per million parts of air by volume or 19 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Acetanilide workers
Acetic anhydride makers
Aniline workers
Antifouling paint makers
Blueprint paper makers
Bromine makers
Camphor makers
Coal tar workers
Color makers
Compositors
Disinfectant makers
Drug makers
Dye makers
Dyers
Explosive makers
Feather workers
Gasoline blenders
Hydroquinone makers
Ink makers
Leather makers
Lithographers
Millinery workers
beta-Naphthylamine workers
Nitraniline workers
Painters
Paint makers
Paint remover makers
Pencil makers, colored
Perfume makers
Petroleum refinery workers
Photographic chemical makers
Plastic workers
Polish makers
Printers
Quinone makers
Rocket fuel makers
Rubber chemical makers
Rubber mixers
Rubber reclaimers
Rubber workers
Rubber workers, pressroom
Tannery workers
Tetryl makers
Textile printers
Varnishers
Varnish makers
Vulcanizers

References

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

HALSTED, H. C.: Industrial methemoglobinemia. *J. Occup. Med.* 2: 591, 1960.

HILL, D. L.: Excretion of diazotizable metabolites in man after aniline exposure. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 347, 1953.

MUNN, A.: An unusual source of aniline poisoning. *Trans. Assoc. Indust. Med. Officers* 7: 78, 1957. Refers to marking ink used on wearing apparel.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(16) Antimony and Compounds

Harmful Effects

Local Antimony and certain of its salts, notably antimony fluoride, antimony trichloride, antimony tartrate, and antimony pentasulfide are irritant to skin and may produce contact dermatitis. Antimony trichloride has been reported to cause irritation and excoriation of mucous membranes of mouth and pharynx as well as swelling and vesiculation of lips and perforation of nasal septum.

Routes of Entry Ingestion or inhalation of dust or fume.

Systemic Trivalent antimony compounds are many times more toxic than pentavalent derivatives. Ingestion may produce gastrointestinal irritation with nausea, vomiting, and diarrhea. In acute severe poisonings due either to ingestion or inhalation of excessive amounts of antimony, there may be death from circulatory or respiratory failure or, as a late complication, toxic hepatitis proceeding to acute yellow atrophy. Inhalation of antimony dust may cause acute pneumonitis.

Chronic antimony poisoning is similar to chronic arsenic poisoning. There may be lassitude, irritability, stomatitis, nausea, constipation, myalgia, arthralgia, and leukopenia. There is some evidence that the heart may be injured in course of chronic antimony intoxication.

Special Diagnostic Tests

Examination of blood and urine for excessive amounts of antimony. *See* Elkins, 1959.

Recommended Threshold Limit

(Antimony) 0.5 milligram per cubic meter of air.

Potential Occupational Exposures

Antimony ore smelters
Antimony workers
Babbitt metal workers
Battery workers, storage
Britannia metal workers
Bronzers
Cable splicers
Ceramic makers
Dye makers
Fireworks makers
Flameproofers
Glass makers
Gold refiners
Lake color makers
Lead burners
Lead hardeners

Leather mordanters
Match workers
Metal bronzers
Miners
Organic chemical synthesizers
Paint makers
Pewter workers
Pigment makers
Plaster cast bronzers
Rubber makers
Solder makers
Textile dyers
Textile flameproofers
Textile printers
Type metal workers
Typesetters

References

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

RENES, L. E.: Antimony poisoning in industry. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 7: 99, 1953.

(17) Arsenic

Arsenic trioxide, the principal form in which the element is used, is frequently designated as arsenic or white arsenic. The element is considered nonpoisonous.

Harmful Effects

Local Contact with arsenic may produce facial and flexural eczematous dermatitis, ulcerations of the skin, conjunctivitis, rhinitis, nasal perforation, folliculitis, and pustules. Most of these effects are due to primary irritation, but some cases of contact dermatitis are due to allergic hypersensitivity. Prolonged absorption may result in generalized "rain drop" hyperpigmentation, premalignant keratoses on palms and soles, hair loss, and nail dystrophy.

Routes of Entry Ingestion or inhalation of dust or fume.

Systemic Acute systemic poisoning from ingestion produces a violent gastroenteritis, which may be followed by nephritis, hepatitis, or neuritis, but this type of poisoning is rare in industry. A massive inhalation exposure can produce bronchitis, but acute systemic intoxication is unlikely by this route. When arsenical intoxication occurs in industry, it is usually chronic in form. High exposures are frequently tolerated without symptoms of systemic poisoning.

Chronic exposure is characterized by insidious onset of malaise, abdominal complaints, pruritis, weakness, anorexia, and weight loss. There may be gingivitis and stomatitis with *garlic breath.* However, the garlic breath may be due to contamination with tellurium. Peripheral nerve degeneration resulting in progressive sensory alterations and motor disturbances is common. Kidney and liver damage may also occur. Prolonged inhalation of dust may result in laryngitis and bronchitis. Arsenic

has been suspected, but not proved, as a cancer producing agent in the liver and lungs.

Special Diagnostic Tests

Analysis of urine, hair, or nails for abnormal amounts of arsenic trioxide. The presence of arsenic in urine in amounts greater than 0.2 mg. per liter, is strongly suggestive of excessive absorption. *See* Elkins, 1959, and Vallee et al., 1960.

Recommended Threshold Limit

0.5 milligram per cubic meter of air.

Potential Occupational Exposures

Alloy makers
Aniline color makers
Arsenic workers
Babbitt metal workers
Boiler operators
Brass makers
Bronze makers
Bronzers
Cattle dip workers
Ceramic enamel makers
Ceramic makers
Copper smelters
Drug makers
Dye makers
Enamelers
Farmers
Fireworks makers
Glass makers
Gold refiners
Hair remover makers
Herbicide makers
Hide preservers
Insecticide makers
Lead shot makers
Lead smelters
Leather workers
Painters
Paint makers
Petroleum refinery workers
Pigment makers
Printing ink workers
Rodenticide makers
Semiconductor compound makers
Sheep dip workers
Silver refiners
Taxidermists
Textile printers
Tree sprayers
Type metal workers
Water weed controllers
Weed sprayers
Wood preservative makers
Wood preservers

References

DINMAN, B. D.: Arsenic; chronic human intoxication. *J. Occup. Med.* 2: 137, 1960.

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

HOLMQVIST, I.: Occupational arsenical dermatitis; a study among employees at a copper-ore smelting works including investigations of skin reactions to contact with arsenic compounds. *Acta dermat.-venereol. Supp.* 26, 1951.

PINTO, S. S. AND MCGILL, C. M.: Arsenic trioxide exposure in industry. *Indust. Med. & Surg.* 22: 281, 1953.

VALLEE, B. L.; ULMER, D. D., AND WACKER, W. E. C.: Arsenic toxicology and biochemistry. *A.M.A. Arch. Indust. Health* 21: 132, 1960.

(18) Arsine

hydrogen arsenide, arsenic trihydride, arseniuretted hydrogen

Arsine may be produced wherever nascent hydrogen comes in contact with arsenic. The hydrogen is usually produced by the action of acid upon a metal, the arsenic being present as an impurity in the metal or in the acid.

Harmful Effects

Local Bronze discoloration of skin.

Route of Entry Inhalation of gas.

Systemic Hemolysis of red blood corpuscles with resulting anemia and jaundice. Peripheral neuritis, visual disturbances, and delirium. Chronic intoxication may result in nephritis, myocarditis, and hepatitis. Garlic-like odor may be noted on breath.

Special Diagnostic Tests

Analysis of urine and blood for arsenic. In cases of chronic exposure, analysis of body hair for arsenic. *See* Elkins, 1959.

Recommended Threshold Limit

0.05 part per million parts of air by volume or 0.2 milligram per cubic meter of air.

Potential Occupational Exposures

Acetylene workers
Acid dippers
Aniline workers
Arsine workers
Bleaching powder makers
Bronzers
Cadmium workers
Dimethyl sulfate makers
Dye makers
Electrolytic copper makers
Electroplaters
Etchers
Ferrosilicon workers
Fertilizer makers
Galvanizers
Gold extractors
Hydrochloric acid workers
Illuminating gas workers
Jewelers
Lead burners
Lime burners
Metal cleaners
Metal refiners
Nitrocellulose makers
Ore smelter workers
Organic chemical synthesizers
Paper makers
Petroleum refinery workers
Plastic workers
Plumbers
Rayon makers
Soda makers
Solderers
Submarine workers
Sulfuric acid workers
Tinners
Zinc chloride makers

References

BULMER, F. M. R.; ROTHWELL, H. E.; POLACK, S. S., AND STEWART, D. W.: Chronic arsine poisoning among workers employed in the cyanide extraction of gold; a report of fourteen cases. *J. Indust. Hyg. & Toxicol.* 22: 111, 1940.

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

JOSEPHSON, C. J.; PINTO, S. S., AND PETRONELLA, S. J.: Arsine; electrocardiographic changes produced in acute human poisoning. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 43, 1951.

(19) Barium and Compounds

Harmful Effects

Local The soluble barium salts are irritating to skin and mucous membranes and may produce dermatitis, conjunctivitis, and marked bronchial irritation. Barium sulfide is known for its depilatory and bleaching action.

Route of Entry Ingestion or inhalation of dust or fume.

Systemic The soluble barium salts are highly toxic. Barium stimulates smooth, striated, and cardiac muscle and may produce violent peristalsis, arterial hypertension, muscle twitching, and cardiac dysfunction.

Barium sulfate is relatively insoluble and therefore innocuous when ingested; however, prolonged inhalation has been reported to cause a benign form of pneumoconiosis known as *baritosis*.

Special Diagnostic Test

Analysis of urine for barium. *See* Stewart and Stolman, 1961.

Recommended Threshold Limit

(Soluble compounds) 0.5 milligram per cubic meter of air.

Potential Occupational Exposures

Animal oil refiners
Barite millers
Barite miners
Barium workers
Bearing packing makers
Black ash workers
Boiler operators
Brick makers
Ceramic enamel makers
Ceramic makers
Core makers
Crystal makers (spectroscope, storage devices, digital calculators)
Disinfectant makers
Drug makers
Dye makers
Dyers
Electroplaters
Embalming fluid workers
Enamel makers
Explosive makers
Fat processors
Fireworks makers
Frit makers
Fungicide makers
Glass makers
Glazers

Grease additive makers
Hair removers
Ink makers
Insecticide makers
Laboratory workers, chemical
Lake color makers
Linoleum makers
Lithopone makers
Luminous paint workers
Match makers
Oil additive makers
Oilcloth makers
Oil well drillers
Organic chemical synthesizers
Paint makers
Paper makers
Photographic chemical makers
Pigment makers
Plastic makers
Rodenticide makers
Rubber makers
Soap makers
Steel carburizers
Straw hat bleachers
Tannery workers
Textile bleachers
Textile mordanters
Textile printers
Tracer bullet makers
Varnish makers
Vegetable oil processors
Vulcanizers
Waterproofers
Water treaters
Wax processors

References

PENDERGRASS, E. P. AND GREENING, R. R.: Baritosis; report of a case. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 7: 44 1953.

STEWART, C. P. AND STOLMAN, A. (EDITORS): *Toxicology; Mechanisms and Analytical Methods.* Vol. 2. Academic Press, New York, 1961.

(20) Benzene

benzol, phenyl hydride, coal naphtha, phene, benzole, cyclohexatriene

Harmful Effects

Local Exposure to liquid or vapor may produce primary irritation of skin, eyes, and mucous membranes of upper respiratory tract. Skin effects may include erythema, vesiculation, or a dry, scaly dermatitis.

Routes of Entry Inhalation of vapor. Percutaneous absorption of liquid leading to systemic toxicity is unlikely to occur.

Systemic Acute high exposures are responsible for initial exhilaration followed by signs and symptoms of central nervous system depression, including drowsiness, fatigue, headaches, dizziness, loss of consciousness, convulsions, and death.

Chronic low-level exposures may produce alterations of blood elements most commonly resulting in anemia, leukopenia, and thrombocytopenia. The bone marrow effects may be normal, hyperplastic, or hypoplastic and do not necessarily reflect the state of peripheral blood. Symptoms and signs relative to depression of these blood cellular elements include headache, fatigue, dizziness, loss of appetite, weakness, breathlessness, bleeding

from the nose and other mucous membranes, purpura, easy bruising, and proneness to infection. These effects generally improve after removal of the worker from areas of excessive exposure.

Benzene is a suspected carcinogenic agent. All forms of acute and chronic leukemia have been observed in workers with benzene intoxication.

Special Diagnostic Tests

Analysis of urinary sulfate and calculation of the urinary sulfate ratio (inorganic/total sulfate). Analysis of blood and urine benzene or urine phenol may be helpful in evaluating type and degree of exposure. *See* Gerarde, 1960.

Recommended Threshold Limit

25 parts per million parts of air by volume or 80 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Adhesive makers
Airplane dope makers
Alcohol workers
Aniline makers
Art glass workers
Asbestos product impregnators
Battery makers, dry
Belt scourers
Benzene hexachloride makers
Benzene workers
Bronzers
Burnishers
Can makers
Carbolic acid makers
Chlorobenzene makers
Coal tar refiners
Coal tar workers
Cobblers
Coke oven workers
Cyclohexane makers
DDT makers
Degreasers
Detergent makers
Dichlorobenzene makers
Diphenyl makers
Drug makers
Dry cleaners
Dye makers
Explosive makers
Fumigant makers
Fungicide makers
Furniture finishers
Glue makers
Hairdressers
Herbicide makers
Histology technicians
Ink makers
Insecticide makers
Lacquer makers
Leather makers
Linoleum makers
Lithographers
Maleic acid makers
Millinery workers
Nitrobenzene makers
Nitrocellulose workers
Oilcloth makers
Oil processors
Organic chemical synthesizers
Painters

Paint makers
Paraffin processors
Pencil makers
Perfume makers
Petrochemical workers
Petroleum refinery workers
Photographic chemical makers
Picric acid makers
Pottery decorators
Printers
Putty makers
Resin makers
Rubber cementers
Rubber gasket makers
Rubber makers
Shellac makers
Solvent makers
Stainers
Stain makers
Styrene makers
Synthetic fiber makers
Type cleaners
Varnish makers
Wax makers
Welders
Wire insulators

References

GERARDE, H. W.: *Toxicology and Biochemistry of Aromatic Hydrocarbons.* Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1960.

HUEPER, W. C.: Carcinogens in the human environment. *Arch. Path.* 71: 237, 1961.

SAITA, G. AND VIGLIANI, E. C.: The action of benzol in inducing leukemia. *Med. Lavoro* 53: 581, 1962.

(21) Benzidine

benzidine base, paradiaminodiphenyl

Harmful Effects

Local Primary irritant contact dermatitis has been reported; allergic contact dermatitis is rare.

Routes of Entry Percutaneous absorption, from dust; inhalation of dust, and ingestion.

Systemic Benzidine is a urinary bladder carcinogen. The actual carcinogens are probably metabolites, 4,4-diamino-3-diphenyl hydrogen sulfate or the orthohydroxy benzidine. Urinary manifestations are frequency, dysuria, and hematuria. Benzidine is unimportant as a methemoglobin former.

Special Diagnostic Test

Analysis of quinonizable substances in urine. *See* Glassman and Meigs, 1951.

Recommended Threshold Limit

Because of high incidence of bladder tumors in man, any exposure, including skin, is extremely hazardous.

218-695 O-66-7

Potential Occupational Exposures

Benzidine workers
Biochemists
Congo red makers
Crime detection laboratory workers (blood stains)
Dye intermediate makers
Dye makers
Microscopists
Organic chemical synthesizers
Rubber makers
Wood chemists

References

BAER, R. L.: Benzidine as cause of occupational dermatitis in a physician. *J. Am. Med. Assoc.* 129: 442, 1945.

CASE, R. A. M.; HOSKER, M. E.; MCDONALD, D. B., AND PEARSON, J. T.: Tumors of the uninary bladder in workmen engaged in the manufacture and use of certain dyestuff intermediates in the British chemical industry. 1, The role of aniline, benzidine, alpha-naphthylamine, and beta-naphthylamine. *Brit. J. Indust. Med.* 11: 75, 1954.

GLASSMAN, J. AND MEIGS, J. W.: Benzidine (4,4'-diaminobiphenyl) and substituted benzidines; a microchemical screening technic for estimating levels of industrial exposure from urine and air samples. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 519, 1951.

MEIGS, J. W.; BROWN, R. M., AND SCIARINI, L. J.: A study of exposure to benzidine and substituted benzidines in a chemical plant. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 533, 1951.

(22) Benzyl Chloride

alpha-chlorotoluene

Harmful Effects

Local Both liquid and concentrated vapor are highly irritating to eyes and mucous membranes. In eye, benzyl chloride is not only a potent lacrimator but also a protein denaturant. On skin, liquid is a vesicant.
Route of Entry Inhalation of vapor.
Systemic Systemic effects are usually prevented by intolerable irritation of eyes and nose. Continued exposure can produce bronchial irritation, cough, and pulmonary edema.

Special Diagnostic Test

None.

Recommended Threshold Limit

1 part per million parts of air by volume or 5 milligrams per cubic meter of air.

Potential Occupational Exposures

Benzyl chloride workers
Drug makers
Dye intermediate makers
Dye makers
Gasoline additive workers
Germicide makers
Motor fuel blenders
Penicillin makers

Perfume makers
Photographic developer makers
Resin makers
Rubber makers
Tannin makers
Wetting agent makers

(23) Beryllium and Compounds

Prior to 1949 the fluorescent lamp industry used beryllium in the inside coating of lamps. With the recognition of the ill effects of the metal and its compounds, the industry on June 30, 1949, by general agreement eliminated beryllium by substituting another material.

Harmful Effects

Local Contact with beryllium salts may produce contact dermatitis of the hypersensitivity or primary irritant type. Contamination of abrasions or superficial lacerations with the more soluble beryllium salts may cause a chronic, indolent ulcer. Intracutaneous implantation of spicules of beryllium metal or certain beryllium salts may result in the formation of a low-grade granulomatous lesion. Irritation of conjunctiva and cornea may follow contact with beryllium salts, as may rhinitis and nasopharyngitis.

Route of Entry Inhalation of fume or dust.

Systemic Inhalation of beryllium dust or fume may result in the production of systemic disease either of an acute or of a chronic nature, depending upon the extent of exposure and the nature of the beryllium compound involved.

Acute beryllium disease has resulted from exposure to beryllium compounds in industrial plants producing beryllium from the ore, in metallurgic and ceramics laboratories, and in the fluorescent lamp industry. The following beryllium compounds, in addition to the metal, have been shown to cause acute poisoning: beryllium oxide, sulfate, fluoride, hydroxide, and chloride. The cases associated with the preparation of phosphors involved exposure to beryllium oxide and to zinc beryllium silicate.

Chronic beryllium poisoning has been reported as resulting from exposure in plants handling beryllium phosphors, in beryllium copper founding, in ceramics laboratories, in metallurgic shops and in plants producing beryllium compounds from the ore. This disease has also been reported as occurring among individuals exposed to atmospheric pollution in the vicinity of plants processing beryllium and in persons dwelling in the same household as beryllium workers. Inhalation of the dust of beryl, the beryllium ore, has produced to date no known cases of acute or chronic beryllium poisoning.

Granulomatous lesions of the skin, liver, kidneys, spleen, and lymph nodes may be seen in some patients with beryllium disease; however,

the most striking features of both the acute and chronic forms are referable to the lungs.

Although of dissimilar roentgenologic and histopathologic appearance, both the acute and the chronic forms of beryllium poisoning have some similar signs and symptoms. These include a relatively nonproductive cough, progressive dyspnea, anorexia, and loss of weight. The chief differences between the two forms are seen in the suddenness of onset and in the rate of progression. In neither the acute nor the chronic form of beryllium disease has there been reported any evidence to suggest that microorganisms might play a significant role in pathogenesis.

In the acute pulmonary form, the symptoms of pneumonitis may appear within several hours to several weeks following the initial exposure of the patient to beryllium, and the radiographic changes may become noticeable within from one to three weeks after the onset of symptoms. There is usually rapid progression of signs and symptoms including dyspnea, anorexia, and extreme weight loss. There is generally complete recovery within a period of about 6 months. Cases which terminate fatally usually do so as a result of acute cor pulmonale.

The typical pattern shown by the chest roentgenogram in acute beryllium pneumonitis is a bilateral, patchy infiltrate which resembles the pattern seen in pulmonary edema. This infiltrate may be superseded by a coarse, nodular appearance before final clearance or recovery occurs.

The pathologic lesion seen in the lung in acute beryllium disease is a chemical pneumonitis or bronchoalveolitis, the severity of which is usually proportional to the intensity of exposure.

In chronic beryllium disease the symptoms are generally delayed in onset and persistent in character. They are commonly precipitated or exacerbated by stresses such as pregnancy, respiratory infection, and thyrotoxicosis. The pulmonary manifestations may be mimicked by symptoms of other lung diseases, such as the fibrosing interstitial pneumonitis of the Hamman-Rich syndrome and the pulmonary granulomatosis of sarcoidosis. Dyspnea, cough, anorexia, and weight loss are among the most frequent manifestations of chronic beryllium disease. As the disease progresses, signs and symptoms of cor pulmonale may supervene.

The earliest roentgenographic evidence of pulmonary involvement may appear within a few weeks of the first symptoms of the disease. The most significant feature of the roentgenogram is a uniform distribution of fine granulation, with variation from a ground glass appearance through a diffuse reticular pattern to distinct nodulation superimposed on a granular background.

Additional aid in the diagnosis of chronic beryllium poisoning may be gained through the study of pulmonary function, by use of the beryllium

patch test, through determinations of the beryllium content of body fluids, and through histologic and chemical study of the surgical lung biopsy.

It is generally accepted that the basic pulmonary physiopathology in this disease is an alveolar-capillary block. This diffusion defect can usually be demonstrated in patients with chronic beryllium disease and, while it is not pathognomonic, it may often be helpful in ruling out certain other of the pulmonary granulomatoses.

The place of the patch test in the diagnosis of beryllium disease is uncertain. Some investigators have shown excellent correlation between positive skin reactions to beryllium and proved poisoning, while others have not been able to show such correlation and have pointed out certain hazards inherent in the test itself.

The finding of increased amounts of beryllium in the body tissues and fluids does not, by any means, justify in itself a diagnosis of beryllium disease, nor does the absence of increased amounts of beryllium rule out chronic beryllium poisoning.

The more liberal application of the use of the surgical lung biopsy has been of major aid in the diagnosis of beryllium disease. It must be pointed out, however, that in some cases even the most experienced pathologist may find it impossible to distinguish between this condition and sarcoidosis by examination of histologic sections.

There is no available evidence to implicate beryllium disease as predisposing to pulmonary tuberculosis. Moreover, a causal relationship between beryllium disease and lung cancer has not been established.

Special Diagnostic Tests

Analysis of urine and tissue for abnormal amounts of beryllium. *See* Cholak, 1959.

Recommended Threshold Limit

(Beryllium) 0.002 milligram per cubic meter of air.

Potential Occupational Exposures

Beryllium alloy machiners
Beryllium alloy makers
Beryllium compound makers
Beryllium copper founders
Beryllium copper grinders
Beryllium copper polishers
Beryllium extractors
Beryllium metal machiners
Beryllium phosphor makers
Beryllium workers
Cathode ray tube makers
Ceramic makers
Electric equipment makers
Fluorescent screen makers
Gas mantle makers
Missile technicians
Neon sign workers
Neon tube makers
Nonsparking tool makers
Nuclear physicists
Nuclear reactor workers
Precision instrument makers
Refractory material makers

References

BRESLIN, A. J. AND HARRIS, W. B.: Health protection in beryllium facilities, summary of ten years of experience. HASL–36. U.S. Atomic Energy Commission, New York, 1958.

CHOLAK, J.: The analysis of traces of beryllium. *A.M.A. Arch. Indust. Health* 19: 205, 1959.

CURTIS, G. H.: The diagnosis of beryllium disease, with special reference to the patch test. *A.M.A. Arch. Indust. Health* 19: 150, 1959.

EISENBUD, M.; WANTA, R. C.; DUSTAN, C.; STEADMAN, L. T.; HARRIS, W. B., AND WOLF, B. S.: Non-occupational berylliosis. *J. Indust. Hyg. & Toxicol.* 31: 282, 1949.

GROSS, P.: The concept of the Hamman-Rich syndrome; a critique. *Am. Rev. Resp. Dis.* 85: 828, 1962.

HARDY, H. L.: Beryllium disease, a continuing diagnostic problem. *Am. J. Med. Sc.* 242: 150, 1961.

HARDY, H. L.: Reaction to toxic beryllium compounds; terminology. *J. Occup. Med.* 4: 532, 1962. Regards beryllium disease as a systemic intoxication, not as a pneumoconiosis.

LEWIS, C. E.: Workshop on beryllium. *J. Occup. Med.* 4: 80, 1962.

MUSCHENHEIM, C.: Some observations on the Hamman-Rich disease. *Am. J. Med. Sc.* 241: 279, 1961.

PEYTON, M. F. AND WORCESTER, J.: Exposure data and epidemiology of the beryllium case registry, 1958. *A.M.A. Arch. Indust. Health* 19: 94, 1959.

STERNER, J. H. AND EISENBUD, M.: Epidemiology of beryllium intoxication. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 123, 1951.

VAN ORDSTRAND, H. S.: Acute beryllium poisoning. *In* Vorwald, A. J. (editor): *Pneumoconiosis; Beryllium, Bauxite Fumes, Compensation.* Sixth Saranac Laboratory Symposium, 1947. Paul B. Hoeber, New York, 1950.

VAN ORDSTRAND, H. S.: Diagnosis of beryllium disease. *A.M.A. Arch. Indust. Health* 19: 157, 1959.

(24) Bismuth and Compounds

Harmful Effects

Local Bismuth subnitrate may cause skin irritation.

Route of Entry Ingestion of powder.

Systemic Basic salts are insoluble and exhibit low oral toxicity. Formerly used in an injectable form as a treatment for syphilis. Toxic symptoms following injection include loss of appetite, foul breath, gingivitis, stomatitis, weakness, and diarrhea. Toxic hepatitis and nephritis rarely occur. No poisonings related to occupation have been found in the literature.

Special Diagnostic Tests

Analysis of blood and urine for excessive amounts of bismuth. *See* Von Oettingen, 1958.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Bismuth workers
Ceramic capacitor makers
Ceramic colorers
Ceramic enamel makers
Cosmetic makers
Deodorant makers
Disinfectant makers
Drug makers
Dyers
Face powder makers
Fuse makers
Fusible alloy makers
Laboratory workers, chemical
Luminous enamel makers
Luminous paint makers
Metallic bath workers
Pearl makers
Perfume makers
Permanent magnet makers
Pigment makers
Semiconductor makers
Solder makers
Tin lusterers

References

BROWNING, E.: *Toxicity of Industrial Metals.* Butterworths, London, 1961.
VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(25) Boron Compounds

Harmful Effects

Local Boric acid may produce primary skin irritation and conjunctivitis.

Routes of Entry Percutaneous absorption of liquid; inhalation of gas or vapor.

Systemic Boron hydrides (diborane, pentaborane, decaborane) are the most important compounds of this group.

Inhalation of diborane may result in chest tightness, cough, headaches, nausea, chills, dizziness, and drowsiness. These complaints are generally of short duration. Pneumonia may develop following severe exposures.

Pentaborane and decaborane produce predominantly central nervous system symptoms and signs. Hyperexcitability, headaches, muscle twitching, convulsions, dizziness, disorientation, and unconsciousness may occur early or be delayed for 24 hour or more following excessive exposures to these compounds.

Skin and respiratory tract irritation and central nervous system effects have been reported from animal experiments with amine and alkyl boranes. The alykyl boranes seem to be more toxic than the amine compounds and decaborane, but less toxic than pentaborane. The major effect of repeated inhalation of boron trifluoride in laboratory animals was respiratory irritation which resulted in a pneumonitis.

Special Diagnostic Tests

Analysis of boron in blood, urine and body tissues. *See* Jacobson, 1958.

Recommended Threshold Limit

Diborane, 0.1 part per million parts of air by volume or 0.1 milligram per cubic meter of air.

Pentaborane, 0.005 part per million parts of air by volume or 0.01 milligram per cubic meter of air.

Decaborane, 0.05 part per million parts of air by volume or 0.3 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Boron trifluoride, 1 part per million parts of air by volume or 3 milligrams per cubic meter of air.

Potential Occupational Exposures

Diborane

Diborane workers
Organic chemical synthesizers
Rocket fuel handlers
Rocket fuel makers

Pentaborane

Gasoline additive makers
Pentaborane workers
Rocket fuel handlers
Rocket fuel makers

Decaborane

Chemical scavenger makers
Chemical stabilizer makers
Decaborane workers
Dyers
Gasoline additive makers
Insecticide makers
Organic chemical synthesizers
Resin makers
Rocket fuel handlers
Rocket fuel makers
Rubber makers
Rust inhibitor makers
Welding flux makers

Boron trifluoride

Boron trifluoride workers
Fumigant makers
Fumigators
Nuclear instrument makers
Organic chemical synthesizers

References

JACOBSON, K. H.: Transactions, symposium on health hazards of military chemicals. CWL Special Publication 2–10. U.S. Army Chemical Warfare Laboratories, Army Chemical Center, Maryland, 1958.

LOWE, H. J. AND FREEMAN, G.: Boron hydride (borane) intoxication in man. *A.M.A. Arch. Indust. Health* 16: 523, 1957.

OFFICE OF DIRECTOR OF DEFENSE RESEARCH AND ENGINEERING, DEPARTMENT OF DEFENSE: *The Handling and Storage of Liquid Propellants.* The Department, Washington, D.C., 1961.

ROUSH, C., JR.: The toxicology of the boranes. *J. Occup. Med.* 1: 46, 1959.

TORKELSON, T. R.; SADEK, S. E., AND ROWE, V. K.: The toxicity of boron trifluoride when inhaled by laboratory animals. *Am. Indust. Hyg. Assoc. J.* 22: 263, 1961.

(26) Brass

Harmful Effects

Local Brass dust and slivers may cause dermatitis by mechanical irritation.

Route of Entry Inhalation of fume.

Systemic Brass is chiefly an alloy of copper and zinc, usually in the ratio of 2 to 1. Since zinc melts at a lower temperature than copper, the fusing of brass is attended by liberation of considerable quantities of zinc oxide. Inhalation of zinc oxide fumes may result in production of signs and symptoms of metal fume fever; *see* Zinc and Compounds. *Brass founder's ague* is the name often given to metal fume fever occurring in the brass-founding industry.

Since brass may contain significant quantities of lead, brass foundings may release sufficient amounts of lead fumes into working environment to produce lead intoxication in those workers exposed.

Special Diagnostic Test

None. *See* Lead and Compounds.

Recommended Threshold Limit

Not established. Zinc oxide, 5 milligrams per cubic meter of air; lead, 0.2 milligram per cubic meter of air.

Potential Occupational Exposures

Bench molders
Brass founders
Brass workers
Braziers
Bronzers
Core makers
Galvanizers
Junk metal refiners
Welders
Zinc founders
Zinc smelters

Reference

HAMILTON, A. AND HARDY, H. L.: *Industrial Toxicology.* Paul B. Hoeber, New York, 1949.

(27) Bromine and Compounds

Compounds include hydrogen bromide, methyl bromide (bromomethane), and ethyl bromide (bromoethane). Ethylene dibromide is presented separately.

Harmful Effects

Local Bromine and most of its compounds are highly irritating to eyes, mucous membranes of nose and throat, and to skin.

Routes of Entry Inhalation of vapor and gas. Percutaneous absorption of methyl bromide may occur.

Systemic Acute exposure to high concentrations of bromine or hydrogen bromide can produce pulmonary edema, which may be delayed. There may be a bromine odor on breath.

In acute methyl bromide poisoning, the central nervous system, lungs, liver, and kidney are damaged but effects may be delayed hours after exposure. Pulmonary effects include bronchitis and pulmonary edema. Neurologic effects include headache, visual disturbances, speech dysfunction, mental aberrations, tremors, incoordination, convulsions, and coma. Death is not uncommon. In chronic poisoning the damage is usually limited to central nervous system.

Ethyl bromide is less toxic than methyl bromide but produces similar effects. In addition ethyl bromide has a pronounced narcotic effect and can damage heart.

Special Diagnostic Tests

Blood and urinary bromide determinations. *See* Von Oettingen, 1958, and Rathus and Landy, 1961.

Recommended Threshold Limit

Bromine, 0.1 part per million parts of air by volume or 0.7 milligram per cubic meter of air.

Hydrogen bromide, 3 parts per million parts of air by volume or 10 milligrams per cubic meter of air.

Methyl bromide, 20 parts per million parts of air by volume or 80 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Ethyl bromide, 200 parts per million parts of air by volume or 890 milligrams per cubic meter of air.

Potential Occupational Exposures

Bromine

Bromine workers
Drug makers
Dye makers
Ethylene bromide makers
Ethylene dibromide makers
Fire extinguisher fluid makers
Gold extractors
Methyl bromide makers
Photographic chemical makers
Silk bleachers
Water treaters
Wool shrinkproofers

Hydrogen bromide

Barbiturate makers	Hydrogen bromide workers
Bromide makers	Organic chemical synthesizers
Drug makers	Petroleum refinery workers
Hormone makers	

Methyl bromide

Color makers	Ionization chamber workers
Drug makers	Organic chemical synthesizers
Dye makers	Refrigerant makers
Fire extinguisher workers	Refrigeration workers
Fumigant makers	Soil fumigators
Grain fumigators	Vegetable oil extractors
Insecticide makers	Wool degreasers

Ethyl bromide

Anesthetists	Grain fumigators
Drug makers	Organic chemical synthesizers
Ethyl bromide workers	Refrigerant makers
Fruit fumigators	Refrigeration workers
Fumigant makers	Solvent workers

References

INGRAM, F. R.: Methyl bromide fumigation and control in the date-packing industry. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 193, 1951.

RATHUS, E. M. AND LANDY, P. J.: Methyl bromide poisoning. *Brit. J. Indust. Med.* 18: 53, 1961.

VON OETTINGEN, W. F.: The halogenated aliphatic, olefinic, cyclic, aromatic, and aliphatic-aromatic hydrocarbons including the halogenated insecticides, their toxicity and potential dangers. Pub. Health Service Pub. No. 414. U.S. Government Printing Office, Washington, D.C., 1955.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(28) Butadiene

biethylene, bivinyl, butadiene monomer, divinyl, erythrene, methylallene, pyrrolylene, vinylethylene

Harmful Effects

Local Gas is irritating to mucous membranes and eyes, and liquid can produce a primary irritant type of contact dermatitis.

Route of Entry Inhalation of gas.

Systemic In high concentrations, gas can act as irritant, producing cough, and as narcotic, producing fatigue, drowsiness, headache, vertigo, loss of consciousness, respiratory paralysis, and death. Probably no cumulative effects.

Special Diagnostic Test

None.

Recommended Threshold Limit

1,000 parts per million parts of air by volume or 2,200 milligrams per cubic meter of air.

Potential Occupational Exposures

Butadiene workers
Organic chemical synthesizers
Rocket fuel handlers
Rocket fuel makers
Rubber makers

Reference

CARPENTER, C. P.; SHAFFER, C. B.; WEIL, C. S., AND SMYTH, H. F., JR.: Studies on the inhalation of 1,3-butadiene; with a comparison of its narcotic effect with benzol, toluol, and styrene, and a note on the elimination of styrene by the human. *J. Indust. Hyg. & Toxicol.* 26: 69, 1944.

(29) Butanone. *See* Ketones

(30) n-Butyl Acetate

butyl ethanoate, acetic acid butyl ester

Harmful Effects

Local High vapor concentrations irritate mucous membranes of eyes, nose, and throat. Vacuolization of corneal epithelium has been observed. Repeated contact with liquid can produce dry, scaly, and fissured dermatitis.

Route of Entry Inhalation of vapor.

Systemic Based on animal studies, vapor has narcotic effect in high concentrations and can cause drowsiness and loss of consciousness.

Special Diagnostic Test

None.

Recommended Threshold Limit

200 parts per million parts of air by volume or 950 milligrams per cubic meter of air.

Potential Occupational Exposures

Airplane dope makers
n-Butyl acetate workers
Cellulose acetopropionate finishers
Dope workers
Enameled leather makers
Enamel workers
Flavoring makers
Lacquerers
Lacquer makers
Leather dope workers
Paper makers, coated
Patent leather makers
Perfume makers

Photographic film makers
Plastic workers
Safety glass makers
Stainers
Stain makers
Stain removers
Varnish workers
Vinyl resin workers

Reference

VON OETTINGEN, W. F.: The aliphatic acids and their esters: toxicity and potential dangers; the saturated monobasic aliphatic acids and their esters. *A.M.A. Arch. Indust. Health* 21: 28, 1960.

(31) n-Butyl Alcohol

1-butanol, butyl hydroxide, propylcarbinol, butyric alcohol, hydroxybutane

Harmful Effects

Local Vapor is an irritant to conjunctiva and mucous membranes of upper respiratory tract. A peculiar keratitis characterized by numerous vacuoles has been reported. Liquid is primary skin irritant.

Route of Entry Inhalation of vapor.

Systemic No cases of systemic toxicity in humans have been reported, either from n-butyl alcohol or its isomers.

Special Diagnostic Test

None.

Recommended Threshold Limit

100 parts per million parts of air by volume or 300 milligrams per cubic meter of air.

Potential Occupational Exposures

Butyl acetate makers
n-Butyl alcohol workers
Butyric acid makers
Detergent makers
Di-n-butyl phthalate makers
Dye makers
Hydraulic fluid makers
Lacquerers
Lacquer makers
Melamine resin makers
Nitrocellulose makers
Photographic film makers
Plasticizer makers
Polyvinyl resin makers
Rubber cement makers
Shellac makers
Stainers
Stain makers
Urea-formaldehyde resin makers
Varnish makers

References

HENSON, E. V.: The toxicology of some aliphatic alcohols; part 2. *J. Occup. Med.* 2: 497, 1960.

STERNER, J. H.; CROUCH, H. W.; BROCKMYRE, H. F., AND CUSACK, M.: A ten-year study of butyl alcohol exposure. *Am. Indust. Hyg. Assoc. Quart.* 10: 53, 1949.

TABERSHAW, I. R.; FAHY, J. P., AND SKINNER, J. B.: Industrial exposure to butanol. *J. Indust. Hyg. & Toxicol.* 26: 328, 1944.

VON OETTINGEN, W. F.: The aliphatic alcohols, their toxicity and potential dangers in relation to their chemical constitution and their fate in metabolism. Pub. Health Bull. No. 281. U.S. Government Printing Office, Washington, D.C., 1943.

(32) n-Butylamine

1-aminobutane

Harmful Effects

Local Liquid is irritating and produces severe contact dermatitis and corneal injury.

Route of Entry Inhalation of vapor.

Systemic Vapor is irritating to respiratory tract and can produce pulmonary edema. Stimulation of central nervous system, followed by depression, convulsions, and narcosis.

Special Diagnostic Test

None.

Recommended Threshold Limit

5 parts per million parts of air by volume or 15 milligrams per cubic meter of air.

Potential Occupational Exposures

n-Butylamine workers
Butylaminophenol makers
Drug makers
Dye makers
Emulsifier makers
Insecticide makers
Petroleum dewaxers
Rubber makers
Tanning chemical makers

(33) Butyl Mercaptan. *See* Mercaptans

(34) Cadmium

Harmful Effects

Local Irritant to mucous membranes. Produces yellow discoloration of teeth. Certain salts may cause contact dermatitis due to allergic hypersensitization.

Routes of Entry Ingestion or inhalation of fume or dust.

Systemic Ingestion results in production of signs and symptoms of acute gastroenteritis. Inhalation of cadmium oxide fume may cause respiratory tract irritation with attendant sore, dry throat and a metallic taste followed by cough, chest pain, and dyspnea. Bronchitis, pneumonitis, and pulmonary edema may occur as result of irritative action of fume. Additional complaints of headache, dizziness, loss of appetite and weight loss may be pronounced. Liver, kidneys, and bone marrow may be injured by the metal.

It is probable that cadmium, under certain conditions, can produce chronic intoxication. Reports suggest that at least 2 years of exposure are necessary for this type of poisoning to develop. The most commonly accepted manifestations of prolonged exposure to cadmium are pulmonary emphysema, renal damage, and proteinuria. The last is not necessarily a result of renal damage and often may be demonstrated in exposed workers with apparently healthy kidneys. Other conditions that have been reported following long exposure to cadmium include anosmia, an increased incidence of nephrolithiasis, and occasional evidence of liver damage.

Special Diagnostic Test

Analysis of urine for increased amounts of cadmium. *See* Elkins, 1959.

Recommended Threshold Limit

(Cadmium oxide fume) 0.1 milligram per cubic meter of air.

Potential Occupational Exposures

Alloy makers
Aluminum solder makers
Battery makers, storage
Cadmium compound collecting bag cleaners
Cadmium compound collecting bag handlers
Cadmium platers
Cadmium smelters
Cadmium vapor lamp makers
Cadmium workers
Ceramic makers
Dental amalgam makers
Electric instrument makers
Electroplaters
Engravers
Glass makers
Incandescent lamp makers
Lithographers
Lithopone makers
Metalizers
Paint makers
Paint sprayers
Photoelectric cell makers
Pigment makers
Small arms ammunition makers
Smoke bomb makers
Solderers
Solder makers
Textile printers
Welders, cadmium alloy
Welders, cadmium plated object
Zinc refiners

References

ANNOTATION: Danger at work. *Lancet* 2: 656, 1962. Review of Annual report of the Chief Inspector of Factories on industrial health, 1961. Cmd. 1815. Her Majesty's Stationery Office, London, 1962.

DIVISION OF INDUSTRIAL HYGIENE, U.S. PUBLIC HEALTH SERVICE: Cadmium poisoning. 109 references. *Pub. Health Rep.* 57: 601, 1942. Reprint no. 2371.

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

FRIBERG, L.: Chronic cadmium poisoning. *A.M.A. Arch. Indust. Health* 20: 401, 1959.

LANE, R. E. AND CAMPBELL, A. C. P.: Fatal emphysema in two men making a copper cadmium alloy. *Brit. J. Indust. Med.* 11: 118, 1954.

SMITH, J. C.; WELLS, A. R., AND KENCH, J. E.: Observations on the urinary protein of men exposed to cadmium dust and fume. *Brit. J. Indust. Med.* 18: 70, 1961.

TAYLOR, C. M.: Cadmium as a health hazard. *Trans. Assoc. Indust. Med. Officers* 7: 122, 1957.

(35) Calcium Cyanamide

nitrolim, calcium carbimide, cyanamide

Harmful Effects

Local Contact with cyanamide lumps or powder can cause dermatitis from primary irritation or allergic hypersensitivity. Dust can cause irritation of conjunctiva and mucous membranes of nose and throat. Perforation of nasal septum can occur.

Route of Entry Inhalation of dust.

Systemic Transient attacks of vasodepression manifested by flushing of skin of face, neck, and hands, sweating, hypotension, tachycardia, dyspnea, headache, vertigo, vomiting, and tremors. Symptoms are aggravated by ingestion of alcohol.

Special Diagnostic Tests

Analysis of blood and urine for cyanamide. Blood spectrum may resemble cyanhemoglobin or cyanhematin. *See* Von Oettingen, 1958, and Buyske and Downing, 1960.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Ammonia makers
Calcium cyanamide workers
Cyanamide makers
Farmers
Fertilizer workers
Herbicide workers
Nitrogen compound makers
Organic chemical synthesizers
Steel carburizers
Steel casehardeners

References

BUYSKE, D. A. AND DOWNING, V.: Spectrophotometric determination of cyanamide. *Anal. Chem.* 32: 1798, 1960.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(36) Calcium Oxide

lime, burnt lime, quicklime, calx, fluxing lime

Harmful Effects

Local Calcium oxide is irritating to skin, conjunctiva, cornea, and mucous membranes of upper respiratory tract.

Route of Entry Inhalation of dust.

Systemic A few cases of pneumonia presumed to be due to inhalation of dust have been reported.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Tentative) 5 milligrams per cubic meter of air.

Potential Occupational Exposures

Brick masons
Calcium carbide makers
Calcium cyanamide makers
Calcium oxide workers
Candle makers
Cement workers
Ceramic workers
Chloride of lime makers
Dye makers
Electroplaters
Farmers
Fertilizer makers
Food processors
Fungicide workers
Glass makers
Insecticide workers
Metal smelters
Mortar workers
Paint makers
Paper hangers
Paper makers
Petroleum refinery workers
Plaster makers
Rubber makers
Soap makers
Steel workers
Sugar refiners
Tannery workers
Water treaters

(37) Carbon Dioxide

carbonic acid gas

Harmful Effects

Local When carbon dioxide combines in high concentrations with water, carbonic acid is formed. This material upon contact may produce slight skin, eye, or mucous membrane irritation.

Route of Entry Inhalation of gas.

Systemic Inhalation of 3 to 5 percent carbon dioxide will generally produce an increase in respiratory rate. Concentrations of 8 to 15 percent when inhaled may produce headache, dizziness, nausea, vomiting, and unconsciousness. Exposure to higher concentrations may cause immediate collapse, coma, and death.

Because carbon dioxide is heavier than air, it tends to accumulate in high concentrations in lowermost portions of enclosed or walled-in areas such as at bottom of fermenting tanks or shipholds. This is more likely to occur when the gas is being continuously formed, as during fermenting processes. Because of this tendency, carbon dioxide may dilute normal air in these enclosed areas and act as simple asphyxiant.

218-695 O-66-8

Special Diagnostic Test

None.

Recommended Threshold Limit

5,000 parts per million parts of air by volume or 9,000 milligrams per cubic foot of air.

Potential Occupational Exposures

Aerosol packagers
Alkali salt makers
Bakers
Baking powder makers
Beverage carbonators
Blast furnace workers
Boiler room workers
Brass founders
Brewers
Brick burners
Bronze founders
Caisson workers
Canners
Carbonated water makers
Carbon dioxide makers
Carbon dioxide workers
Carbonic acid makers
Cave explorers
Charcoal burners
Cupola men
Dairy farmers
Disinfectant makers
Divers
Dock workers
Drug makers
Dry ice workers
Drying room workers
Dye makers
Ensilage diggers
Explosive makers
Fertilizer workers
Fire extinguisher makers
Firemen
Foundry workers
Furnace workers
Glue makers
Grain elevator workers
Ice cream makers
Insecticide makers
Lime kiln workers
Linseed oil boilers
Mineral water bottlers
Miners
Natural carbon dioxide workers
Pottery workers
Refrigerating car workers
Refrigerating plant workers
Salicylic acid makers
Sewer workers
Silo cleaners
Soda makers
Starch makers
Submarine crewmen
Sugar refiners
Tannery pit men
Tobacco moisteners, storehouse
Tunnel workers
Urea makers
Vatmen
Vault workers
Vinegar makers
Vulcanizers
Welders, inert atmosphere
Well cleaners
White lead makers
Yeast makers

Reference

WILLIAMS, H. L.: Carbon dioxide poisoning; report of eight cases with two deaths. *Brit. Med. J.* 2: 1012, 1958.

(38) Carbon Disulfide

carbon bisulfide, dithiocarbonic anhydride

Harmful Effects

Local Liquid and concentrated vapor are irritating to eyes, nose, and skin. Carbon disulfide is one of the most severe of organic solvents in its irritating action on skin.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid or vapor.

Systemic Carbon disulfide is potent narcotic agent. Signs and symptoms of acute carbon disulfide poisoning stem from its narcotic action.

In chronic carbon disulfide poisoning, the nervous system bears the brunt of damage. There may be neuritis involving peripheral and cranial nerves (optic and retrobulbar neuritis). Transient mental aberrations are common. These may include mania, depression, hallucinations, and other abnormal mental states. Gastric disturbances are common, and symptoms may simulate those complained of by patients with peptic ulcers. Heart, liver, and kidney damage may result from chronic intoxication.

Special Diagnostic Tests

Analysis of urine and blood for carbon disulfide. *See* Von Oettingen, 1958.

Recommended Threshold Limit

20 parts per million parts of air by volume or 60 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Acetylene workers
Ammonium salt makers
Bromine processors
Carbanilide makers
Carbon disulfide workers
Carbon tetrachloride makers
Cellophane makers
Cementers, rubber shoe
Cement mixers, rubber
Coal tar distillers
Degreasers
Dry cleaners
Dyestuff makers
Electroplaters
Enamelers
Enamel makers
Explosive workers
Fat processors
Flotation agent makers
Fumigant workers
Glass makers
Glue workers
Iodine processors
Laboratory workers, chemical
Lacquer makers
Match makers
Oil processors
Optical glass makers
Painters
Paint makers
Paint remover makers
Paraffin workers

Pesticide makers
Phosphorus processors
Preservative makers
Putty makers
Rayon makers
Resin makers
Rocket fuel makers
Rubber cement makers
Rubber dryers
Rubber makers
Rubber reclaimers
Selenium processors
Smokeless powder makers
Soil fumigators
Sulfur processors
Tallow makers
Textile makers
Vacuum tube makers
Varnish makers
Varnish remover makers
Veterinarians
Vulcanizers
Wax processors

References

HENDERSON, Y. AND HAGGARD, H. W.: *Noxious Gases and the Principles of Respiration Influencing Their Action.* 2nd ed. Reinhold Publishing Corp., New York, 1943.

KLEINFELD, M. AND TABERSHAW, I. R.: Carbon disulfide poisoning; report of two cases. *J. Am. Med. Assoc.* 159: 677, 1955.

VIGLIANI, E. C.: Clinical observations on chronic carbon disulfide intoxication in Italy. *Indust. Med. & Surg.* 19: 240, 1950.

(39) Carbon Monoxide

Harmful Effects

Local None.

Route of Entry Inhalation of gas.

Systemic Combines with hemoglobin to form carboxyhemoglobin which interferes with oxygen carrying capacity of blood, resulting in a state of tissue hypoxia. Except for this, carbon monoxide is essentially a physiologically inert gas. It is probable that exposure to carbon monoxide gas does not produce a truly chronic type of intoxication but may, upon repeated intermittent exposures, produce repeated transient episodes of mild acute poisoning.

Special Diagnostic Test

Analysis of blood for carboxyhemoglobin. *See* Von Oettingen, 1958.

Recommended Threshold Limit

100 parts per million parts of air by volume or 110 miligrams per cubic meter of air.

Potential Occupational Exposures

Acetic acid makers
Acetylene workers
Airplane pilots
Ammonia makers
Artificial gas workers
Automobile users
Blast furnace gas users
Blast furnace workers
Boiler room workers
Brass founders

Brewers
Brick burners
Carbon monoxide workers
Diesel engine operators
Dock workers
Firemen
Foundry workers
Furnace starters
Garage mechanics
Gasoline engine testers
Heat treaters
Lift truck operators
Metal oxide reducers
Methanol makers
Miners
Mond process workers
Nickel refiners
Nickel smelters
Organic chemical synthesizers
Oxalic acid makers
Producer gas workers
Steel makers
Tunnel attendants
Water gas workers
Zinc white makers

References

BELL, M. A.: Subacute carbon monoxide poisoning. *Arch. Environ. Health* 3: 108, 1961.

BREYSSE, P. A.: Chronic carbon monoxide poisoning. *Indust. Med. & Surg.* 30: 20, 1961.

DRINKER, C. K.: *Carbon Monoxide Asphyxia.* Oxford University Press, New York, 1938.

HOFREUTER, D. H.; CATCOTT, E. J., AND XINTARAS, C.: Carboxyhemoglobin in men exposed to carbon monoxide. *Arch. Environ. Health* 4: 81, 1962.

KATZ, M.: Chronic carbon monoxide asphyxia, a common clinical entity. *Canad. Med. Assoc. J.* 78: 182, 1958.

PFRENDER, R. E.: Chronic carbon monoxide poisoning. A critical résumé. *Indust. Med. & Surg.* 31: 99, 1962.

VON OETTINGEN, W. F.: Carbon monoxide, its hazards and the mechanism of its action. Pub. Health Bull. No. 290. U.S. Government Printing Office, Washington, D.C., 1944.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

ZORN, O. AND KRUGER, P. D.: The problem of chronic carbon monoxide poisoning. *Indust. Med. & Surg.* 29: 580, 1960.

(40) Carbon Tetrachloride

tetrachloromethane, perchloromethane

Harmful Effects

Local Repeated or prolonged contact with liquid can produce a dry, scaly, fissured dermatitis.

Routes of Entry Ingestion of liquid; inhalation of vapor. Percutaneous absorption of liquid leading to systemic intoxication is unlikely to occur.

Systemic Excessive exposure will result initially in gastrointestinal irritation or central nervous system depression or both. After a few hours to several days following exposure, signs and symptoms of liver and kidney damage may develop. Nausea, vomiting, abdominal pain, diarrhea, enlarged and tender liver, jaundice, and abnormal liver function tests result from toxic hepatitis. Pulmonary and peripheral edema, elevated blood

pressure, diminished urinary volume, abnormal urinalysis, coma, and death may be the consequence of acute renal failure.

Headache, loss of appetite, and lassitude are characteristic of chronic exposure to carbon tetrachloride.

Special Diagnostic Test

Determination of carbon tetrachloride in blood. *See* Von Oettingen, 1958, and Stewart et al., 1960.

Recommended Threshold Limit

10 parts per million parts of air by volume or 65 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Carbon tetrachloride workers
Degreasers
Dry cleaners
Fat processors
Fire extinguisher makers
Fire extinguisher testers
Firemen
Freon makers
Fumigant makers
Fur storage workers
Grain fumigators
Ink makers
Insecticide makers
Laboratory workers, chemical
Lacquerers
Lacquer makers
Lacquer removers
Metal cleaners
Oil processors
Propellant makers
Refrigerant makers
Rotenone extractors
Rubber makers
Seed oil extractors
Semiconductor makers
Solvent workers
Stainers
Stain makers
Type cleaners
Varnish removers
Wax makers

References

LEWIS, C. E.: The toxicology of carbon tetrachloride. *J. Occup. Med.* 3: 82, 1961.

STEWART, R. D.; TORKELSON, T. R.; HAKE, C. L., AND ERLEY, D. S.: Infrared analysis of carbon tetrachloride and ethanol in blood. *J. Lab. & Clin. Med.* 56: 148, 1960.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(41) Carbonyls

See also Nickel and Compounds, and Nickel Carbonyl.

Harmful Effects

Local Contact dermatitis from nickel carbonyl, possibly allergic, has been reported.

Route of Entry Inhalation of vapor.

Systemic In acute intoxications, nickel carbonyl (1) exerts a toxic action on central nervous system with early production of frontal headache, dizziness, nausea, and vomiting and (2) irritates the lungs, producing delayed pneumonitis and pulmonary edema. Retrosternal pain and increased respiratory rate are indications of the delayed pulmonary complications. Metallic taste is occasionally noted. Diarrhea and abdominal distension also occur as a delayed effect. Nickel carbonyl degenerates into metallic nickel and carbon monoxide. The deposition of finely divided nickel within the lungs is thought to be responsible for the pneumonitis and pulmonary edema. The formation of carbon monoxide within the lungs is not thought to be important in the pathogenesis of nickel carbonyl toxicity. Since carbon monoxide is given off from nickel carbonyl in the Mond process of nickel refining, carbon monoxide poisoning may also occur. Allergic bronchial asthma and Loeffler's syndrome have been reported from exposure to nickel carbonyl. Chronic intoxication from nickel carbonyl has not been reported. There is a high incidence of carcinoma of the respiratory tract among nickel refiners, but nickel dust is a more likely carcinogen than nickel carbonyl vapor.

Except for nickel carbonyl no cases of human toxicity to the other existing carbonyls have been reported. Chromium, cobalt, radium, iron, molybdenum, osmium, iridium, rhenium, ruthenium, and tungsten carbonyls exist. Iron and cobalt carbonyls have been shown in animals to be pulmonary irritants. Until further toxicologic information on the lesser known carbonyls becomes available, extreme caution should be exercised with their use.

Special Diagnostic Tests

None, except for analysis of blood and urine for nickel in nickel carbonyl intoxications. *See* Kincaid et al., 1956.

Recommended Threshold Limit

(Nickel carbonyl) 0.001 part per million parts of air by volume or 0.007 milligram per cubic meter of air.

Potential Occupational Exposures

Acetylene welders
Blast welders
Blast furnace workers
Carbonyl workers
Metal refiners
Mond process workers
Nickel refiners
Organic chemical synthesizers
Petroleum refinery workers

References

KINCAID, J. F.; STRONG, J. S., AND SUNDERMAN, F. W.: Toxicity studies of cobalt carbonyls. *A.M.A. Arch. Indust. Hyg. & Occup. Med.*, 10: 210, 1954.

KINCAID, J. F.; STANLEY, E. L.; BECKWORTH, C. H., AND SUNDERMAN, F. W.: Nickel poisoning. 3, Procedures for detection, prevention, and treatment of nickel carbonyl exposure including a method for the determination of nickel in biologic materials. *Am. J. Clin. Path.* 26: 107, 1956.

MORGAN, J. G.: Some observations on the incidence of respiratory cancer in nickel workers. *Brit. J. Indust. Med.* 15: 224, 1958.

SUNDERMAN, F. W. AND KINCAID, J. F.: Nickel poisoning. 2, Studies on patients suffering from acute exposure to vapors of nickel carbonyl. *J. Am. Med. Assoc.* 155: 889, 1954.

SUNDERMAN, F. W.; WEST, B., AND KINCAID, J. F.: A toxicity study of iron pentacarbonyl. *A.M.A. Arch. Indust. Health* 19: 11, 1959.

SUNDERMAN, F. W. AND SUNDERMAN, F. W., JR.: Loeffler's syndrone associated with nickel sensitivity. *Arch. Int. Med.* 107: 405, 1961.

(42) Cellosolve ®

Cellosolve: ethylene glycol monoethyl ether, 2-ethoxyethanol

Cellosolve acetate: ethylene glycol monoethyl ether acetate, 2-ethoxyethyl acetate

Methyl cellosolve: ethylene glycol monomethyl ether, 2-methoxyethanol

Methyl cellosolve acetate: ethylene glycol monomethyl ether acetate, 2-methoxyethyl acetate

Butyl cellosolve: ethylene glycol monobutyl ether, 2-butoxyethanol

Harmful Effects

Local Contact dermatitis from primary irritation. Vapors are mild irritants to conjunctiva and upper respiratory tract.

Route of Entry Inhalation of vapor, and percutaneous absorption of liquid.

Systemic Both neurologic and hematologic effects may be seen in methyl cellosolve intoxication; the former are more pronounced in acute exposures, and the latter are more pronounced in low-grade chronic exposures. Neurologic effects include headache, drowsiness, fatigue, forgetfulness, personality aberrations, dysarthria, disorientation, hyperreflexia, tremors, and ataxia. The most important hematologic effect is depression of red blood cell formation.

One human death resulting from accidental ingestion of methyl cellosolve was reported in 1946. This patient was comatose throughout period of observation. Autopsy revealed severe liver and kidney damage and moderate hemorrhagic gastritis.

Cellosolve, butyl cellosolve, and the cellosolve acetates have not produced systemic intoxication in industry. These compounds have been responsible for central nervous system depression, renal damage, and alterations in blood elements and toxicity in certain laboratory animals. *See* Ethylene Glycol.

Special Diagnostic Test

None.

Recommended Threshold Limit

Cellosolve, 200 parts per million parts of air by volume or 740 milligrams per cubic meter of air.

Cellosolve acetate, 100 parts per million parts of air by volume or 540 milligrams per cubic meter of air.

Methyl cellosolve, 25 parts per million parts of air by volume or 80 milligrams per cubic meter of air.

Methyl cellosolve acetate, 25 parts per million parts of air by volume or 120 milligrams per cubic meter of air.

Butyl cellosolve, 50 parts per million parts of air by volume or 240 milligrams per cubic meter of air.

Potential Occupational Exposures

Cellophane sealers
Cellosolve workers
Cleaning solution makers
Cotton thread makers
Dope makers
Dry cleaners
Dry cleaning agent makers
Dye makers
Enamel makers
Film makers
Gum processors
Hydraulic fluid makers
Insecticide makers
Lacquer makers
Lacquer thinner makers
Leather makers
Nail polish makers
Nitrocellulose makers
Oil processors
Paint makers
Perfume makers
Photographic film makers
Printers
Resin makers
Sludge removing agent makers
Soap makers
Stainers
Stain makers
Textile dyers
Textile printers
Varnish makers
Varnish remover makers
Wax processors
Wood stain makers

References

CARPENTER, C. P.; POZZANI, U. C.; WEIL, C. S.; NAIR, J. H., III; KECK, G. A.; SMYTH, H. F., JR.: The toxicity of butyl cellosolve solvent. *A.M.A. Arch. Indust. Health* 14: 114, 1956.

YOUNG, E. G. AND WOOLNER, L. B.: A case of fatal poisoning from 2-methoxyethanol. *J. Ind. Hyg. & Toxicol* 28: 267, 1946.

ZAVON, M. R.: Methyl cellosolve intoxication. *Am. Indust. Hyg. Assoc. J.* 24: 36, 1963.

(43) Cement, Portland

Harmful Effects

Local Exposure may produce cement dermatitis which is usually due to primary irritation from alkalinity, hygroscopicity, or abrasive property of

cement. In some cases cement workers have developed an allergic sensitivity to constituents of cement such as hexavalent chromate. It is not unusual for cement dermatitis to be prolonged and to involve covered areas of body.

Route of Entry Inhalation of dust.

Systemic No systemic manifestations attributable to cement exposure have been reported.

Special Diagnostic Test

None.

Recommended Threshold Limit

50 million particles per cubic foot of air.

Potential Occupational Exposures

Asbestos cement pipe makers
Asbestos cement sheet makers
Asbestos cement shingle makers
Barge builders
Brick masons
Bridge builders
Building construction workers
Burial vault builders
Cement insulation makers
Cement insulation workers
Cement makers
Cement pipe makers
Cement workers
Concrete runway builders
Dam builders
Drain tile makers
Heat insulation makers
Oil well builders
Pier builders
Post makers
Reservoir builders
Road construction workers
Sidewalk builders
Silo builders
Smokestack builders
Sound insulation makers
Stadium builders
Storage tank builders
Swimming pool builders
Tunnel builders
Water pipe makers

References

CALNAN, C.: Cement dermatitis. *J. Occup. Med.* 2: 15, 1960.

MORRIS, G. E.: The primary irritant nature of cement. *Arch. Environ. Health* 1: 301, 1960.

SANDER, O. A.: Roentgen resurvey of cement workers.. *A.M.A. Arch. Indust. Health* 17: 96, 1958.

(44) Cerium

Harmful Effects

Local None reported.

Route of Entry Inhalation of dust.

Systemic No cases of industrial poisoning have been found in literature. Cerium and its salts have produced alterations in blood elements in certain experimental animals.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Alloy makers
Ammonia makers
Cerium workers
Enamel makers, vitreous
Explosive makers
Glass makers
Glass polish makers
Ink makers
Lighter flint makers
Metal refiners
Phosphor makers
Photographic illuminant makers
Rocket fuel makers
Textile workers

(45) Chlordane. *See* Pesticides Section

(46) Chloride of Lime

chlorinated lime, bleaching powder

Chloride of lime is a mixture of calcium chloride, calcium hypochlorite and calcium hydroxide.

Harmful Effects

Local The powder and its solutions have corrosive action on skin, eyes and mucous membranes and can produce conjunctivitis, blepharitis, corneal ulceration, gingivitis, and contact dermatitis.

Route of Entry Inhalation of dust.

Systemic Dust is irritating to respiratory tract, and can produce laryngitis and pulmonary edema.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Chloride of lime workers
Deodorant makers
Disinfectant makers
Dyers
Laundry workers
Oil bleachers
Organic chemical synthesizers
Paper makers
Sewage treaters
Soap bleachers
Straw bleachers
Textile bleachers
Textile printers
Water treaters
Wood pulp bleachers

(47) Chlorinated Benzenes

Chlorobenzene: phenyl chloride, monochlorobenzene, chlorobenzol
o-Dichlorobenzene: 1,2-dichlorobenzene
p-Dichlorobenzene: 1,4-dichlorobenzene
Trichlorobenzenes: 1,2,4-trichlorobenzene; 1,3,5-trichlorobenzene
Hexachlorobenzene: perchlorobenzene

Harmful Effects

Local Chlorinated benzenes are irritating to skin, conjunctiva, and mucous membranes of upper respiratory tract.

Routes of Entry Inhalation of vapor, percutaneous aborption of liquid. Percutaneous route is of little importance when contact is with solid p-dichlorobenzene.

Systemic Studies of industrial populations exposed to o-dichlorobenzene and p-dichlorobenzene reveal no significant systemic effects. Liver injury and cataracts have been reported with high exposures to certain of these compounds.

Special Diagnostic Test

None.

Recommended Threshold Limit

Chlorobenzene, 75 parts per million parts of air by volume or 350 milligrams per cubic meter of air.

o-Dichorobenzene, 50 parts per million parts of air by volume or 300 milligrams per cubic meter of air.

p-Dichlorobenzene, 75 parts per million parts of air by volume or 450 milligrams per cubic meter of air.

Potential Occupational Exposures

Chlorobenzene
- Aniline makers
- Carbolic acid makers
- Cellulose acetate workers
- Chlorobenzene workers
- DDT makers
- Drug makers
- Dry cleaners
- Dyers
- Ethyl cellulose workers
- Heat transfer workers
- Ink makers
- Lacquerers
- Lacquer makers
- Organic chemical synthesizers
- Paint workers
- Perfume makers
- Picric acid makers
- Resin makers
- Rubber makers
- Sulfur dye makers
- Varnish makers

o-Dichlorobenzene
- Asphalt makers
- Cleaning compound makers
- Deodorant makers

o-Dichlorobenzene workers
Dry cleaners
Dye makers
Fumigant workers
Greasemakers
Gum makers
Heat transfer workers
Hide processors
Insecticide workers
Lacquerers
Lacquer workers
Metal degreasers
Metal polish makers
Organic chemical synthesizers
Paint remover workers
Paint workers
Polishing compound makers
Resin makers
Rubber makers
Solvent workers
Stainers
Stain makers
Sulfur processors
Tannery workers
Tar makers
Tar remover workers
Termite exterminator workers
Varnish makers
Varnish remover workers
Wax makers
Wood preservative workers
Wool processors

p-Dichlorobenzene
Deodorant makers
p-Dichlorobenzene workers
Disinfectant workers
Drug makers
Dye makers
Insecticide workers
Moth ball makers
Soil fumigators

Trichlorobenzenes
Dye makers
Electric equipment makers
Heat transfer workers
Insecticide workers
Lubricant makers
Trichlorobenzene workers

Hexachlorobenzene
Fungicide workers
Hexachlorobenzene workers
Organic chemical synthesizers
Seed disinfectors

References

HOLLINGSWORTH, R. L.; ROWE, V. K.; OYEN, F.; HOYLE, H. R., AND SPENCER, H. C.: Toxicity of paradichlorobenzene; determinations on experimental animals and human subjects. *A.M.A. Arch. Indust. Health* 14: 138, 1956.

HOLLINGSWORTH, R. L.; ROWE, V. K.; OYEN, F.; TORKELSON, T. R., AND ADAMS, E. M.: Toxicity of o-dichlorobenzene; studies on animals and industrial experience. *A.M.A. Arch. Indust. Health* 17: 180, 1958.

(48) Chlorinated Diphenyls and Naphthalenes

Harmful Effects

Local Prolonged contact with fume or the cold wax leads on exposed skin to comedones, sebaceous cysts and pustules, known as chloracne.

Routes of Entry Inhalation of fume or vapor; percutaneous absorption of liquid.

Systemic Acute or chronic exposure can produce varying degrees of liver damage depending on amount of chlorine in compound and preexisting

state of liver. Symptoms include jaundice, anorexia, nausea, indigestion, abdominal pains, and edema. Death from acute yellow atrophy of liver has occurred.

Special Diagnostic Test

None.

Recommended Threshold Limit

Chlorodiphenyl (42 percent chlorine), 1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Chlorodiphenyl (54 percent chlorine), 0.5 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Chlorinated diphenyl oxide, 0.5 milligram per cubic meter of air.

Pentachloronaphthalene, 0.5 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Trichloronaphthalene, 5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Aniline dye makers
Cable coaters
Carbon removers
Chlorinated diphenyl workers
Condenser impregnators
Crankcase oil additive makers
Dye makers
Electric equipment makers
Electricians
Electroplaters
Flameproofers
Gum processors
Heat transfer workers
Herbicide workers
Ink makers
Insecticide workers
Insect proofers
Lacquerers
Lacquer makers
Light fixture makers
Machinists
Metal degreasers
Mineral oil processors
Moisture proofers
Paint makers
Paper treaters
Petroleum refinery workers
Plasticizer makers
Plastic makers
Rayon makers
Resin makers
Rubber workers
Solvent workers
Stainers
Stain makers
Textile flameproofers
Transformer workers
Upper cylinder oil makers
Varnish makers
Vegetable oil processors
Wax makers
Wire coaters
Wood preservers

Reference

MEIGS, J. W.; ALBOM, J. J.; AND KARTIN, B. L.: Chloracne from an unusual exposure to arochlor. *J. Am. Med. Assoc.* 154: 1417, 1954

(49) Chlorine

Harmful Effects

Local Extreme irritation of skin, eyes, and mucous membranes; corrosion of teeth.

Route of Entry Inhalation of gas.

Systemic Acute respiratory distress including cough, hemoptysis, chest pain, dyspnea, and cyanosis. Later, tracheobronchitis, bronchopneumonia, and pulmonary edema may supervene.

Special Diagnostic Test

None.

Recommended Threshold Limit

1 part per million parts of air by volume or 3 milligrams per cubic meter of air.

Potential Occupational Exposures

Aerosol propellant makers
Alkali salt makers
Aluminum purifiers
Benzene hexachloride makers
Bleachers
Bleaching powder makers
Bromine makers
Broom makers
Carpet makers
Chemical synthesizers
Chloride of lime makers
Chlorinated solvent makers
Chlorine workers
Color makers
DDT makers
Disinfectant makers
Dye makers
Ethylene glycol makers
Ethylene oxide makers
Flour bleachers
Freon makers
Gasoline additive workers
Gold extractors
Ink makers
Iodine makers
Iron detinners
Iron dezinkers
Laundry workers
Methyl chloride makers
Paper bleachers
Petroleum refinery workers
Phosgene makers
Photographic workers
Pulp bleachers
Rayon makers
Refrigerant makers
Rubber makers
Sewage treaters
Silver extractors
Sodium hydroxide makers
Submarine workers
Sugar refiners
Sulfur chloride makers
Swimming pool maintenance workers
Tetraethyl lead makers
Textile bleachers
Tin recovery workers
Toxaphene makers
Vinyl chloride makers
Vinylidene chloride makers
Water treaters
Zinc chloride makers

References

CHASIS, H.; ZAPP, J. A.; BANNON, J. H.; WHITTENBERGER, J. L.; HELM, J.; DOHENY, J. J., AND MACLEOD, C. M.: Chlorine accident in Brooklyn. *Occup. Med.* 4: 152, 1947.

JOYNER, R. E., AND DURIEL, E. G.: Accidental liquid chlorine spill in a rural community. *J. Occup. Med.* 4: 152, 1962.

(50) Chloroprene

chlorobutadiene

Harmful Effects

Local May cause contact dermatitis from primary skin irritation; may produce mucous membrane irritation and temporary hair loss.

Routes of Entry Percutaneous absorption; inhalation of vapor.

Systemic Chloroprene may cause lung irritation, liver and kidney damage, and central nervous system depression.

Special Diagnostic Test

None.

Recommended Threshold Limit

25 parts per million parts of air by volume or 90 milligrams per cubic meter of air.

Potential Occupational Exposures

Chloroprene workers

Rubber makers, neoprene

References

RITTER, W. L. AND CARTER, A. S.: Hair loss in neoprene manufacture. *J. Indust. Hyg. & Toxicol.* 30: 192, 1948.

VON OETTINGEN, W. F.; HUEPER, W. C.; DEICHMANN-GRUEBLER, W., AND WILEY, F. H.: 2-Chloro-butadiene (chloroprene); its toxicity and pathology, and the mechanism of its action. *J. Indust. Hyg. & Toxicol.* 18: 240, 1936.

(51) Chromium Compounds

Chromium compounds include chromic acid (chromic trioxide), chromates, and bichromates.

Harmful Effects

Local Contact with chromates or chromic acid can produce small, painless cutaneous ulcers as well as dermatitis from primary irritation or allergic hypersensitivity. Cutaneous allergy is not uncommon from hexavalent chromium compounds but is extremely rare from trivalent chromium compounds. Yellowish discoloration of teeth and tongue; perforation of nasal septum; conjunctivitis.

Route of Entry Inhalation of dust or mist.

Systemic Allergic bronchial asthma from chromium trioxide fume. Bronchogenic carcinoma has occurred at an abnormally high rate among chromate workers. The carcinogenic form of chromium has not been determined.

Special Diagnostic Test

Determination of chromium in blood and urine. *See* Division of Occupational Health, 1953.

Recommended Threshold Limit

Chromic acid and chromates (as CrO_3), 0.1 milligram per cubic meter of air.

Potential Occupational Exposures

Abrasive makers
Acetylene purifiers
Adhesive workers
Airplane sprayers
Alizarin makers
Alloy makers
Aluminum anodizers
Aniline black makers
Anodizers
Battery makers, dry
Biologists
Blue print makers
Boiler scalers
Candle makers, colored
Cement workers
Ceramic workers
Chromate workers
Chrome alloy workers
Chrome alum workers
Chromium platers
Chromium workers
Color makers
Copper etchers
Copper plate strippers
Corrosion inhibitor workers
Crayon makers, colored
Diesel locomotive repairmen
Drug makers
Dry color makers
Dye makers
Dyers
Electroplaters
Enameler workers
Explosive makers
Fat purifiers
Fireworks makers
Fly paper makers
Furniture polishers
Fur processors
Glass fiber makers
Glass frosters
Glass makers
Glass makers, colored
Glue makers
Histology technicians
Ink makers
Jewelers
Laboratory workers, chemical
Leather finishers
Linoleum workers
Lithographers
Magnesium treaters
Match makers
Metal cleaners
Metal cutters
Metal etchers
Metal treaters
Milk preservers
Mordanters
Oil drillers
Oil purifiers
Organic chemical synthesizers

218-695 O-66-9

Painters
Paint makers
Palm oil bleachers
Paper dyers
Paper waterproofers
Pencil makers, colored
Perfume makers
Photoengravers
Photographers
Photographic chemical makers
Pigment makers
Platinum polishers
Porcelain decorators
Pottery frosters
Pottery glaze makers
Pottery glazers
Printers
Printing ink workers
Process engravers
Pyrotechnic workers
Railroad engineers
Refractory brick makers
Rubber makers
Rust inhibitor workers
Shingle makers
Silk screen makers
Smokeless powder makers
Soap makers
Sponge bleachers
Stainless steel workers
Tanners
Textile dyers
Textile mordanters
Textile printers
Textile waterproofers
Wallpaper printers
Wax bleachers
Wax ornament workers
Welders
Wood preservative workers
Wood stainers
Wood stain makers

References

BAETJER, A. M.: Pulmonary carcinoma in chromate workers. 1, A review of the literature and report of cases. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 2: 487, 1950.

BERNHARDT, H. J.: Chromate dermatitis; its natural history and treatment. *A.M.A. Arch. Dermat.* 76: 13, 1957.

DENTON, C. R.; KEENAN, R. G., AND BIRMINGHAM, D. J.: The chromium content of cement and its significance in cement dermatitis. *J. Invest. Dermat.* 23: 189, 1954.

DIVISION OF OCCUPATIONAL HEALTH, PUBLIC HEALTH SERVICE: Health of workers in chromate producing industry. Pub. Health Service Pub. No. 192. U.S. Government Printing Office, Washington, D.C., 1953.

MANCUSO, T. F. AND HUEPER, W. C.: Occupational cancer and other health hazards in a chromate plant; a medical appraisal. 1, Lung cancer in chromate workers. *Indust. Med. & Surg.* 20: 358, 1951.

MANCUSO, T. F.: Occupational cancer and other health hazards in a chromate plant; a medical appraisal. 2, Clinical and toxicologic aspects. *Indust. Med. & Surg.* 20: 393, 1951.

WINSTON, J. R. AND WALSH, E. N.: Chromate dermatitis in railroad employees working with diesel locomotives. *J. Am. Med. Assoc.* 147: 1133, 1951.

(52) Coal Tar and Fractions

Coal tar, derived from destructive distillation of coal during manufacture of coke and illuminating gas, can be divided by distillation into several crude fractions: crude naphtha, creosote oil, anthracene oil and pitch. For specific constituents see Acridine, Benzene, Cresol, Naphtha, Naphthalenes (Chlorinated Diphenyls and Naphthalenes), Phenol, and Toluene.

Harmful Effects

Local Photosensitization may occur and is manifested by erythema, edema, burning, and subsequent hyperpigmentation of exposed areas. Other cutaneous effects include folliculitis, acne, and comedones; keratoses, papillomas, and squamous cell epitheliomas following years of exposure; contact dermatitis from either primary irritation or allergic hypersensitivity; and conjunctivitis.

Route of Entry Inhalation of dust or vapor.

Systemic Overexposure to vapor produces anorexia, nausea, vomiting, and cough. Bronchogenic carcinoma has been suspected from inhalation of coal tar vapors and dust in Great Britain, Canada, and Japan.

Special Diagnostic Tests

Examination of skin under Wood's light for fluorescence of residual tar. Histologic examination of skin biopsy for malignancy.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Artificial stone makers
Asbestos goods workers
Asphalt workers
Battery box makers
Battery workers, dry
Boat builders
Brick masons
Brick pressers
Brickyard workers
Briquette makers
Brush makers
Cable makers
Carpenters
Coal tar still cleaners
Coal tar workers
Coke oven workers
Corkstone makers
Creosoters
Diesel engine engineers
Electric equipment makers
Electricians
Electrode makers
Electrometallurgic workers
Farmers
Fishermen
Flue cleaners
Fuel pitch workers
Furnace men
Gas house workers
Glass blowers
Impregnated felt makers
Insecticide bomb makers
Insulation board makers
Insulators
Lens grinders
Linemen
Miners
Painters
Paper conduit makers
Pavers
Pipeline workers
Pipe pressers
Pitch workers
Railroad track workers
Riveters
Road workers
Roofers
Roofing paper workers
Rope makers
Rubber workers

Shingle makers
Shipyard workers
Soap makers
Smokeless fuel makers
Stokers
Tar paint makers
Tile pressers
Waterproof concrete workers
Waterproofers
Wood preservers

References

DOLL, R.: Occupational lung cancer; a review. *Brit. J. Indust. Med.* 16: 181, 1959.
FISHER, R. E. W.: Skin cancer in tar workers. *Trans. Assoc. Indust. Med. Officers* 3: 315, 1954.

(53) Cobalt and Compounds

Harmful Effects

Local Metallic cobalt dust and cobalt salts may produce allergic contact dermatitis. Dust of certain cobalt ores may irritate cornea.

Route of Entry Inhalation of dust.

Systemic Bronchial asthma from inhalation of cobalt dust has been suspected. Inhalation of anyhdrous cobalt acetate dust has produced gastrointestinal irritation. A pneumoconiosis of tungsten carbide workers, manifested by cough and dyspnea, has been reported.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Cobalt) 0.5 milligram per cubic meter of air.

Potential Occupational Exposures

Acetic acid makers
Actors
Alloy makers
Alnico magnet makers
Ammonia mask makers
Barometer makers
Bright platers
Catalyst workers
Cement makers
Cemented (tungsten) carbide workers
Ceramic workers
Cermet makers
Cobalt soap makers
Cobalt workers
Cosmetic makers
Drug makers
Electroplaters
Enamelers
Ethyl acrylate makers
Fertilizer workers
Frit workers
Gas mask makers
Gasoline blenders
Glass colorers
Glaze workers
High speed tool steel workers
Hygrometer makers
Ink makers, sympathetic
Iron cobalt-platers
Lacquer dryer makers
Lacquer makers
Lamp filament makers
Magnet steel workers

Metallurgists
Mineral feed makers
Moisture indicator makers
Nickel workers
Nuclear technologists
Oilcloth color workers
Oil dryer makers
Oil hydrogenators
Oil pigment makers
Paint dryer makers
Paint makers
Phthalic anhydride makers
Polyester resin workers
Porcelain colorers
Porcelain enamel workers
Pottery glazers
Rubber colorers
Varnish dryer makers
Varnish makers

References

FAIRHALL, L. T.; KEENAN, R. G., AND BRINTON, H. P.: Cobalt and the dust environment of the cemented tungsten carbide industry. *Pub. Health Rep.* 64: 485, 1949.

MILLER, C. W.; DAVIS, M. W.; GOLDMAN, A., AND WYATT, J. P.: Pneumoconiosis in the tungsten-carbide tool industry. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 453, 1953.

(54) Copper and Compounds

Harmful Effects

Local The following copper salts have been reported to be skin and mucous membrane irritants: copper arsenite, copper cyanide, copper fluoride, copper naphthenate, copper oxide, and copper sulfate.

Route of Entry Inhalation of dust or fume.

Systemic Inhalation of copper fumes has been reported to produce signs and symptoms of metal fume fever. These include chills, transient fever, nausea, thirst, and exhaustion. Prolonged inhalation of copper salts may cause perforation of nasal septum.

Special Diagnostic Tests

Examination of blood and urine for excessive amounts of copper. *See* Stewart and Stolman, 1960.

Recommended Threshold Limits

(Tentative) Copper (fume), 0.1 milligram per cubic meter of air; copper (dusts, mists), 1 milligram per cubic meter of air.

Potential Occupational Exposures

Antifouling paint makers
Asphalt makers
Battery makers
Brass founders
Canvas preservative workers
Copper founders
Copper platers
Copper refiners
Copper smelters
Coppersmiths
Copper workers
Electroplaters
Enamel workers
Flotation workers
Fungicide workers
Gem colorers

Glass makers
Glue makers
Hair dye workers
Hide preservative workers
Ink makers
Insecticide workers
Lithographers
Organic chemical synthesizers
Paint makers
Petroleum refinery workers
Pigment makers
Propeller polishers
Railroad tie preservative workers
Rayon makers
Refrigerator makers
Rope preservative workers
Rubber makers
Solderers
Steel makers
Tanners
Textile dyers
Textile makers
Wallpaper makers
Water treaters
Wood preservative workers

References

DAVENPORT, S. J.: Review of literature on health hazards of metals. 1, Copper. Bureau of Mines Information Circular 7666. U.S. Department of Interior, Washington, D.C., 1953.

STEWART, C. P. AND STOLMAN, A.: *Toxicology; Mechanisms and Analytical Methods.* Vol. 1. Academic Press, New York, 1960.

(55) Cresol

cresylic acid, cresylol, hydroxytoluene, methyl phenol, oxytoluene, tricresol

Cresol is a mixture of the three isomeric cresols: ortho-, meta-, and para-.

Harmful Effects

Local Cresol, a potent primary irritant, has a corrosive action on skin and mucous membranes. Intense irritation is produced upon contact with eye.

Routes of Entry Inhalation or percutaneous absorption of liquid or vapor.

Systemic Inhalation of vapor may cause pulmonary edema. Severe poisoning is followed by collapse, hypothermia, and death. Nonfatal poisoning may be followed by severe liver and kidney damage which appear after a period of apparent full recovery.

Special Diagnostic Test

None.

Recommended Threshold Limit

(All isomers), 5 parts per million parts of air by volume or 22 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Coal tar workers
Cresol soap makers
Cresol workers
Cresylic acid makers
Deodorant workers
Disinfectant makers
Disinfectors
Dye makers
Enamel makers
Explosive workers
Flotation agent makers
Flotation workers
Foundry workers
Glue workers
Ink makers
Ink remover makers
Ink removers
Insecticide workers
Insulation enamel workers
Oil additive makers
Paint remover makers
Paint removers
Perfume makers
Photographic developer workers
Pitch workers
Resin makers
Roofers
Rubber makers
Scouring compound makers
Stainers
Stain makers
Surfactant makers
Tar distillery workers
Textile sizers
Varnish remover makers
Varnish removers
Veterinarians
Wool scourers

Reference

FAIRHALL, L. T.: *Industrial Toxicology.* 2nd ed. Williams & Wilkins Co., Baltimore, 1957.

(56) Cyanides. *See* Hydrogen Cyanide

(57) Cyclohexane. *See* Cycloparaffins

(58) Cyclohexene. *See* Cycloparaffins

(59) Cycloparaffins

cycloalkanes; included in this classification are cyclohexane or hexahydrobenzene, and cyclohexene or 1,2,3,4-tetrahydrobenzene

Harmful Effects

Local Eye irritation and dry, scaly, fissured dermatitis can be produced by contact with liquid.

Route of Entry Inhalation of vapor.

Systemic Cycloparaffins are weakly narcotic; in high concentrations may produce headache, dizziness, nausea, vomiting, and unconsciousness.

Special Diagnostic Test

None.

Recommended Threshold Limit

Cyclohexane, 400 parts per million parts of air by volume or 1,400 milligrams per cubic meter of air.

Cyclohexene, 400 parts per million parts of air by volume or 1,350 milligrams per cubic meter of air.

Potential Occupational Exposures

Adipic acid makers
Benzene makers
Bitumen processors
Cellulose plastic makers
Cycloparaffin workers
Essential oil extractors
Fat processors
Fungicide makers
Lacquerers
Lacquer makers
Maleic acid makers
Nylon makers
Oil processors
Organic chemical synthesizers
Paint remover makers
Paint removers
Perfume makers
Plastic molders
Resin makers
Rubber makers
Solid fuel makers, camp stove
Varnish remover makers
Varnish removers
Wax makers

(60) DDT. *See* Pesticides Section

(61) Diacetone Alcohol

diacetone, diacetonyl alcohol, dimethylacetonyl carbinol

Harmful Effects

Local Irritation of eyes, nose, and throat by high vapor concentrations.
Route of Entry Inhalation of vapor.
Systemic Effects have not been described for man. In experimental animals there may be somnolence, narcosis, hypotension, transient anemia, and kidney damage.

Special Diagnostic Test

None.

Recommended Threshold Limit

50 parts per million parts of air by volume or 240 milligrams per cubic meter of air.

Potential Occupational Exposures

Animal tissue preservers
Antifreeze makers
Celluloid cement makers
Cellulose acetate workers
Cellulose ester lacquer makers
Cellulose nitrate workers

Diacetone alcohol workers
Dope workers
Drug makers
Dye makers
Fat processors
Garage mechanics
Gold leaf makers
Hydraulic brake fluid makers
Ink makers, quick drying
Lacquerers
Lacquer makers
Leather makers
Metal cleaners
Nitrocellulose workers
Oil processors
Paint remover makers
Paint removers
Paper coaters
Printers
Rayon makers
Resin makers
Solvent workers
Stainers
Stain makers
Tar processors
Textile workers
Wax makers
Wood preservative workers

Reference

VON OETTINGEN, W. F.: The aliphatic alcohols; their toxicity and and potential dangers in relation to their chemical constitution and their fate in metabolism. Pub. Health Bull. No. 281. U.S. Government Printing Office, Washington, D.C., 1943.

(62) 1,2-Dichloroethylene

acetylene dichloride

Harmful Effects

Local The solvent can act as primary irritant producing contact dermatitis. Vapor can cause irritation of mucous membranes of upper respiratory tract.

Route of Entry Inhalation of vapor.

Systemic Transient narcosis can result from inhalation of vapor. No chronic toxicity in man has been reported.

Special Diagnostic Test

None.

Recommended Threshold Limit

200 parts per million parts of air by volume or 790 milligrams per cubic meter of air.

Potential Occupational Exposures

Camphor processors
Carbolic acid processors
Cellulose acetate workers
Dichloroethylene workers
Drug makers
Dry cleaners
Dye makers
Fat processors
Gum processors
Lacquerers
Lacquer makers
Oil processors

Organic chemical synthesizers
Perfume makers
Plastic makers
Resin makers
Rubber makers
Shellac processors
Solvent workers
Wax makers

Reference

MCBIRNEY, R. S.: Trichloroethylene and dichloroethylene poisoning. ***A.M.A. Arch. Indust. Hyg. & Occup. Med.*** 10: 130, 1954.

(63) Dichloroethyl Ether

dichloroether, dichloroethyl oxide

Harmful Effects

Local Irritation of conjunctiva and mucous membranes of upper respiratory tract.

Route of Entry Inhalation of vapor.

Systemic No cases of industrial systemic intoxication have been reported. Animal studies indicate that vapor is an intense respiratory tract irritant causing pulmonary edema.

Special Diagnostic Test

None.

Recommended Threshold Limit

15 parts per million parts of air by volume or 90 milligrams per cubic meter of air.

Potential Occupational Exposures

Degreasers
Dichloroethyl ether workers
Dry cleaners
Ethyl cellulose processors
Fat processors
Finish remover makers
Fulling compound makers
Gum processors
Lacquer makers
Oil processors
Oil purifiers
Organic chemical synthesizers
Paint makers
Pectin processors
Resin makers
Soap makers
Soil fumigant workers
Soil fumigators
Solvent workers
Stain removers
Tar processors
Textile scourers
Varnish workers

Reference

SCHRENK, H. H.; PATTY, F. A., AND YANT, W. P.: Acute response of guinea pigs to vapors of some new commercial organic compounds. 7, Dichloroethyl ether. ***Pub. Health Rep.*** 48: 1389, 1933. Reprint no. 1602.

(64) Dieldrin. *See* Pesticides Section

(65) Dimethylformamide

the "universal organic solvent", DMF

Harmful Effects

Local Highly irritating to skin, eyes, and mucous membranes.

Route of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic DMF causes gastric irritation with anorexia, nausea, vomiting, epigastric burning, and abdominal pain and tenderness; severe liver and kidney damage reported in experimentally exposed animals.

Special Diagnostic Test

None.

Recommended Threshold Limit

20 parts per million parts of air by volume or 60 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetylene purifiers
Butadiene makers
Dimethylformamide workers
Drug makers
Dye makers
Lubricating oil extractors
Organic chemical synthesizers
Petroleum refinery workers
Resin makers
Solvent workers
Synthetic fiber makers

Reference

MASSMANN, W.: Toxicological investigations on dimethylformamide. *Brit. J. Indust. Med.* 13: 51, 1956.

(66) Dimethylhydrazine

UDMH, 1,1-dimethylhydrazine, asymmetrical dimethylhydrazine

Harmful Effects

Local Liquid is low grade primary irritant of skin. Liquid and vapor are irritating to eyes.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Systemic effects include delayed gastrointestinal irritation, hemolytic anemia, and possible liver damage. Vapor produces irritation of respiratory tract. On the basis of animal experiments, UDMH may cause convulsions and kidney damage.

Special Diagnostic Test

None.

Recommended Threshold Limit

0.5 part per million parts of air by volume or 1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Jet fuel handlers	Photographic developer makers
Jet fuel makers	Rocket fuel handlers
Organic chemical synthesizers	Rocket fuel makers

References

JACOBSON, K. H.; CLEM, J. H.; WHEELWRIGHT, H. J., JR.; RINEHART, W. E., AND MAYES, N.: The acute toxicity of the vapors of some methylated hydrazine derivatives. *A.M.A. Arch. Indust. Health* 12: 609, 1955.

JACOBSON, K. H.: Industrial hygiene aspects of liquid propellants. *Transactions, 22nd annual meeting, American Conference of Governmental Industrial Hygienists, 1960.* Sec.-Treas., 1014 Broadway, Cincinnati 2, Ohio.

OFFICE OF DIRECTOR, DEFENSE RESEARCH AND ENGINEERING, DEPARTMENT OF DEFENSE: *The Handling and Storage of Liquid Propellants.* U.S. Government Printing Office, Washington, D.C., 1961.

SHOOK, B. S., SR., AND COWART, O. H.: Health hazards associated with unsymmetrical dimethylhydrazine. *Indust. Med. & Surg.* 26: 333, 1957.

(67) Dimethyl Sulfate

methyl sulfate

Harmful Effects

Local Liquid or vapor produces vesiculation and analgesia on contact with skin. Analgesia may persist for several months thereafter. Mucous membranes are irritated by liquid or vapor, with production of conjunctivitis, lacrimation, corneal ulcerations, rhinitis, edema of mucosa of mouth and throat, dysphagia, sore throat, and hoarseness. Irritation of skin and mucous membranes may be delayed in appearance.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Irritation of lungs produces bronchitis, pneumonitis, and pulmonary edema. Absorption is followed by cerebral edema and central nervous system effects such as drowsiness, paralysis, convulsions, delirium, and coma. Absorption may also produce liver and kidney damage.

Special Diagnostic Test

None.

Recommended Threshold Limit

1 part per million parts of air by volume or 5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Amine makers
Color makers
Drug makers
Dye makers
Methylation workers
Organic chemical synthesizers
Perfume makers
Phenol derivative makers

References

GAULTIER, M.; FOURNIER, E.; GERVAIS, P.; GORCEIX, A., AND EFTHYMIOU, T.: Two cases of methyl sulfate poisoning. *Arch. Mal. Prof.* 21: 744, 1960. (*Indust. Hyg. Digest,* Abst. No. 987, October 1961.)

HASWELL, R. W.: Dimethyl sulfate poisoning by inhalation. *J. Occup. Med.* 2: 454, 1960.

LITTLER, T. R. AND MCCONNELL, R. B.: Dimethyl sulfate poisoning. *Brit. J. Indust. Med.* 12: 54, 1955.

(68) Dinitrobenzene

Dinitrobenzol; meta-, ortho- and para-isomers

Harmful Effects

Local Dinitrobenzene is a primary skin irritant.

Routes of Entry Percutaneous absorption of liquid; inhalation of vapor.

Systemic Systemic effects are similar to Nitrobenzene (which see).

Special Diagnostic Tests

Analysis of urine for dinitrobenzene and blood for methemoglobin. *See* Von Oettingen, 1958, and Stewart and Stolman, 1961.

Recommended Threshold Limit

1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Celluloid makers
Dinitrobenzene workers
Dye makers
Explosive makers
Explosive users
Organic chemical synthesizers
Plastic makers

References

BERITIC, T.: Two cases of meta-dinitrobenzene poisoning with unequal clinical response. *Brit. J. Indust. Med.* 13: 114, 1956.

STEWART, C. P. AND STOLMAN, A.: *Toxicology; Mechanisms and Analytical Methods.* Vol. 2. Academic Press, New York, 1961.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(69) Dinitrophenol

Of the 6 isomers the 2,4- is the most toxic.

Harmful Effects

Local Yellow staining of skin. Eczematous dermatitis due to either primary irritation or allergic hypersensitivity. Exfoliative dermatitis has occurred.

Routes of Entry Percutaneous absorption from dust; inhalation of dust; to a lesser extent, ingestion.

Systemic Dinitrophenol blocks oxidative phosphorylation and thereby stimulates basal metabolism with resultant effects of anorexia, nausea, vomiting, sweating, thirst, dyspnea, excitement, tachycardia, and fever. Acidosis may develop. Central nervous system effects are those of stimulation followed by depression. There may be cataract formation, kidney or liver damage. Death may result from overwhelming exposure.

Special Diagnostic Test

Detection of dinitrophenol and aminonitrophenol in urine. *See* Von Oettingen, 1958.

Recommended Threshold Limit

0.2 milligram per cubic meter of air. *See* American Industrial Hygiene Association, 1958.

Potential Occupational Exposures

Diaminophenol makers
Dinitrophenol workers
Dye makers
Explosive workers
Herbicide workers
Indicator makers, chemical
Organic chemical synthesizers
Photographic developer makers
Wood preservative workers

References

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION: 2,4-Dinitrophenol. *Hygienic Guide Series.* The Association, Detroit, 1958. Recommended threshold limit: 0.2 milligram per cubic meter of air.

GISCLARD, J. B. AND WOODWARD, M. M.: 2,4-Dinitrophenol poisoning; a case report. *J. Indust. Hyg. & Toxicol.* 28: 47, 1946.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(70) Dinitrotoluene

dinitrotoluol, DNT

Harmful Effects

Local Contact may produce allergic hypersensitization.

Routes of Entry Percutaneous absorption of liquid; inhalation of vapor.
Systemic Symptoms and signs are similar to intoxication from trinitrotoluene. *See* Trinitrotoluene.

Special Diagnostic Test

None.

Recommended Threshold Limit

1.5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Dinitrotoluene workers
Dye makers
Explosive workers
Organic chemical synthesizers

(71) Dioxane

1,4-diethylene dioxide, diethylene ether

Harmful Effects

Local Irritation of eyes, nose and throat.
Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.
Systemic Severe gastric symptoms. Liver necrosis and nephritis.

Special Diagnostic Test

None.

Recommended Threshold Limit

100 parts per million parts of air by volume or 360 milligrams per cubic meter of air.

Potential Occupational Exposures

Adhesive workers
Cellulose acetate workers
Cellulose ester workers
Cement workers
Cosmetic makers
Degreasers
Deodorant makers
Detergent workers
Dioxane workers
Dye makers
Emulsion makers
Fat processors
Fumigant workers
Glue makers
Histology technicians
Lacquerers
Lacquer makers
Metal cleaners
Oil processors
Painters
Paint makers
Paint removers
Paint remover workers
Plastic makers
Polish makers
Printers
Resin makers
Shoe cream makers
Solvent workers
Stainers

Stain makers
Textile makers
Varnish makers
Varnish remover makers
Varnish removers

References

JOHNSTONE, R. T.: Death due to dioxane? *A.M.A. Arch. Indust. Health* 20: 445, 1959.

LEHMANN, K. B. AND FLURY, F. (EDITORS); KING, E. AND SMYTH, H. F., JR. (TRANSLATORS): *Toxicology and Hygiene of Industrial Solvents.* Williams & Wilkins Co., Baltimore, 1943.

(72) Epichlorohydrin

epi, chloropropylene oxide

Harmful Effects

Local Liquid and vapor are highly irritating to skin, eyes, and upper respiratory tract. Cutaneous burns may be delayed in appearance. Allergic eczematous contact dermatitis occurs occasionally.

Route of Entry Inhalation of vapor.

Systemic On the basis of animal experiments, epichlorohydrin is highly irritating to lungs, and may damage liver and kidneys. No cases of pulmonary injury or systemic intoxication have been reported in man.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Tentative) 5 parts per million parts of air by volume.

Potential Occupational Exposures

Cellulose ether workers
Epichlorohydrin workers
Epoxy resin makers
Glycerol derivative makers
Glycerophosphoric acid makers
Glycidol derivative makers
Gum processors
Lacquerers
Lacquer makers
Nail enamel makers
Organic chemical synthesizers
Painters
Paint makers
Solvent workers
Varnish makers

Reference

ANON.: Epichlorohydrin. Toxicity Data Sheet, Industrial Hygiene Bulletin. Shell Chemical Corp., New York, 1959.

(73) Ethyl Acetate

acetic ether, vinegar naphtha

Harmful Effects

Local Vapor may produce irritation of eyes, nose and throat. Concentrated solutions are capable of causing skin irritation. In rare instances, dermatitis from hypersensitivity to ethyl acetate may be encountered.

Route of Entry Inhalation of vapor.

Systemic Exhibits narcotic action through central nervous system depression. Prolonged inhalation may produce acute pulmonary edema.

Special Diagnostic Test

None.

Recommended Threshold Limit

400 parts per million parts of air by volume or 1,400 milligrams per cubic meter of air.

Potential Occupational Exposures

Bristle makers
Confection makers
Denatured alcohol makers
Dope processors
Drug makers
Ethyl acetate workers
Explosive makers
Flavoring makers
Fruit essence makers
Horsehair makers
Ink makers
Lacquerers
Lacquer makers
Leather makers
Nitrocellulose makers
Organic chemical synthesizers
Perfume makers
Photographic film makers
Rayon makers
Resin makers
Smokeless powder makers
Solvent workers
Stainers
Stain makers
Varnish makers

Reference

VON OETTINGEN, W. F.: The aliphatic acids and their esters: toxicity and potential dangers; the saturated monobasic aliphatic acids and their esters. *A.M.A. Arch. Indust. Health* 21: 28, 1960.

(74) Ethyl Alcohol

ethanol, grain alcohol, ethyl hydroxide

Harmful Effects

Local Irritant to eyes and mucous membranes. Repeated contact can produce dry, scaly, and fissured dermatitis.

Route of Entry Inhalation of vapor.

Systemic When inhaled in very high concentrations, a mild degree of alcoholic intoxication may be produced.

Special Diagnostic Tests

Analysis of blood and urine for alcohol. *See* Gonzales et al., 1954, and Stewart et al., 1960.

218-695 O-66-10

Recommended Threshold Limit

1,000 parts per million parts of air by volume or 1,900 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetaldehyde makers
Acetic anhydride makers
Antifreeze makers
Beverage makers
Cleaning compound makers
Cosmetic makers
Denatured alcohol makers
Detergent makers
Disinfectant makers
Distillers
Drug makers
Dye makers
Ethyl alcohol workers
Explosive makers
Histology technicians
Ink makers
Motor fuel blenders
Organic chemical synthesizers
Rocket fuel handlers
Rocket fuel makers
Rubber makers
Shellac processors
Solvent workers
Stainers
Stain makers
Thermometer makers, vapor pressure
Varnish makers

References

GONZALES, T. A.; VANCE, M.; HELPERN, M., AND UMBERGER, C. J.: *Legal Medicine; Pathology and Toxicology.* 2nd ed. Appleton-Century-Crofts, New York, 1954. Ch. 46.

HENSON, E. V.: The toxicology of some aliphatic alcohols; part 2. *J. Occup. Med.* 2: 497, 1960.

STEWART, R. D.; TORKELSON, T. R.; HAKE, C. L., AND ERLEY, D. S.: Infrared analysis of carbon tetrachloride and ethanol in blood. *J. Lab. & Clin. Med.* 56: 148, 1960.

(75) Ethylbenzene

ethylbenzol, phenylethane

Harmful Effects

Local Exposure to liquid or vapor may produce primary irritation of skin, eyes, and mucous membranes of upper respiratory tract.

Route of Entry Inhalation of vapor. Percutaneous absorption of liquid resulting in systemic toxicity is not likely to occur through intact skin.

Systemic No systemic effects from industrial exposures have been reported. In human experimental studies, dizziness was produced with exposure to 2,000 parts per million parts of air after 6 minutes.

Special Diagnostic Test

Analysis of urine for hippuric acid. *See* Gerarde, 1960.

Recommended Threshold Limit

200 parts per million parts of air by volume or 870 milligrams per cubic meter of air.

Potential Occupational Exposures

Ethylbenzene workers
Lacquerers
Lacquer makers
Motor fuel makers
Organic chemical synthesizers
Resin makers
Solvent workers
Styrene makers

Reference

GERARDE, H. W.: *Toxicology and Biochemistry of Aromatic Hydrocarbons.* Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1960.

(76) Ethyl Bromide (Bromoethane). *See* Bromine and Compounds

(77) Ethyl Chloride

monochloroethane, hydrochloric ether, chloroethane

Harmful Effects

Local Rapid evaporation from skin may cause mild frostbite. Both liquid and gas may irritate eyes.

Route of Entry Inhalation of gas.

Systemic Ethyl chloride is a narcotic and produces headache, dizziness, incoordination, and eventual loss of consciousness. In high concentrations it is toxic to cardiac muscle and kidney.

Special Diagnostic Test

None.

Recommended Threshold Limit

1,000 parts per million parts of air by volume or 2,600 milligrams per cubic meter of air.

Potential Occupational Exposures

Anesthetists
Dentists
Drug makers
Dye makers
Ethylation workers
Ethyl cellulose makers
Ethyl chloride workers
Fat processors
Oil processors
Organic chemical synthesizers
Perfume makers
Phosphorus processors
Physicians
Refrigeration workers
Resin makers
Sulfur processors
Tetraethyl lead makers
Wax makers

Reference

VON OETTINGEN, W. F.: The halogenated aliphatic, olefinic, cyclic, aromatic, and aliphatic-aromatic hydrocarbons including the halogenated insecticides; their toxicity and potential dangers. Pub. Health Service Pub. No. 414, U.S. Government Printing Office, Washington, D.C., 1955.

(78) Ethylene Chlorohydrin

glycol chlorohydrin, 2-chloroethanol

Harmful Effects

Local High vapor concentrations are irritating to eyes, nose, and throat.
Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.
Systemic Early symptoms are dizziness, nausea, vomiting, and drowsiness. Several hours after exposure there may be severe headache, dyspnea, fatigue, cyanosis, chest pain, shock, coma, and death. There may also be pulmonary edema, and liver and kidney damage.

Special Diagnostic Test

Analysis of blood and urine for ethylene chlorohydrin. *See* Ballotta et al., 1953.

Recommended Threshold Limit

5 parts per million parts of air by volume or 16 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Cellulose acetate workers
Drug makers
Dye makers
Ethyl cellulose workers
Ethylene chlorohydrin workers
Ethylene glycol makers
Ethylene oxide makers
Indigo makers
Insecticide makers
Lacquer makers
Oil of rose makers
Organic chemical synthesizers
Potato growers
Potato sprouters
Procaine makers
Resin workers
Textile dyers
Textile printers
Varnish makers

References

BALLOTTA, F.; BERTAGNI, P., AND TROISI, F. M.: Acute poisoning caused by ingestion of ethylene chlorohydrin. *Brit. J. Indust. Med.* 10: 161, 1953.

BUSH, A. F.; ABRAMS, H. K., AND BROWN, H. V.: Fatality and illness caused by ethylene chlorhydrin in an agricultural occupation. *J. Indust. Hyg. and Toxicol.* 31: 352, 1949.

(79) Ethylenediamine

ethanediamine, 1,2-diaminoethane

Harmful Effects

Local Liquid and vapor are irritating to skin, eyes, and mucous membranes. Severe corneal injury and allergic contact dermatitis can occur. Upper respiratory tract is irritated by high concentrations.
Route of Entry Inhalation of vapor.

Systemic Headache, vertigo, nausea, and vomiting occur in chronic exposure. Several cases of allergic bronchial asthma have been reported. On the basis of animal experiments, kidney damage may be expected from severe exposures.

Special Diagnostic Test

None.

Recommended Threshold Limit

10 parts per million parts of air by volume or 30 milligrams per cubic meter of air.

Potential Occupational Exposures

Adhesive workers
Albumin processors
Antifreeze workers
Casein processors
Drug makers
Dye makers
Emulsion workers
Ethylenediamine tetraacetic acid (EDTA) makers
Ethylenediamine workers
Labelers
Oil neutralizers
Organic chemical synthesizers
Packagers
Protein processors
Rubber makers
Shellac processors
Skin dehairers
Sulfur processors
Surfactant makers
Textile lubricant workers

Reference

DERNEHL, C. U.: Clinical experiences with exposures to ethylene amines. *Indust. Med. & Surg.* 20: 541, 1951.

(80) Ethylene Dibromide

sym.-dibromoethane, EDB

Harmful Effects

Local Liquid and high vapor concentrations can irritate skin, eyes, and mucous membranes.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Headache, weakness, protracted vomiting, diarrhea, tinnitus, and heart failure. On the basis of animal experiments, irritation of lungs and liver damage may be expected.

Special Diagnostic Test

None.

Recommended Threshold Limit

25 parts per million parts of air by volume or 190 milligrams per cubic meter of air.

Potential Occupational Exposures

Antiknock compound makers
Cabbage growers
Celluloid makers
Corn growers
Drug makers
Ethylene dibromide workers
Fat processors
Fire extinguisher makers
Fruit fumigators
Fumigant workers
Gasoline blenders
Grain elevator workers
Grain fumigators
Gum processors
Lead scavenger makers
Motor fuel workers
Nematode controllers
Oil processors
Organic chemical synthesizers
Resin makers
Seed corn maggot controllers
Soil fumigators
Termite controllers
Tetraethyl lead makers
Waterproofing makers
Wax makers
Wood insect controllers
Wool reclaimers

Reference

OLMSTEAD, E. V.: Pathological changes in ethylene dibromide poisoning. *A.M.A. Arch. Indust. Health* 21: 525, 1960.

(81) Ethylene Dichloride

1,2-dichloroethane, sym.-dichloroethane

Harmful Effects

Local Liquid and vapor are irritating to eyes. Irritation by vapor of upper respiratory tract may produce sneezing. Repeated contact with liquid can produce a dry, scaly, fissured dermatitis. Allergic contact dermatitis is rare.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Vapor acts as narcotic in high concentrations and inhalation may produce headache, dizziness, loss of appetite, nausea, vomiting, epigastric pain, visual disturbances, loss of consciousness, and death. Vapor may irritate respiratory tract with production of cough. Liver damage has been suggested by some cases with enlargement of liver and low blood-sugar levels, but ethylene dichloride does not characteristically affect liver. Corneal opacities, as a systemic effect, have been observed only in dogs.

Special Diagnostic Test

None.

Recommended Threshold Limit

50 parts per million parts of air by volume or 200 milligrams per cubic meter of air.

Potential Occupational Exposures

Alkaloid processors
Bakelite processors
Camphor workers
Cellulose ester workers
Cleaning compound makers
Dry cleaners
Dyers
Ethylene dichloride workers
Exterminators
Fat processors
Flotation workers
Fumigant workers
Gum processors
Insecticide makers
Lacquerers
Lacquer makers
Lacquer remover workers
Lead scavenger makers
Metal degreasers
Oil processors
Ore upgraders
Paint removers
Paint remover workers
Paraffin workers
Plasticizing bath operators
Resin makers
Rubber makers
Soap makers
Solvent workers
Stain removers
Succinic acid makers
Tetraethyl lead makers
Textile cleaners
Tobacco denicotinizers
Trichloroethylene makers
Varnish makers
Varnish remover workers
Vinyl chloride makers
Wax makers
Wire insulators
Wool cleaners

Reference

IRISH, D. D.: Common chlorinated aliphatic hydrocarbon solvents. *Arch. Environ. Health* 4: 320, 1962.

(82) Ethylene Glycol

1,2-ethanediol, glycol alcohol, glycol

Harmful Effects

Local Liquid may irritate conjunctiva. Skin effects have not been reported.

Route of Entry Inhalation of vapor.

Systemic Ethylene glycol is a central nervous system depressant producing symptoms similar to ethyl alcohol intoxication. Cases of poisoning have generally followed ingestion of the compound. Inhalation of vapor is uncommon since liquid has high boiling point; however, episodes of unconsciousness, nystagmus, and lymphocytosis have been reported to follow inhalation. Death usually is result of cardiac or renal failure. *See* Cellosolve.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Antifreeze makers
Brake fluid makers
Cellophane makers
Cosmetic makers
Drug makers
Dye makers
Electrolytic condenser makers
Ethylene glycol workers
Explosive makers
Fire extinguisher makers
Garage workers
Glue makers
Glyoxal makers
Ink makers
Lacquerers
Lacquer makers
Leather dyers
Metal cleaners
Metal polishers
Painters
Paint makers
Resin makers
Textile makers
Tobacco workers
Wax makers
Wood stainers
Wood stain makers

References

MORINI, I.: Several cases of poisoning with commercial ethylene glycol. *Minerva med.* 1: 72, 1954. (*Indust. Hyg. Digest* Abst. No. 210, February 1956)

NADEAU, G.; COTE, R., AND DELANEY, F. J.: Two cases of ethylene glycol poisoning. *Canad. Med. Asso. J.* 70: 69, 1954.

TROISI, F. M.: Chronic intoxication by ethylene glycol vapour. *Brit. J. Indust. Med.* 7: 65, 1950.

(83) Ethylene Glycol Dinitrate

nitroglycol, glycol dinitrate, ethylene dinitrate

Ethylene glycol dinitrate, itself an explosive, is often used to lower the freezing point of dynamite.

Harmful Effects

Local None known.

Routes of Entry Inhalation of vapor or dust; percutaneous absorption or ingestion of liquid.

Systemic EGD is a potent vasodilator and owes much of its toxicity to this property. Acute effects include headache, nausea, vomiting, hypotension, and tachycardia. EGD is a methemoglobin former but methemoglobinemia does not seem to be an important aspect of poisoning. Hypotension is frequently seen in workers exposed daily to EGD. Anginoid pain and cases of sudden death, particularly on hot, humid days, have been reported after removal from daily exposure to EGD following apparent habituation to its vasodilating effects.

Special Diagnostic Tests

None.

Threshold Limit Value

(Ethylene glycol dinitrate with nitroglycerine) 0.2 part per million parts of air by volume or 1.2 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Dynamite makers
Ethylene glycol dinitrate workers
Miners
Powder monkeys

References

BARSOTTI, M.: Attacks of stenocardia in workers engaged in the production of dynamites with nitroglycol. *Med. Lavoro* 45: 544, 1954.

FRIMMER, M.; GROSS, E.; KIESE, M., AND RESAG, K.: Absorption of ethylene glycol dinitrate through the lung. *Arch. Toxikol.* 18: 200, 1960.

GROSS, E.; BOCK, M., AND HELLRUNG, F.: The toxicology of nitroglycol in comparison with that of nitroglycerin. *Arch. exper. Path. Pharmakol.* 200: 271, 1942.

GROSS, E.; KIESE, M., AND RESAG, K.: Skin absorption of ethylene glycol dinitrate. *Arch. Toxikol.* 18: 194, 1960.

SYMANSKI, H.: Severe injury to health from occupational exposure to nitroglycol. *Arch. Hyg. Bakteriol.* 136: 139, 1952.

VON OETTINGEN, W. F.: The effects of aliphatic nitrous and nitric acid esters on the physiological functions with special reference to their chemical constitution. Nat. Inst. Health Bull. No. 186. U.S. Government Printing Office, Washington, D.C., 1946.

(84) Ethylene Oxide

1,2-epoxyethane, oxirane, dimethylene oxide

Harmful Effects

Local Ethylene oxide liquid and gas are irritating to eyes and wet skin, but anhydrous liquid ethylene oxide does not cause primary injury to dry skin. Aqueous solutions near the 50 percent concentration are vesicants. Allergic eczematous dermatitis has also been reported. Ethylene oxide is absorbed by leather and rubber, and may produce belated irritation.

Route of Entry Inhalation of gas.

Systemic Gas is a pulmonary irritant and in high concentrations will produce pulmonary edema, with cough, dyspnea, and respiratory distress. Systemic effects of headache, nausea, vomiting, and narcosis have been noted. Toxic effects may be due to glycols which are formed when ethylene oxide combines with water in the body.

Special Diagnostic Test

None.

Recommended Threshold Limit

50 parts per million parts of air by volume or 90 milligrams per cubic meter of air.

Potential Occupational Exposures

Acrylonitrile makers
Butyl cellosolve makers
Detergent makers
Disinfectant makers
Ethanolamine makers
Ethylene glycol makers
Ethylene oxide workers
Exterminators
Farm product fumigators
Foodstuff fumigators
Fumigant makers
Fungicide workers
Gasoline sweeteners
Grain elevator workers
Organic chemical synthesizers
Polyglycol makers
Polyoxirane makers
Rocket fuel handlers
Rocket fuel makers
Surfactant makers
Textile fumigators
Textile lubricant makers
Tobacco fumigators

References

JACOBSON, K. H.; HACKLEY, E. B., AND FEINSILVER, L.: The toxicity of inhaled ethylene oxide and propylene oxide vapors. *A.M.A. Arch. Indust. Health* 13: 237, 1956.

JACOBSON, K. H.: Industrial hygiene aspects of liquid propellants. *Transactions, 22nd annual meeting, American Conference of Governmental Industrial Hygienists, 1960.* P. 30. Sec.-Treas., 1014 Broadway, Cincinnati 2, Ohio.

ROYCE, A. AND MOORE, W. K. S.: Occupational dermatitis caused by ethylene oxide. *Brit. J. Indust. Med.* 12: 169, 1955.

SEXTON, R. J. AND HENSON, E. V.: Dermatological injuries by ethylene oxide. *J. Indust. Hyg. & Toxicol.* 31: 297, 1949.

(85) Ethyl Ether

ethoxyethane, ether, diethyl ether, sulfuric ether, anesthetic ether, ethyl oxide, diethyl oxide

Harmful Effects

Local Contact with liquid may produce a dry, scaly, fissured dermatitis.

Route of Entry Inhalation of vapor.

Systemic In acute exposure, there is a period of excitation followed by central nervous system depression or anesthesia. Pulmonary edema in rare instances may follow acute exposure.

Special Diagnostic Test

Analysis of blood for ether. *See* Von Oettingen, 1958.

Recommended Threshold Limit

400 parts per million parts of air by volume or 1,200 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetic acid makers
Alcohol denaturers
Alkaloid processors
Anesthetic makers
Collodion makers
Drug makers
Dry cleaners
Ethyl ether workers
Explosive makers
Fat processors
Fumigant makers
Fumigators
Gasoline engine primers
Gum processors
Medical technicians
Motor fuel makers
Nurses
Oil processors
Organic chemical synthesizers
Perfume makers
Physicians
Plastic makers
Rayon makers
Refrigerant makers
Refrigeration workers
Wax makers

References

HAMILTON, A. AND MINOT, G. R.: Ether poisoning in the manufacture of smokeless powder. *J. Indust. Hyg.* 2: 41, 1920.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*, 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(86) Ethyl Mercaptan. *See* Mercaptans

(87) Ethyl Silicate

tetraethyl orthosilicate, tetraethoxy silane

Harmful Effects

Local Vapor is irritating to eyes and nose.

Route of Entry Inhalation of vapor.

System Damage to lungs, liver, and kidney has been observed in experimental animals, but no cases have been reported from industrial exposure.

Special Diagnostic Test

None.

Recommended Threshold Limit

100 parts per million parts of air by volume or 850 milligrams per cubic meter of air.

Potential Occupational Exposures

Acidproof cement makers
Adhesive makers
Brick preserver makers
Building coaters
Casting coaters
Cement preserver makers
Ethyl silicate workers
Heat resistant paint makers
Lacquer makers
Plaster preserver makers

Protective coating makers
Refractory brick makers
Silicate paint makers
Stone preserver makers
Weatherproof cement makers

Reference

POZZANI, U. C. AND CARPENTER, C. P.: Response of rodents to repeated inhalation of vapors of tetraethyl orthosilicate. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 465, 1951.

(88) Fluorine and Compounds

Harmful Effects

Local Fluorine gas, anhydrous hydrofluoric acid, and aqueous hydrofluoric acid are intense primary irritants of skin, eyes, and mucous membranes. Burns may be chemical or thermal. Chemical burns cause deep tissue destruction and may not become symptomatic until several hours after contact.

Route of Entry Inhalation of gas, mist, dust, or fume.

Systemic Fluorine and hydrogen fluoride are pulmonary irritants and produce pulmonary edema. Inhalation of fluoride dust or fume may produce respiratory tract irritation manifested by chills, fever, dyspnea, and cough. Chronic toxicity from inhalation of fluoride as manifested by increased osseous radiopacity is seldom encountered.

Special Diagnostic Tests

Determination of fluorides in blood and urine; roentgenogram of spine. *See* Von Oettingen, 1958; Talvitie and Brewer, 1960, and Dubois et al., 1962.

Recommended Threshold Limit

Fluorine, 0.1 part per million parts of air by volume or 0.2 milligram per cubic meter of air.

Hydrogen fluoride, 3 parts per million parts of air by volume or 2 milligrams per cubic meter of air.

Fluoride, 2.5 milligrams per cubic meter of air.

Potential Occupational Exposures

Fluorine

Antimony fluoride makers
Cobalt fluoride makers
Fluoride makers
Fluorine workers
Metallic fluoride makers
Rocket fuel handlers
Rocket fuel makers
Sulfur hexafluoride makers
Uranium hexafluoride makers

Hydrogen fluoride

Aircraft workers
Alloy steel cleaners
Aluminum fluoride makers
Aluminum makers
Ammonium fluoride makers
Bleachers
Brass cleaners
Brick cleaners
Casting cleaners
Ceramic workers
Copper cleaners
Cryolite makers
Crystal glass polishers
Dye makers
Enamel etchers
Fermentation workers
Fertilizer makers
Filter paper makers
Fluoborate makers
Fluoride makers
Fluorine makers
Fluorocarbon makers
Fluorochemical makers
Fluosilicate makers
Freon makers
Genetron makers
Glass etchers
Graphite purifiers
Hydrogen fluoride workers
Incandescent lamp frosters
Isotron makers
Laundry workers
Metal cleaners
Metal polishers
Oil well acidizers
Ore dissolvers
Petroleum refinery workers
Plastic makers
Polish workers
Rocket fuel handlers
Rocket fuel makers
Stainless steel cleaners
Stone cleaners
Uranium refiners
Yeast makers

Fluoride

Adhesive makers
Aluminum anodizers
Aluminum makers
Aluminum refiners
Aluminum solderers
Aluminum welders
Apatite workers
Bactericide workers
Beryllium refiners
Building workers
Carbon electrode workers
Cement workers
Ceramic workers
Chemical polisher workers
Chlorofluorocarbon makers
Concreters
Construction workers
Copper refiners
Cryolite makers
Dentifrice makers
Diazosalt makers
Disinfectors
Electric arc welders
Electric arc workers
Electroplaters
Electropolishers
Embalmers
Embalming fluid workers
Fluorapatite workers
Fluoride workers
Fluorocarbon makers
Fluorspar miners
Foundry workers
Frit workers
Fungicide workers
Glass etchers

Gold refiners
Grinding wheel makers
Hydrofluoric acid makers
Insect exterminators
Insecticide makers
Latex foam rubber workers
Laundry workers
Lead electroplaters
Lead smelters
Light metal casters
Magnesium foundry workers
Marble hardeners
Masonry preservers
Metal cleaners
Metal coating workers
Metal refiners
Mica makers
Mothproofing workers
Opal glass makers
Open hearth steel workers
Optical equipment makers
Paint workers
Petroleum refinery workers
Phosphorescent tube makers
Phosphoric acid makers
Phosphorus makers
Porcelain enamel workers
Rock phosphate acidulators
Rodent exterminators
Sandy soil treaters
Silver refiners
Silver solder flux workers
Soil improvers
Stainless steel welding rod users
Super phosphate makers
Textile mordanters
Vegetable growers
Vitreous enamel workers
Water treaters
Wood preservative workers
Wood preservers
Zinc miners

References

DERRYBERRY, O. M.; BARTHOLOMEW, M. D., AND FLEMING, R. B. L.; Fluoride exposure and worker health; the health status of workers in a fertilizer manufacturing plant in relation to fluoride exposure. *Arch. Environ. Health* 6: 503, 1963.

DIEFFENBACHER, P. F. AND THOMPSON, J. H.: Burns from exposure to anhydrous hydrofluoric acid. *J. Occup. Med.* 4: 325, 1962.

DUBOIS, L.; MONKMAN, J. L., AND TEICHMAN, T.: The determination of urinary fluorides. *Am. Indust. Hyg. Assoc. J.* 23: 157, 1962.

PATTISON, F. L. M.: *Toxic Aliphatic Fluorine Compounds.* Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1959.

PRINCI, F.: Fluorides; a critical review. 3, The effects on man of the absorption of fluoride. *J. Occup. Med.* 2: 92, 1960.

TALVITIE, N. A. AND BREWER, L. W.: Separation of fluoride by ion exchange; application to urine analysis. *Am. Indust. Hyg. Assoc. J.* 21: 287, 1960.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* W. B. Saunders Co., Philadelphia, 1958.

(89) Formaldehyde

methanal, oxomethane, oxymethylene, methylene oxide, formic aldehyde, methyl aldehyde

Formalin is a 40 percent aqueous solution of formaldehyde.

Harmful Effects

Local Formaldehyde gas is highly irritating to conjunctiva and mucous membranes of upper respiratory tract. Aqueous solutions may cause

contact dermatitis from primary irritation or allegic hypersensitivity. Urticaria has been reported following inhalation of gas.

Route of Entry Inhalation of gas.

Systemic Systemic intoxication appears to be of little importance in industry since intense irritation of upper respiratory tract compels worker to leave the area. Ingestion may result in gastrointestinal irritation, respiratory depression, and death.

Special Diagnostic Test

None.

Recommended Threshold Limit

5 parts per million parts of air by volume or 6 milligrams per cubic meter of air.

Potential Occupational Exposures

Anatomists
Bakers
Biologists
Bookbinders
Botanists
Crease-resistant textile finishers
Deodorant makers
Disinfectant makers
Disinfectors
Dress goods store personnel
Dress makers
Drug makers
Dye makers
Embalmers
Embalming fluid makers
Ethylene glycol makers
Formaldehyde resin makers
Formaldehyde workers
Fungicide workers
Furniture dippers
Furniture sprayers
Fur processors
Glass etchers
Hexamethylenetetramine makers
Hide preservers
Histology technicians
Ink makers
Lacquerers
Lacquer makers
Oil well workers
Paper makers
Pentaerythritol makers
Photographic film makers
Resin makers
Rubber makers
Tannery workers
Textile mordanters
Textile printers
Textile waterproofers
Wood preservers

References

GLASS, W. I.: An outbreak of formaldehyde dermatitis. *New Zealand J. Med.* 60: 423, 1961.

HENSON, E. V.: The toxicology of some aliphatic aldehydes. *J. Occup. Med.* 1: 457, 1959.

INDUSTRIAL HYGIENE RESEARCH LABORATORY, NATIONAL INSTITUTE OF HEALTH, U.S. PUBLIC HEALTH SERVICE: Formaldehyde; its toxicity and potential dangers. *Pub. Health Rep. Supp.* No. 181, 1945.

ROY, M., JR.: Corrosive gastritis after formaldehyde ingestion; report of a case. *New Eng. J. Med.* 266: 1248, 1962.

(90) Formic Acid

methanoic acid, formylic acid, hydrogen carboxylic acid

Harmful Effects

Local Formic acid vapor is irritating to mucous membranes of upper respiratory tract. Liquid in concentrated solution is primary skin irritant.

Routes of Entry Percutaneous absorption of liquid; inhalation of vapor.

Systemic Systemic effects have not been recognized in industry. Fatal poisoning from accidental or suicidal ingestion of formic acid has been frequently encountered. The clinical picture is characterized by salivation, burning sensation in mouth and pharynx, vomiting, hematemesis, diarrhea, and severe abdominal pain. Shock may result with subsequent acute renal failure, or respiratory failure and death.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Acetic acid makers
Airplane dope makers
Allyl alcohol makers
Cellulose formate makers
Dyers
Electroplaters
Food preservers
Formate makers
Formic acid workers
Fumigant makers
Glass silverers
Insecticide makers
Lacquer makers
Laundry workers
Leather makers
Nickel platers
Ore refiners
Organic ester makers
Oxalic acid makers
Paper makers
Perfume makers
Phenolic resin makers
Refrigerant makers
Rubber workers
Tannery workers
Textile makers
Wine makers

Reference

HENSON, E. V.: Toxicology of the fatty acids. *J. Occup. Med.* 1: 339, 1959.

(91) Freon [R]

Freon-11, fluorotrichloromethane
Freon-12, dichlorodifluoromethane
Freon-12B2, difluorodibromomethane
Freon-13, monochlorotrifluoromethane

Freon–13B1, trifluoromonobromomethane
Freon–14, tetrafluoromethane
Freon–21, dichloromonofluoromethane
Freon–22, monochlorodifluoromethane
Freon–23, trifluoromethane
Freon–112, tetrachlorodifluoroethane
Freon–113, trichlorotrifluoroethane
Freon–113B2, dibromomonochlorotrifluoroethane
Freon–114, dichlorotetrafluoroethane
Freon–114B2, dibromotetrafluoroethane
Freon–115, monochloropentafluoroethane
Freon–C318, octafluorocyclobutane

Harmful Effects

Local These fluorinated hydrocarbons may produce very mild irritation of the upper respiratory tract. If chlorine-containing fluoromethanes come into contact with an open flame or hot metal, the decomposition products of hydrogen chloride, hydrogen fluoride, phosgene, sulfur dioxide and chlorine may cause severe irritative effects.

Route of Entry Inhalation of vapor or gas.

Systemic Certain of these Freons may produce mild central nervous system depression. Systemic effect may be due in part to displacement of air, with resultant hypoxia.

Special Diagnostic Test

None.

Recommended Threshold Limit

Freon-11, 1,000 parts per million parts of air by volume or 5,600 milligrams per cubic meter of air.

Freon-12, 1,000 parts per million parts of air by volume or 4,950 milligrams per cubic meter of air.

Freon-12B2, 100 parts per million parts of air by volume or 860 milligrams per cubic meter of air.

Freon-13B1, 1,000 parts per million parts of air by volume or 6,100 milligrams per cubic meter of air.

Freon-21, 1,000 parts per million parts of air by volume or 4,200 milligrams per cubic meter of air.

Freon-112 (tentative), 500 parts per million parts of air by volume or 4,170 milligrams per cubic meter of air.

Freon-113, 1,000 parts per million parts of air by volume or 7,600 milligrams per cubic meter of air.

Freon-114, 1,000 parts per million parts of air by volume or 7,000 milligrams per cubic meter of air.

218-695 O-66-11

Potential Occupational Exposures

Aerosol bomb workers
Ceramic mold makers
Drug makers
Fire extinguisher workers
Freon workers
Heat transfer workers
Metal conditioners
Plastic makers
Pressurized food makers
Refrigerant workers
Rocket fuel makers
Solvent workers
Sponge rubber makers

References

DALHAMN, T.: Freon as a cause of poisoning. *Nordisk Hyg. Tidskr.* 39: 165, 1958. (Abst., *Bull. Hyg.* 34: 912, 1959.)

PATTISON, F. L. M.: *Toxic Aliphatic Fluorine Compounds.* Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1959.

(92) Furfural

furfurol, furfuraldehyde, artificial ant oil, pyromucic aldehyde, furol

Harmful Effects

Local Liquid and high vapor concentrations are irritating to skin, eyes, and mucous membranes; can produce corneal anesthesia. Allergic contact dermatitis and photosensitization may occur.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Acute effects from inhalation of vapor have been limited to headaches and breathing difficulties.

Special Diagnostic Test

None.

Recommended Threshold Limit

5 parts per million parts of air by volume or 20 milligrams per cubic meter of air.

Potential Occupational Exposures

Adipic acid makers
Adiponitrile makers
Butadiene refiners
Cellulose acetate makers
Disinfectant workers
Disinfectors
Fungicide workers
Furfural workers
Grinding wheel makers
Herbicide makers
Highway maintenance workers
Lubricating oil refiners
Lysine makers
Metal refiners
Nitrocellulose makers
Nylon makers
Paint remover makers
Petroleum refinery workers
Phenol furfural makers
Rare earth refiners
Resin makers
Road builders

Rosin refiners
Rubber makers
Shoe dye makers
Varnish makers
Weed sprayers
Wetting agent workers
Wood rosin declorizers

Reference

DUNLOP, A. P. AND PETERS, F. N.: *The Furanes.* American Chemical Society Monograph Series No. 119. Reinhold Publishing Corp., New York, 1953.

(93) Gasoline

petrol, motor spirits

Harmful Effects

Local Gasoline is irritating to skin, conjunctiva, and mucous membranes of upper respiratory tract.

Route of Entry Inhalation of vapor. Ill effects from percutaneous absorption of liquid are questionable.

Systemic Exposure to low concentrations of vapor may produce symptoms similar to ethyl alcohol intoxication, including flushing of face, staggering gait, slurred speech, and mental confusion. Higher concentrations may result in unconsciousness, coma, and death. Ingestion of liquid often results in aspiration with a pneumonitis similar to that seen in kerosine intoxication. Symptoms of gastrointestinal irritation may also occur.

The existence of chronic poisoning has been questioned. The possibility of blood alterations developing from absorption of aromatic hydrocarbons in gasoline should be considered.

Special Diagnostic Test

None.

Recommended Threshold Limit

500 parts per million parts of air by volume or 2,000 milligrams per cubic meter of air.

Potential Occupational Exposures

Gasoline is used as a fuel, diluent, and solvent in numerous occupations throughout various industries.

References

DAVIS, A.; SCHAFER, L. J., AND BELL, L. G.: The effects on human volunteers of exposure to air containing gasoline vapor. *Arch. Environ. Health* 1: 548, 1960.

MACHLE, W.: Gasoline intoxication. *J. Am. Med. Assoc.* 117: 1065, 1941.

TUCKER, R.; KILBOURNE, E. D., AND EVANS, J. B.: Pulmonary manifestations of gasoline intoxication. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 2: 17, 1950.

WANG, C. C. AND IRONS, G. V.: Acute gasoline intoxication. *Arch. Environ. Health* 2: 714, 1961.

(94) Germanium Compounds

Harmful Effects

Local Germanium tetrachloride and tetrafluoride are mucous membrane irritants. No local effects of other germanium compounds have been reported.

Route of Entry Inhalation of gas or vapor.

Systemic No cases of industrial poisoning have been reported.

Special Diagnostic Tests

Analysis of urine and feces for germanium. *See* Fairhall, 1957.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Argyrodite workers
Dental alloy makers
Feldistor makers
Germanite workers
Germanium workers
Glass makers
Phosphor makers
Photodiode makers
Rectifier makers
Semiconductor makers
Transistor makers
Vacuum tube makers
Zinc residue workers

References

DUDLEY, H. C. AND WALLACE, E. J.: Pharmacological studies of radio-germanium (Ge–71). *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 263, 1952.

FAIRHALL, L. T.: *Industrial Toxicology.* 2nd ed. Williams & Wilkins Co., Baltimore, 1957.

HARROLD, G. C. AND MEEK, S. F.: The physiologic properties of germanium. *Indust. Med.* 13: 236, 1944.

HUEPER, W. C.: Germanium. *Occup. Med.* 4: 208, 1947.

(95) Graphite

plumbago, black lead, mineral carbon

Harmful Effects

Local None.

Route of Entry Inhalation of dust.

Systemic Natural graphite contains free crystalline silica and exposures of several years to this silica-graphite combination may produce a disabling pneumoconiosis that is similar to other modified silica pulmonary reactions, for example, anthracosilicosis. Pure graphite has not been shown to produce this picture of severe pulmonary change.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Battery makers, dry
Brake lining makers
Cathode ray tube makers
Commutator brush makers
Crucible makers
Electric appliance makers
Electrode makers
Electroplaters
Electrotypers
Explosive makers
Foundry workers
Gasket makers
Graphite cement makers
Graphite miners
Graphite workers
Lubricant makers
Match makers
Nuclear reactor workers
Paint makers
Pencil lead makers
Pigment makers
Pipe joint compound makers
Polish makers
Radio resistor makers
Refractory material makers
Retort makers
Roofing makers
Steel makers
Stove polish makers
Thermocouple (with tungsten) makers

References

HARDING, H. E. AND OLIVER, G. B.: Changes in the lungs produced by natural graphite. *Brit. J. Indust. Med.* 6: 91, 1949.

HIRSCH, M. J.; KASS, I.; SCHAEFER, W. B., AND DENST, J.: Infection with atypical tubercle bacilli in graphite pneumoconiosis. *A.M.A. Arch. Int. Med.* 103: 814, 1959.

(96) Hexamethylenetetramine

methenamine, hexamine, formamine, ammonioformaldehyde

Harmful Effects

Local Contact with solid, or its fumes when heated, can irritate skin or produce an allergic eczematous contact dermatitis.

Route of Entry Ingestion.

Systemic Gastrointestinal irritation and kidney damage have occurred following large oral doses.

Special Diagnostic Tests

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Anticorrosion additive workers
Drug makers
Explosive makers
Foundry workers
Fuel tablet makers
Fungicide makers
Gas mask makers
Hexamethylenetetramine workers
Phenol-formaldehyde resin workers
Phosgene absorption cannister makers
Resin makers
Rubber makers
Textile makers
Urea-formaldehyde resin workers
Veterinarians

Reference

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(97) Hydrazine

hydrazine base, diamine

Harmful Effects

Local Contact of this hygroscopic liquid with skin and eyes produces penetrating burns. Contact with vapor results in eczematous dermatitis from either primary irritation or allergic hypersensitivity. Irritation of eyes and nose by high concentrations is so intense as to compel workers to leave the area usually before lower respiratory tract suffers damage.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Low grade exposure produces headache, nausea, and dizziness. Bronchitis and pneumonitis may result if early irritative warnings are not heeded. On basis of animal experiments, hydrazine may also produce central nervous system symptoms of excitement and convulsions, fatty necrosis of liver, nephritis, hemolytic anemia, hypoglycemia, and hypotension.

Special Diagnostic Test

Pyridyl test for hydrazine assay in blood plasma. *See* Prescott et al., 1955.

Recommended Threshold Limit

1 part per million parts of air or 1.3 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Agricultural chemical makers
Anticorrosion additive workers
Antioxidant workers
Boiler operators
Chlorine scavenger makers
Drug makers
Explosive makers
Hydraulic fluid workers
Hydrazine workers
Insecticide makers

Jet fuel handlers
Jet fuel makers
Oxygen scavenger makers
Photographic developer makers
Rocket fuel handlers
Rocket fuel makers
Solder flux makers
Sponge rubber makers
Textile dyers, acrylic and vinyl
Vat dye makers
Water treaters

References

EVANS, D. M.: Two cases of hydrazine hydrate dermatitis without systemic intoxication. *Brit. J. Indust. Med.* 16: 126, 1959.

JACOBSON, K. H.: Industrial hygiene aspects of liquid propellants. In *Transactions, 22nd annual meeting, American Conference of Governmental Industrial Hygienists, 1960.* Sec.-Treas., 1014 Broadway, Cincinnati 2, Ohio.

KROP, S.: Toxicity of hydrazine. A review. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 9: 199, 1954.

OFFICE OF DIRECTOR, DEFENSE RESEARCH AND ENGINEERING, DEPARTMENT OF DEFENSE: *The Handling and Storage of Liquid Propellants.* U.S. Government Printing Office, Washington, D.C., 1961.

PRESCOTT, B.; KAUFFMANN, G., AND JAMES, W. D.: The "pyridyl" test for hydrazine assay in blood plasma. *A.M.A. Arch. Indust. Health* 12: 393, 1955.

SCHULTHEISS, E.: Hypersensitiveness to hydrazine. *Berufsdermatosen* 7: 131, 1959.

(98) Hydrogen Bromide. *See* Bromine and Compounds

(99) Hydrogen Chloride

anhydrous hydrochloric acid, chlorohydric acid

An aqueous solution of hydrogen chloride gas is hydrochloric acid or muriatic acid.

Harmful Effects

Local Hydrochloric acid and high concentrations of hydrogen chloride gas are highly irritating to eyes, skin, and mucous membranes. Discoloration of teeth and tooth decay have been noted from exposure to low concentrations of gas.

Route of Entry Inhalation of gas or mist.

Systemic Pulmonary edema is possible, but usually the cough and choking sensation from intense irritation of upper respiratory tract compel worker to leave the area.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Hydrogen chloride) 5 parts per million parts of air by volume or 7 milligrams per cubic meter of air.

Potential Occupational Exposures

Alkyl chloride makers
Battery makers
Bleachers
Boiler scale removers
Bronzers
Chloride makers
Chloroprene makers
Corn syrup makers
Drug makers
Dye makers
Electroplaters
Enamelers
Fertilizer makers
Food processors
Galvanizers
Gas well treaters
Glass finishers
Glass mixers
Glue makers
Hydrogen chloride workers
Jewelers
Lithographers
Metal cleaners
Oil well treaters
Ore reduction workers
Organic chemical synthesizers
Photoengravers
Pigment workers
Plastic workers
Pottery workers
Rubber makers
Silica gel makers
Soap makers
Sugar cane refiners
Tannery workers
Tantalum ore refiners
Tetraethyl lead makers
Textile workers
Tin ore refiners
Veterinarians
Vinyl chloride makers
Wire annealers

References

QUERIES AND MINOR NOTES: Effects of hydrochloric acid fumes. *J. Am. Med. Assoc.* 131: 1182, 1946.

THIELE, E.: Fatal poisoning from use of hydrochloric acid in a confined space. *Zentralbl. Arbeitsmed. u. Arbeitsschutz* 3: 146, 1953. (*Indust. Hyg. Digest*, Abst. No. 387, April 1954)

(100) Hydrogen Cyanide

prussic acid, hydrocyanic acid

Harmful Effects

Local None.

Route of Entry Inhalation of gas and percutaneous absorption of gas or liquid.

Systemic Symptoms are caused by chemical asphyxia, that is, inhibition of cellular oxidative processes. Acute and subacute symptoms include headache, lassitude, nausea, vomiting, shortness of breath, irritation of throat, convulsions, respiratory paralysis, coma, and death. Chronic toxicity is debatable. In general, systemic toxicity is similar for other cyanides and cyanogen compounds.

Special Diagnostic Tests

Cyanide determination in blood and tissues; thiocyanate determination in serum and urine. *See* Amdur, 1959, and Elkins, 1959.

Recommended Threshold Limit

10 parts per million parts of air by volume or 11 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Acid dippers
Acrylate makers
Acrylonitrile makers
Adipic acid makers
Adiponitrile makers
Aircraft workers
Ammonium salt makers
Art printing workers
Blacksmiths
Blast furnace workers
Bone distillers
Bronzers
Browners, gun barrel
Cadmium platers
Case hardeners
Cellulose product treaters
Coal tar distillery workers
Coke oven operators
Cyanide workers
Cyanogen makers
Disinfectant makers
Dye makers
Electroplaters
Exterminators
Fertilizer makers
Fulminate mixers
Fumigant makers
Fumigators
Gas purifiers
Gas workers, illuminating
Gilders
Gold extractors
Gold refiners
Heat treaters
Hexamethylenediamine makers
Hydrocyanic acid makers
Hydrogen cyanide workers
Insecticide makers
Jewelers
Metal cleaners
Metal polishers
Methacrylate makers
Mirror silverers
Mordanters
Nylon makers
Organic chemical synthesizers
Oxalic acid makers
Phosphoric acid makers
Photoengravers
Pigment makers
Plastic workers
Polish makers
Rayon makers
Rubber makers
Silver extractors
Silver refiners
Solderers
Steel carburizers
Tannery workers
Temperers
Textile printers
Tree sprayers
White cyanide makers
Zinc platers
Zinkers

References

AMDUR, M. L.: Accidental exposure to acetonitrile; a clinical study. *J. Occup. Med.* 1: 627, 1959.

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

WOLFSIE, J. H. AND SHAFFER, C. B.: Hydrogen cyanide; hazards, toxicology, prevention and management of poisoning. *J. Occup. Med.* 1: 281, 1959.

(101) Hydrogen Fluoride. *See* Fluorine and Compounds

(102) Hydrogen Peroxide

peroxide, hydrogen dioxide

Harmful Effects

Local Concentrated liquid and mist are extremely caustic to skin and eyes. Damage to eyes may be delayed in appearance.

Route of Entry Inhalation of vapor or mist.

Systemic Inhalation of vapor or mist produces effects ranging from mild bronchitis to pulmonary edema. No chronic systemic effects have been observed.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Hydrogen peroxide, 90 percent) 1 part per million parts of air by volume or 1.4 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetone makers
Alcoholic liquor agers
Antichlor makers
Antiseptic makers
Benzol peroxide makers
Bleachers
Bone bleachers
Button makers
Disinfectant makers
Drug makers
Dyers
Electroplaters
Fat refiners
Feather bleachers
Felt hat makers
Flour bleachers
Fruit bleachers
Fruit preservers
Fur bleachers
Fur dyers
Gelatin bleachers
Glue bleachers
Hair bleachers
Hide disinfectors
Hydrogen peroxide workers
Ivory bleachers
Metal cleaners
Oil painting renovators
Oil refiners
Photographic film developers
Plastic foam makers
Rocket fuel handlers
Rocket fuel makers
Silk bleachers
Soap bleachers
Sponge rubber makers
Straw bleachers
Textile bleachers
Torpedo propellant workers
Veterinarians
Water treaters
Wax bleachers
Wine agers
Wood pulp bleachers
Wool printers

(103) Hydrogen Sulfide

sulfuretted hydrogen, stink damp

Hydrogen sulfide is usually encountered as an industrial byproduct, but also occurs in mines, natural gas, and crude oil, and is formed from decomposing sewage and other organic matter. Certain chemicals such as thioglycolic acid and lithopone liberate hydrogen sulfide on decomposition.

Harmful Effects

Local Irritating to eyes and to mucous membranes of nose and throat.
Route of Entry Inhalation of gas.
Systemic Hydrogen sulfide is an asphyxiant because of its ability to paralyze the respiratory centers of brain with resultant cessation of respiration. Unless death occurs during period of respiratory paralysis, recovery is usually complete. An exception to this tendency toward complete recovery is occasionally seen when period of hypoxia produces permanent brain injury.

Prolonged exposure to moderately high concentrations of hydrogen sulfide may irritate tissues of respiratory tract sufficiently to produce pneumonitis or pulmonary edema. Excessive exposure to concentrations of this order of magnitude may also be attended by such symptoms as headache, gastrointestinal disturbances, dizziness, chest pain, and cough.

Although the physiologic response to different concentrations of hydrogen sulfide is subject to considerable individual variation, the accompanying table indicates the general response that might be expected to occur at a given concentration.

Parts per million	Percent	Response
0.20	0.00002	Detectable odor
20	0.002	Maximum allowable concentration for daily 8-hour exposure
150	0.015	Olfactory nerve paralysis
250	0.025	Prolonged exposure may cause pulmonary edema
500	0.05	Systemic symptoms may occur in ½ to 1 hour
1,000	0.10	Rapid collapse; respiratory paralysis imminent
5,000	0.5	Immediate death

Special Diagnostic Test

None.

Recommended Threshold Limit

20 parts per million parts of air by volume or 30 milligrams per cubic meter of air.

Potential Occupational Exposures

Barium carbonate makers
Barium salt makers
Blast furnace workers
Brewery workers
Cable splicers
Caisson workers
Carbon disulfide makers
Cellophane makers
Cistern cleaners
Coke oven workers
Copper ore sulfidizers
Depilatory makers
Dye makers
Fat renderers
Felt makers
Fertilizer makers
Fur dressers
Glue makers
Gold ore workers
Heavy metal precipitators
Hydrochloric acid purifiers
Hydrogen sulfide workers
Laboratory workers, chemical
Lead ore sulfidizers
Lead removers
Lithographers
Lithopone makers
Manholes, workers in
Miners
Natural gas makers
Paper pulp makers
Petroleum refinery workers
Phosphate purifiers
Photoengravers
Pyrite burners
Rayon makers
Refrigerant makers
Septic tank cleaners
Sewage treatment plant workers
Sewer workers
Sheep dippers
Silk makers
Slaughterhouse workers
Soap makers
Sugar beet processors
Sulfuric acid purifiers
Sulfur makers
Synthetic fiber makers
Tannery workers
Textile printers
Tunnel workers
Vulcanizers
Well diggers

References

AHLBORG, G.: Hydrogen sulfide poisoning in shale oil industry. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 3: 247, 1951.

FREIREICH, A. W.: Hydrogen sulfide poisoning. Report of two cases, one with fatal outcome from associated mechanical asphyxia. *Am. J. Path.* 22: 147, 1946.

HAGGARD, H. W.: The toxicology of hydrogen sulfide. *J. Indust. Hyg.* 7: 113, 1925.

HENDERSON, Y. AND HAGGARD, H. W.: *Noxious Gases and the Principles of Respiration Influencing Their Action.* 2nd ed. Reinhold Publishing Corp., New York, 1943.

MCCABE, L. C. AND CLAYTON, G. D.: Air pollution by hydrogen sulfide in Poza Rica,

Mexico, An evaluation of the incident of Nov. 24, 1950. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 199, 1952.

MILBY, T. H.: Hydrogen sulfide intoxication; review of the literature and report of unusual accident resulting in two cases of nonfatal poisoining. *J. Occup. Med.* 4: 431. 1962.

YANT, W. P.: Hydrogen sulfide in industry; occurrence, effects, and treatment. *Am. J. Pub. Health* 20: 598, 1930.

(104) Hydroquinone

quinol, hydroquinol, paradiphenol, hydrochinone, dihydroxybenzene

Harmful Effects

Local Contact dermatitis due to either primary irritation or allergic hypersensitivity. Eye irritation manifested by conjunctivitis, lacrimation, photophobia, corneal stains, and opacities. Reddish discoloration of hair and exposed skin. Skin may be depigmented by contact with hydroquinone as well as with the monobenzyl ether derivative.

Route of Entry Inhalation of dust.

Systemic Based on symptoms following ingestion, inhalation of hydroquinone may produce blurred speech, tinnitus, dyspnea, tremors, convulsions, cyanosis from methemoglobinemia, and hemolytic anemia.

Special Diagnostic Test

Detection of hydroquinone in urine. *See* Von Oettingen, 1958.

Recommended Threshold Limit

2 milligrams per cubic meter of air.

Potential Occupational Exposures

Ceramic decorators
Drug makers
Dye makers
Fatty oil processors
Fur dyers
Hydroquinone workers
Lubricating oil workers
Motor fuel blenders
Paint makers
Photographic developer makers
Plastic makers
Plastic stabilizer workers
Rubber coating workers
Stone coating workers
Styrene monomer workers
Textile coating workers
Varnish makers

References

ANDERSON, B. AND OGLESBY, F.: Corneal changes from quinone-hydroquinone exposure. *A.M.A. Arch. Ophthalmol.* 59: 495, 1958.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(105) Iron Compounds

Harmful Effects

Local Ferric chloride, ferric ferrocyanide, and ferric sesquichloride are known skin sensitizers.

Route of Entry Inhalation of dust.

Systemic Iron salts may irritate respiratory tract. Iron oxide, when inhaled, may produce roentgenographic changes in lungs which resemble silicosis. This condition is referred to as siderosis and is thought to be benign. *See* Pulmonary Siderosis, Pneumoconioses section. Iron carbonyl is a liquid with highly toxic vapors which, upon inhalation, may produce extreme pulmonary irritation.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Iron oxide fume) 15 milligrams per cubic meter of air.

Potential Occupational Exposures

Arc cutters
Arc welders
Bessemer operators
Buttwelders
Electric arc welders
Electric furnace operators
Flame cutters
Friction saw operators
Iron workers
Metalizers
Oxyacetylene cutters
Seam welders
Stainless steel makers
Steel foundry workers

(106) Isopropyl Acetate

Harmful Effects

Local Vapor can be irritating to conjunctiva and to mucous membranes of upper respiratory tract.

Route of Entry Inhalation of vapor.

Systemic No ill effects from use of isopropyl acetate in industry have been recorded. Vapors can produce central nervous system depression following excessive exposure.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Dope processors
Fat processors
Gum processors
Isopropyl acetate workers
Lacquerers
Lacquer makers
Leather makers, artificial
Nitrocellulose makers
Oil processors
Organic chemical synthesizers
Perfume makers
Plastic makers
Resin makers
Silk makers
Solvent workers
Wax makers

(107) Isopropyl Alcohol

isopropanol, 2-propanol, secondary propyl alcohol, dimethyl-carbinol

Harmful Effects

Local Inhalation of vapor can produce mild irritation of conjunctiva and mucous membranes of upper respiratory tract.

Route of Entry Inhalation of vapor.

Systemic No industrial poisoning has been recorded. Isopropyl alcohol is potentially narcotic.

Special Diagnostic Tests

Analysis of isopropyl alcohol and acetone in blood, urine and body tissues. *See* Patty, 1949.

Recommended Threshold Limit

400 parts per million parts of air by volume or 980 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetone makers
Alkaloid processors
Antifreeze makers
Cosmetic makers
De-icing compound makers
Drug makers
Gasoline makers
Glass makers
Gum processors
Ink makers
Isopropyl alcohol workers
Laboratory workers, chemical
Lacquerers
Lacquer makers
Nurses
Oil processors
Perfume makers
Photographic film developers
Physicians
Resin makers
Rocket fuel handlers
Rocket fuel makers
Solvent workers
Stainers
Stain makers
Varnish makers

References

HENSON, E. V.: The toxicology of some aliphatic alcohols; part 2. *J. Occup. Med.* 2: 497, 1960.

PATTY, F. A. (EDITOR): *Industrial Hygiene and Toxicology.* 1st ed., vol. 2. Interscience Publishers, New York, 1949.

(108) Kerosine (kerosene)

Harmful Effects

Local Contact with liquid may produce primary skin irritation.

Route of Entry Inhalation of vapor; ingestion of liquid.

Systemic Toxic manifestations include central nervous system depression and pneumonia. Pulmonary effects may follow aspiration of liquid accidentally ingested.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Farmers
Garage workers
Heating fuel handlers
Insecticide workers
Jet fuel handlers
Jet fuel makers
Kerosine workers
Metal cleaners
Petroleum refinery workers
Rocket fuel handlers
Rocket fuel makers

Reference

HENSON, E. V.: Toxicology of some of the aliphatic and alicyclic hydrocarbons. *J. Occup. Med.* 1: 105, 1959.

(109) Ketones

Commonly used ketone solvents include

acetone (dimethyl ketone, beta-ketopropane, pyroacetic ether)
butanone (methyl ethyl ketone, MEK, ethyl methyl ketone)
pentanone (methyl propyl ketone, MPK, ethyl acetone)
methyl butyl ketone (propyl acetone)

Harmful Effects

Local These solvents can produce a dry, scaly, and fissured dermatitis after repeated exposure. High vapor concentrations may irritate conjunctive and mucous membranes of nose and throat.

Route of Entry Inhalation of vapor.

Systemic In high concentrations, narcosis is produced, with symptoms of headache, nausea, vomiting, dizziness, incoordination, and unconsciousness.

Special Diagnostic Tests

Acetone, determination of acetone in blood and urine. *See* Von Oettingen, 1958.

Recommended Threshold Limits

Acetone, 1,000 parts per million parts of air by volume or 2,400 milligrams per cubic meter of air.

Butanone, 200 parts per million parts of air by volume or 590 milligrams per cubic meter of air.

Pentanone, 200 parts per million parts of air by volume or 700 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetone

Acetic acid makers
Acetic anhydride makers
Acetone workers
Acetylene cylinder fillers
Adhesive makers
Bronzers
Celluloid makers
Cellulose acetate makers
Chloroform makers
Diacetone alcohol makers
Drug makers
Electronic equipment cleaners
Electronic equipment dryers
Explosive makers
Glycol makers
Iodoform makers
Isoprene makers
Lacquerers
Lacquer makers
Lubricating oil dewaxers
Mesityl oxide makers
Metal cleaners
Methyl isobutyl ketone makers
Methyl methacrylate workers
Painters
Paint makers
Paint remover workers
Paraffin processors
Pesticide makers
Photographic film makers
Phorone makers
Resin makers
Rubber cement workers
Rubber workers
Solvent workers
Stainers
Stain makers
Textile makers
Varnish makers
Varnish remover workers

Butanone

Adhesive makers
Butanone workers
Cellulose cement makers
Cleaning compound makers
Colorless synthetic resin makers
Cosmetic makers
Dewaxers
Dope processors
Drug makers
Dye makers

218-695 O-66-12

Explosive makers
Lacquerers
Lacquer makers
Lacquer remover workers
Leather workers, artificial
Oil processors
Organic chemical synthesizers
Painters
Paint remover makers
Petroleum refinery workers
Photographic film makers
Printers
Printing ink makers
Raincoat makers
Rubber makers
Shoemakers
Smokeless powder makers
Solvent workers
Stainers
Stain makers
Varnish makers
Varnish remover workers
Vinyl raincoat makers

Pentanone

Pentanone workers — Solvent workers

References

HENSON, E. V.: Toxicology of some aliphatic ketones. *J. Occup. Med.* 1: 607, 1959.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(110) Lead

Harmful Effects

Local None.

Routes of Entry Ingestion of dust; inhalation of dust or fume.

Systemic Lead poisoning in industry almost always results from inhalation of lead-containing dust or lead fume. Signs and symptoms of lead poisoning may include abdominal pain (colic) with tenderness, constipation, headache, weakness, muscular aches or cramps, loss of appetite, nausea, vomiting, weight loss, anemia with pallor, and a *lead line* of the gingival margin. Lead palsy and lead encephalopathy resulting from industrial exposure occur infrequently. *See* Tetraethyl Lead, and Tetramethyl Lead.

Special Diagnostic Tests

Analysis of blood and urine for lead, and of urine for coproporphyrins; examination of blood smear for stipple cells. *See* Kehoe, 1951.

Recommended Threshold Limit

0.2 milligram per cubic meter of air.

Potential Occupational Exposures

Babbitters
Battery makers
Bookbinders
Bottle cap makers
Brass founders
Brass polishers
Braziers
Brick burners

Brick makers
Bronzers
Brush makers
Cable makers
Cable splicers
Canners
Cartridge makers
Ceramic makers
Chemical equipment makers
Chippers
Cutlery makers
Demolition workers
Dental technicians
Diamond polishers
Dye makers
Electronic device makers
Electroplaters
Electrotypers
Emery wheel makers
Enamel burners
Enamelers
Enamel makers
Farmers
File cutters
Filers
Flower makers, artificial
Foundry molders
Galvanizers
Glass makers
Glass polishers
Gold refiners
Gun barrel browners
Incandescent lamp makers
Insecticide makers
Insecticide users
Japan makers
Japanners
Jewelers
Junk metal refiners
Lacquer makers
Lead burners
Lead counterweight makers
Lead flooring makers
Lead foil makers
Lead mill workers
Lead miners
Lead pipe makers
Lead salt makers
Lead shield makers
Lead smelters
Lead stearate makers
Lead workers
Linoleum makers
Linotypers
Lithographers
Match makers
Metal burners
Metal cutters
Metal grinders
Metal miners
Metal polishers
Metal refiners
Mirror silverers
Motor fuel blenders
Musical instrument makers
Painters
Paint makers
Paint pigment makers
Patent leather makers
Pearl makers, imitation
Pipe fitters
Plastic workers
Plumbers
Pottery glaze mixers
Pottery workers
Putty makers
Riveters
Roofers
Rubber buffers
Rubber makers
Scrap metal workers
Sheet metal workers
Shellac makers
Ship dismantlers
Shoe stainers
Shot makers
Solderers
Solder makers

Steel engravers
Stereotypers
Tannery workers
Temperers
Tetraethyl lead makers
Tetramethyl lead makers
Textile makers
Tile makers
Tin foil makers
Tinners
Type founders
Typesetters
Varnish makers
Wallpaper printers
Welders
Zinc mill workers
Zinc smelter chargers

References

KEHOE, R. A.: A critical appraisal of current practices in the clinical diagnosis of lead intoxication. *Indust. Med. & Surg.* 20: 253, 1951.

KEHOE, R. A.: Lead poisoning. *In* Cecil, R. L. and Loeb, R. F. (editors): *Textbook of Medicine.* 10th ed. W. B. Saunders Co., Philadelphia, 1959.

SASSI, C.; FINULLI, M., AND NAVA, C.: Saturnism in the processing of lead stearate. *Med. Lavoro* 52: 658, 1961.

SKINNER, H. L., JR.: The lead problem. An outline of current knowledge and opinion. *J. Occup. Med.* 3: 429, 1961.

VARIOUS AUTHORS: *Lead Symposium,* February 25–27, 1963. University of Cincinnati, Cincinnati, Ohio, 1963.

ZIMMER, F. E.: Lead poisoning in scrap-metal workers. *J. Am. Med. Assoc.* 175: 238, 1961.

(111) Lindane. *See* Pesticides Section

(112) Manganese Compounds

Harmful Effects

Local Manganese dust may be irritating to upper respiratory tract.

Route of Entry Inhalation of dust or fume.

Systemic Among the various manganese compounds used in industry, the oxides of manganese have been almost exclusively responsible for the development of disease. Symptoms generally appear between one and two years following initial exposure. Workers appear to vary in their susceptibility to these compounds.

The course of chronic manganese intoxication may be divided into three phases.

(1) Prodromal phase—characterized by insidious onset and subjective symptoms of headache, asthenia, anorexia, apathy, insomnia or somnolence, leg cramps, impotence, and a diminished desire to talk.

(2) Intermediate phase—characterized by objective symptoms and signs of speech disturbances (monotonous speech, slowness, poor articulation, stuttering, blocking, muteness), masklike face, spasmodic laughing, euphoria, slow and clumsy movements, diminished reflexes, and gait disturbances.

(3) Established phase—symptoms and signs may be exaggerated. Gait disturbances may consist of slow, spasmodic, staggering, high-stepping, or swinging gait. Falls are frequent. Tremors of extremities may appear. Central nervous system manifestations are often permanent resulting in partial or total disability.

There is no unanimity of opinion on the relationship between manganese and pneumonia. A report on manganese pneumonitis, published in 1946, disclosed that men exposed to inhalation of oxide dust suffered a pneumonia rate that averaged 26 per thousand workers over the seven years 1938–1945 as compared with 0.73 per thousand in a control group. No permanent pulmonary changes were observed in exposed group, either on clinical or radiologic examination.

Special Diagnostic Tests

Analysis of blood, urine, and feces for manganese. *See Bolton* et al., 1962.

Recommended Threshold Limit

(Manganese) 5 milligrams per cubic meter of air.

Potential Occupational Exposures

Battery makers
Brick makers
Ceramic makers
Copper manganese alloy makers
Drug makers
Dyers
Enamel makers
Feed additive makers
Ferromanganese alloy makers
Fertilizer makers
Fireworks makers
Glass makers
Hydroquinone makers
Ink makers
Linoleum makers
Manganese alloy makers
Manganese ore crushers
Manganese ore miners
Manganese ore smelters
Manganese soap makers
Manganese steel makers
Manganese workers
Match makers
Metal refiners
Organic chemical synthesizers
Paint makers
Permanganate workers
Rubber makers
Textile fiber bleachers
Textile printers
Varnish makers
Water treaters
Welders, electric arc
Wood preservative workers

References

BOLTON, N. E.; CAVENDER, J. D., AND STACK, V. T., JR.: Determination of manganese in biological specimens. *Am. Indust. Hyg. Assoc. J.* 23: 319, 1962.

COTZIAS, G. C.: Manganese in health and disease. *Physiol. Rev.* 38: 503, 1958.

DAVIES, T. A. L.: Manganese pneumonitis. *Brit. J. Indust. Med.* 3: 111, 1946.

FLINN, R. H.; NEAL, P. A.; REINHART, W. H.; DALLA VALLE, J. M.; FULTON, W. B., AND DOOLEY, A. E.: Chronic manganese poisoning in an ore-crushing mill. Pub. Health Bull. No. 247. U.S. Government Printing Office, Washington, D.C., 1940.

PENALVER, R.: Manganese poisoning. *Indust. Med. & Surg.* 24: 1, 1955.

RODIER, J.: Manganese poisoning in Moroccan miners. *Brit. J. Indust. Med.* 12: 21, 1955.

TEPPER, L. B.: Hazards to health; manganese. *New Eng. J. Med.* 264: 347, 1961.

(113) Mercaptans

The mercaptans include butyl mercaptan (butanethiol), ethyl mercaptan (ethanethiol, ethyl sulfhydrate), methyl mercaptan (methanethiol), and perchloromethyl mercaptan.

Harmful Effects

Local Contact dermatitis from primary irritation by liquid.

Route of Entry Inhalation of vapor.

Systemic In acute exposures, mercaptans have a narcotic effect and produce headache, nausea, vomiting, dizziness, and unconsciousness. Strong and disagreeable odors normally prevent overexposure.

Special Diagnostic Test

None.

Recommended Threshold Limit

Butyl mercaptan, 10 parts per million parts of air by volume or 35 milligrams per cubic meter of air.

Ethyl mercaptan, 20 parts per million parts of air by volume or 52 milligrams per cubic meter of air.

Methyl mercaptan, 20 parts per million parts of air by volume or 40 milligrams per cubic meter of air.

Perchloromethyl mercaptan, 0.1 part per million parts of air by volume or 0.8 milligram per cubic meter of air.

Potential Occupational Exposures

Dye makers
Fumigant makers
Fumigators
Mercaptan workers
Methionine makers
Motor fuel blenders
Organic chemical synthesizers
Rubber makers
Skunk trappers
Warning agent workers

Reference

FAIRCHILD, E. J. AND STOKINGER, H. E.: Toxicologic studies on organic sulfur compounds. 1, Acute toxicity of some aliphatic and aromatic thiols (mercaptans). *Am. Indust. Hyg. Assoc. J.* 19: 171, 1958.

(114) Mercury and Compounds

metallic mercury: quicksilver, hydrargyrum

Harmful Effects

Local Certain mercurial compounds are primary skin and mucous membrane irritants. Allergic hypersensitization is seen less frequently.

Routes of Entry Inhalation of vapor. Percutaneous absorption of metal and organic compounds.

Systemic Acute severe exposures may produce abdominal pain, vomiting, diarrhea, gingivitis, pneumonitis, renal damage, and circulatory or respiratory failure.

Chronic excessive eposure to many inorganic mercury compounds may result in one or more of the three classical signs of gingivitis, tremor, and emotional instability. Headaches, insomnia, digestive disturbances, renal damage, hearing impairment, restriction of visual fields, and crystalline lens discoloration have also been described.

Toxicity resulting from exposure to certain organic mercurials, such as diethyl mercury and methyl mercury iodide, can often be differentiated from inorganic mercury toxicity. This condition is characterized by ataxia, tremor, dysarthria, impaired hearing, paresthesias, emotional instability, and restriction of visual fields.

Permanent sequelae may occur following either acute or chronic intoxication from inorganic or organic mercurial compounds.

Special Diagnostic Test

Analysis of urine for mercury. *See* Kopp and Keenan, 1963.

Recommended Threshold Limit

Mercury, 0.1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Mercury (*organic compounds*), 0.01 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Amalgam makers
Bactericide makers
Barometer makers
Battery makers, mercury
Boiler makers
Bronzers
Calibration instrument makers
Cap loaders, percussion
Carbon brush makers
Caustic soda makers
Ceramic workers
Chlorine makers
Dental amalgam makers
Dentists
Direct current meter workers
Disinfectant makers
Disinfectors
Drug makers
Dye makers
Electric apparatus makers

Electroplaters
Embalmers
Explosive makers
Farmers
Fingerprint detectors
Fireworks makers
Fungicide makers
Fur preservers
Fur processors
Gold extractors
Histology technicians
Ink makers
Insecticide makers
Investment casting workers
Jewelers
Laboratory workers, chemical
Lamp makers, fluorescent
Lamp makers, mercury arc
Manometer makers
Mercury workers
Miners, mercury
Neon light makers
Paint makers
Paper makers
Percussion cap makers
Pesticide workers
Photographers
Pressure gage makers
Refiners, mercury
Seed handlers
Silver extractors
Switch makers, mercury
Tannery workers
Taxidermists
Textile printers
Thermometer makers
Vinyl chloride makers
Wood preservative workers

References

BATTIGELLI, M. C.: Mercury toxicity from industrial exposure. A critical review of the literature. *J. Occup. Med.* 2: 337 and 394, 1960.

GOLDWATER, L. J.; JACOBS, M. B., AND LADD, A. C.: Absorption and excretion of mercury in man. 1, Relationship of mercury in blood and urine. *Arch. Environ. Health* 5: 537, 1962.

GRIEVE, W. T. AND WARD, W. M.: Report on organic mercury hazard to personnel involved in the testing and grading of seed grains. *Occup. Health Rev.* (Ottawa) 14(3): 14, 1962.

KOPP, J. F. AND KEENAN, R. G.: Determination of submicrogram quantities of mercury in urine by ion exchange separation. *Am. Indust. Hyg. Assoc. J.* 24: 1, 1963.

KURLAND, L. T.; FARO, S. N., AND SIEDLER, H.: Minamata disease; the outbreak of a neurologic disorder in Minamata, Japan, and its relationship to the ingestion of seafood contaminated by mercuric compounds. *World Neurology* 1: 370, 1960.

(115) Methyl Alcohol

methanol, carbinol, wood alcohol, wood spirit

Harmful Effects

Local Contact with liquid can produce a dry, scaly, and fissured dermatitis. Both liquid and vapor irritate mucous membranes of eyes, nose, and throat.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Toxic effect of methyl alcohol on optic nerve is mediated through its oxidation product, formaldehyde, and may result in blurring of vision, pain in eyes, loss of central vision, or blindness. Other central

nervous system effects result from narcosis and include headache, nausea, giddiness, and loss of consciousness. Another oxidation product, formic acid, may produce acidosis. Severe intoxication may produce kidney and liver damage. Inhalation of vapor may irritate respiratory tract and produce bronchitis or broncho-pneumonia.

Special Diagnostic Tests

Determination of methyl alcohol in blood, and methyl alcohol and formic acid in urine. Estimation of alkali reserve which may be impaired because of acidosis. *See* Von Oettingen, 1958.

Recommended Threshold Limit

200 parts per million parts of air by volume or 260 milligrams per cubic meter of air.

Potential Occupational Exposures

Acetic acid makers
Adhesive workers
Alcohol distillery workers
Alcohol lamp users
Aldehyde pumpmen
Antifreeze workers
Art glass workers
Automobile painters
Aviation fuel handlers
Aviation fuel makers
Bookbinders
Bronzers
Brush makers
Denatured alcohol workers
Dimethyl sulfate makers
Drug makers
Dry cleaners
Dye makers
Dyers
Ester makers
Explosive workers
Feather workers
Felt hat makers
Flower makers, artificial
Formaldehyde makers
Foundry workers
Furniture polishers
Gilders
Glass makers, safety
Hectograph operators
Incandescent lamp makers
Ink makers
Japan makers
Japanners
Jet fuel workers
Lacquerers
Lacquer makers
Lasters
Leather workers
Linoleum makers
Lithographers
Metal polishers
Methyl acrylate makers
Methyl alcohol workers
Methyl amine makers
Methylation workers
Methyl bromide makers
Methyl chloride makers
Methyl methacrylate makers
Millinery workers
Motor fuel blenders
Organic chemical synthesizers
Painters
Paint makers
Paint remover workers
Patent leather makers
Perfume makers
Photoengravers

Photographic film makers
Polish makers
Printers
Rayon makers
Resin makers
Rocket fuel handlers
Rocket fuel makers
Rubber shoe cementers
Rubber workers
Shellackers
Shellac makers
Shoe factory workers
Shoe finishers
Shoe heel coverers, wood
Shoe stitchers
Soap makers
Solvent workers
Straw hat makers
Sugar refiners
Textile printers
Type cleaners
Upholsterers
Vacuum tube makers
Varnish workers
Vulcanizers
Wood alcohol distillers
Wood stainers
Wood stain makers

References

KEENEY, A. H. AND MELLINKOFF, S. M.: Methyl alcohol poisoning. *Ann. Int. Med.* **34**: 331, 1951.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(116) Methyl Bromide (Bromomethane). *See* Bromine and Compounds

(117) Methyl Butyl Ketone. *See* Ketones

(118) Methyl Chloride

monochloromethane, chloromethane

Harmful Effects

Local Evaporation of liquid from skin produces frostbite. Liquid also damages eye.

Route of Entry Inhalation of gas or vapor.

Systemic Methyl chloride acts as narcotic and also damages liver, kidneys, bone marrow, and central nervous system. Central nervous system effects are characteristically delayed and include headache, dizziness, vomiting, blurred or double vision, mental confusion, drowsiness, convulsion, unconsciousness, and death. Recovery may be erratic.

Special Diagnostic Test

None.

Recommended Threshold Limit

100 parts per million parts of air by volume or 210 milligrams per cubic meter of air.

Potential Occupational Exposures

Aerosol packagers
Drug makers
Flavor extractors
Low temperature polymerization workers
Low temperature solvent workers
Methylation workers
Methyl cellulose makers
Organic chemical synthesizers
Petroleum refinery workers
Polystyrene foam makers
Refrigeration workers
Rubber makers
Silicone makers
Thermometer makers, vapor pressure
Thermometric equipment makers
Thermostatic equipment makers

References

HANSEN, H.; WEAVER, N. K., AND VENEBLE, F. S.: Methyl chloride intoxication. Report of fifteen cases. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 328, 1953.

MACKIE, I. J.: Methyl chloride intoxication. *Med. J. Australia* 1: 203, 1961.

(119) Methyl Chloroform

1,1,1-trichloroethane

Harmful Effects

Local Liquid and high vapor concentrations will irritate eyes on contact. Repeated skin contact will produce a dry, scaly, and fissured dermatitis.

Route of Entry Inhalation of vapor.

Systemic Narcotic effects of dizziness, incoordination, drowsiness, and unconsciousness have been produced by acute exposure to vapor concentrations approaching 1,000 p.p.m. If the worker is not removed after he has been overcome, death can result from respiratory failure or possibly ventricular arrhythmia. Fatty degeneration of liver occurred in laboratory animals undergoing chronic exposure to high concentrations. In human subjects transient elevation of urinary urobilinogen has been noted following exposure to anesthetic concentrations.

Special Diagnostic Test

Infrared analysis of blood for 1,1,1-trichloroethane. *See* Stewart et al., 1961.

Recommended Threshold Limit

350 parts per million parts of air by volume or 1,900 milligrams per cubic meter of air.

Potential Occupational Exposures

Dry cleaners
Machinery cleaners
Metal degreasers
Stain removers

References

STEWART, R. D.; GAY, H. H.; ERLEY, D. S.; HAKE, C. L., AND SCHAFFER, A. W.: Human exposure to 1,1,1-trichloroethane vapor; relationship of expired air and blood concentrations to exposure and toxicity. *Am. Indust. Hyg. Assoc. J.* 22: 252, 1961.

TORKELSON, T. R.; OYEN, F.; MCCOLLISTER, D. D., AND ROWE, V. K.: Toxicity of 1,1,1-trichloroethane as determined on laboratory animals and human subjects. *Am. Indust. Hyg. Assoc. J.* 19: 353, 1958.

(120) Methylene Chloride

dichloromethane, methylene dichloride, methylene bichloride

Harmful Effects

Local Repeated contact with this solvent will cause a dry, scaly, and fissured dermatitis. Liquid and vapor are irritating to eyes and upper respiratory tract.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Methylene chloride acts as narcotic in high concentrations, causing headache, nausea, vomiting, drowsiness, incoordination, paresthesias, and coma. High concentrations may also produce bronchitis and pulmonary edema.

Special Diagnostic Test

None.

Recommended Threshold Limit

500 parts per million parts of air by volume or 1,750 milligrams per cubic meter of air.

Potential Occupational Exposures

Aerosol packagers
Alkaloid processors
Anesthetic makers
Bitumen processors
Cellulose acetate workers
Cellulose ester workers
Cellulose ether workers
Crude rubber workers
Degreasers
Dentists
Drug makers
Dye makers
Fat extractors
Fire extinguisher workers
Flavoring makers
Fumigant makers
Fumigators
Lacquerers
Lacquer workers
Leather finish workers
Methylene chloride workers
Oil extractors
Oil processors
Organic chemical synthesizers
Paint remover workers
Perfume makers
Photographic film makers
Refrigeration workers
Resin makers
Rubber workers
Solvent workers
Stain removers

Textile finishers
Varnish remover workers
Wax makers
Wax removers

References

IRISH, D. D.: Common chlorinated aliphatic hydrocarbon solvents. *Arch. Environ. Health* 4: 320, 1962.

MOSKOWITZ, S. AND SHAPIRO, H.: Fatal exposure to methylene chloride vapor. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 116, 1952.

(121) Methyl Formate

methyl methanoate

Harmful Effects

Local High concentrations are irritating to mucous membranes of upper respiratory tract. Repeated contact can produce a dry, scaly, and fissured dermatitis.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Vapors of methyl formate may have a narcotic effect. Systemic toxicity in industry is unusual.

Special Diagnostic Test

None.

Recommended Threshold Limit

100 parts per million parts of air by volume or 250 milligrams per cubic meter of air.

Potential Occupational Exposures

Cellulose acetate workers
Fumigant makers
Fumigators
Grain fumigators
Methyl formate workers
Organic chemical synthesizers
Pesticide workers
Tobacco fumigators

References

VON OETTINGEN, W. F.: The aliphatic acids and their esters: toxicity and potential dangers; the saturated monobasic aliphatic acids and their esters. *A.M.A. Arch. Indust. Health* 20: 517, 1959.

(122) Methyl Mercaptan. *See* Mercaptans

(123) Molybdenum and Compounds

Harmful Effects

Local Unknown.

Route of Entry Inhalation of dust or fume.

Systemic No human cases of industrial toxicity following exposure to molybdenum or its compounds have been reported. Animal studies with

molybdenite, molybdic oxide, ammonium molybdate, calcium molybdate, and metallic molybdenum indicate a low order of toxicity. Available information concerning chronic exposure to molybdenum compounds is insufficient to define a health hazard.

Special Diagnostic Test

Analysis of blood and urine for molybdenum. *See* Fairhall, 1957.

Recommended Threshold Limits

Molybdenum (soluble compounds), 5 milligrams per cubic meter of air.
Molybdenum (insoluble compounds), 15 milligrams per cubic meter of air.

Potential Occupational Exposures

Ceramic makers
Coal technologists
Drug makers
Dye makers
Electric arc welders
Electroplaters
Ferroalloy workers
Fertilizer makers
Glass makers
Lubricant makers
Metal platers
Molybdenum iron workers
Molybdenum ore miners
Molybdenum ore smelters
Molybdenum sheet makers
Molybdenum steel workers
Molybdenum wire makers
Molybdenum workers
Petroleum refinery workers
Pigment makers
Steel alloy makers
Tannery workers
Vacuum tube makers

References

FAIRHALL, L. T.; DUNN, R. C.; SHARPLESS, N. E., AND PRITCHARD, E. A.: The toxicity of molybdenum. Pub. Health Bull. No. 293. U.S. Government Printing Office, Washington, D.C., 1945.

FAIRHALL, L. T.: *Industrial Toxicology*. 2nd ed. Williams & Wilkins Co., Baltimore, 1957.

(124) Naphtha

Petroleum naphtha (ligroin, benzine, petroleum ether, petroleum benzine)
Coal tar naphtha (hi-flash naphtha)

Certain petroleum naphthas contain varying amounts of benzene. It is known that a potential benzene hazard is associated with the use of such naphthas. *See* Benzene.

Harmful Effects

Local The naphthas are irritating to skin, conjunctiva, and mucous membranes of upper respiratory tract.

Routes of Entry Inhalation of vapor. Percutaneous absorption of liquid is probably not important in development of systemic illness.

Systemic The naphthas may produce symptoms and signs of central nervous system depression similar to those resulting from gasoline intoxication. Coal tar naphtha, a mixture of aromatic hydrocarbons, including toluene, xylene, and pseudocumene has a greater propensity to produce toxicity than petroleum naphtha, consisting principally of a mixture of paraffin hydrocarbons.

Special Diagnostic Test

None.

Recommended Threshold Limit

Naphtha (petroleum), 500 parts per million parts of air by volume or 2,000 milligrams per cubic meter of air.

Naphtha (coal tar), 200 parts per million parts of air by volume or 800 milligrams per cubic meter of air.

Potential Occupational Exposures

Detergent makers
Dry cleaners
Fat processors
Insecticide workers
Laboratory workers, chemical
Metal degreasers
Naphtha workers
Oil processors
Painters
Paint makers
Petroleum refinery workers
Photographic chemicals makers
Rubber coaters
Rubber makers
Solvent workers
Stainers
Stain makers
Varnish makers
Wax makers
Wool processors
Xylene makers

References

ELKINS, H. B.; COMPRONI, E. M., AND PAGNOTTO, L. D.: Industrial benzene exposure from petroleum naphtha. 2, Pertinent physical properties of hydrocarbon mixtures. *Am. Indust. Hyg. Assoc. J.* 24: 99, 1963. Consideration should be given to the benzene exposure possibly resulting from the use of petroleum naphtha containing as little as 2 percent benzene by weight.

GERARDE, H. W.: *Toxicology and Biochemistry of Aromatic Hydrocarbons.* Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1960.

PAGNOTTO, L. D.; ELKINS, H. B.; BRUGSCH, H. G., AND WALKLEY, J. E.: Industrial benzene exposure from petroleum naphtha. 1, Rubber coating industry. *Am. Indust. Hyg. Assoc. J.* 22: 417, 1961. Benzene in certain naphtha solvents varies up to 9.3 percent by weight. Saturators, using naphthas containing more than 3 percent benzene, showed excessive benzene exposure; for churn men the exposure was lower and for spreaders lower than that for churn men.

(125) Naphthalene

naphthalin, naphthene, moth flakes, tar camphor, white tar

Harmful Effects

Local Contact dermatitis from primary irritation or allergic hypersensitivity, or both. Eye irritation from vapors.

Route of Entry Inhalation of vapor or dust.

Systemic High concentrations can produce headache, nausea, vomiting, profuse perspiration, optic neuritis, and hematuria. Prolonged exposure to high concentrations can produce opacity of lens.

Special Diagnostic Tests

Determination of naphthalene in urine and blood. Heinz bodies may be seen in erythrocytes. *See* Von Oettingen, 1958.

Recommended Threshold Limit

(Tentative) 10 parts per million parts of air by volume or 50 milligrams per cubic meter of air.

Potential Occupational Exposures

o-Aminobenzoic acid makers
Beta naphthol makers
Celluloid makers
Coal tar workers
Cutting fluid workers
Dye chemical makers
Dye intermediate makers
Fumigant workers
Fungicide makers
Hydronaphthalene makers
Insecticide workers
Lampblack makers
Lubricant workers
Moth repellent workers
Naphthalene workers
Phthalic anhydride makers
Resin makers
Scintillation counter makers
Smokeless powder makers
Soil treaters
Tannery workers
Textile chemical makers

References

GERARDE, H. W.: *Toxicology and Biochemistry of Aromatic Hydrocarbons.* Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1960.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(126) Naphthylamine (Beta)

Pennsylvania was the first State to adopt a regulation for the control of this material. On October 27, 1961 the following section was adopted and made a part of Chapter 4, Article 434 (Regulations for Control of Dangerous Materials) of the Rules and Regulations of the Commonwealth of Pennsylvania Department of Health:

Section 1. Beta-naphthylamine

No person, corporation, partnership or association shall manufacture, use or permit to be used, store, transport, or otherwise handle beta-naphthylamine. Any area where beta-naphthylamine has been used, stored, or otherwise handled shall be decontaminated to assure that no individual shall be exposed.

Harmful Effects

Local Beta-naphthylamine is mildly irritating to skin and has produced contact dermatitis.

Routes of Entry Inhalation of dust and percutaneous absorption.

Systemic A metabolite, the 1-hydroxy derivative of beta-naphthylamine, is a potent carcinogen. The metabolite acts on urinary bladder mucosa causing cystitis and papillomata which may become malignant. Symptoms are frequent urination, dysuria, and hematuria, which appear after several years of exposure or several years after last exposure. Alpha-naphthylamine is unimportant toxicologically except for its frequent contamination by beta-naphthylamine.

Special Diagnostic Test

Analysis of urine for naphthylamine. *See* Von Oettingen, 1958.

Recommended Threshold Limit

Not established.

Potential Occupational Exposure

Dye makers beta-Naphthylamine workers

References

CASE, R. A. M.; HOSKER, M. E.; MC DONALD, D. B., AND PEARSON, J. T.: Tumors of the urinary bladder in workmen engaged in the manufacture and use of certain dyestuff intermediates in the British chemical industry. 1, The role of aniline, benzidine, alpha-naphthylamine, and beta-naphthylamine. *Brit. J. Indust. Med.* 11: 75, 1954.

VIGLIANI, E. C. AND BARSOTTI, M.: Environmental tumors of the bladder in some Italian dyestuff factories. *Med. Lavoro* 52: 241, 1961.

VON OETTINGEN, W. F.: The aromatic amino and nitro compounds, their toxicity and potential dangers. A review of the literature. Pub. Health Bull. No. 271. U.S. Government Printing Office, Washington, D.C., 1941.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(127) Natural Gas

Natural gas consists primarily of methane (85 percent) with lesser amounts of ethane (9 percent), propane (3 percent), nitrogen (2 percent), and butane (1 percent).

218-695 O-66-13

Harmful Effects

Local None.

Route of Entry Inhalation of gas.

Systemic Displacement of air by the gas may lead to shortness of breath, unconsciousness, and death from hypoxemia. A mild central nervous system depressant effect has been attributed to the homologs of methane in the gas. Incomplete combustion may result in production of carbon monoxide.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Acetaldehyde makers
Acetylene makers
Ammonia makers
Carbon black makers
Coal miners
Ethanol makers
Formaldehyde makers
Gas fuel users
Helium extractors
Hydrocarbon fuel makers
Hydrogenated oil makers
Hydrogen makers
Methanol makers
Natural gas workers
Nitric acid makers
Organic chemical synthesizers
Petroleum refinery workers
Power plant workers, electric
Synthesis gas makers
Vinyl chloride makers

Reference

HENSON, E. V.: Toxicology of some of the aliphatic and alicyclic hydrocarbons. *J. Occup. Med.* 1: 105, 1959.

(128) Nickel and Compounds

Harmful Effects

Local Nickel salts produce allergic contact dermatitis. A type of dermatitis referred to as *nickel itch* may be seen in in nickel miners, smelters, and refiners. This condition is characterized by an erythematous, papular, pruritic rash, often beginning in web of fingers and spreading to fingers, wrists, and forearms.

Route of Entry Inhalation of dust or fume.

Systemic Nickel carbonyl is thought to be the most toxic of nickel compounds. *See* Carbonyls. Metallic nickel and its salts are considered to be of very low level of toxicity when taken into body. There has been reported an increase in incidence of cancer of lung and ethmoid sinuses in men exposed to dust in nickel refining.

Special Diagnostic Tests

Analysis of blood and urine for nickel. *See* Kincaid et al., 1956.

Recommended Threshold Limit

(Nickel carbonyl) 0.001 part per million parts of air by volume or 0.007 milligram per cubic meter of air.

Potential Occupational Exposures

Battery makers, storage
Cemented carbide makers
Ceramic makers
Disinfectant makers
Dyers
Electroplaters
Enamelers
Gas mask makers
Ink makers
Jewelers
Magnet makers
Mond process workers
Nickel alloy makers
Nickel miners
Nickel refiners
Nickel smelters
Nickel workers
Oil hydrogenators
Organic chemical synthesizers
Paint makers
Pen point makers
Petroleum refinery workers
Spark plug makers
Steel makers, stainless
Textile dyers
Vacuum tube makers
Varnish makers

References

DOLL, R.: Cancer of the lung and nose in nickel workers. *Brit. J. Indust. Med.* 15: 217, 1958.

KINCAID, J. F.; STANLEY, E. L.; BECKWORTH, C. H., AND SUNDERMAN, F. W.: Nickel poisoning. 3, Procedures for detection, prevention, and treatment of nickel carbonyl exposure including a method for the determination of nickel in biologic materials. *Am. J. Clin. Path.* 26: 107, 1956.

MORGAN, J. G.: Some observations on the incidence of respiratory cancer in nickel workers. *Brit. J. Indust. Med.* 15: 224, 1958.

SUNDERMAN, F. W. AND KINCAID, J. F.: Nickel poisoning. 2, Studies on patients suffering from acute exposure to vapors of nickel carbonyl. *J. Am. Med. Assoc.* 155: 889, 1954.

SUNDERMAN, F. W. AND SUNDERMAN, F. W., JR.: Loeffler's syndrome associated with nickel sensitivity. *Arch. Int. Med.* 107: 405, 1961.

(129) Nickel Carbonyl

nickel tetracarbonyl

Harmful Effects

Local Contact dermatitis, possibly allergic, has been reported.

Route of Entry Inhalation of vapor.

Systemic Acute intoxication from nickel carbonyl vapor is due to irritation of lungs and to toxic effect on central nervous system. Early effects following acute exposure consist of headache, giddiness, unsteady

gait, nausea, vomiting, and a dry cough. A latent period of several hours to days may follow initial symptoms. Delayed symptoms of retrosternal pain, chest tightness, cough, dyspnea, extreme weakness, convulsions, hallucinations, delirium, nausea, and vomiting may terminate with pulmonary edema and respiratory or circulatory failure. Long exposure to low concentrations of nickel carbonyl is suspected of causing an increased incidence of carcinoma of respiratory tract including nasal sinuses.

Special Diagnostic Tests

Analysis of blood and urine for nickel. *See* Sunderman and Kincaid, 1954.

Recommended Threshold Limit

0.001 part per million parts of air by volume or 0.007 milligram per cubic meter of air.

Potential Occupational Exposures

Foundry workers
Gas platers
Mond process workers
Nickel carbonyl workers
Petroleum refinery workers

References

HUEPER, W. C.: Carcinogens in the human environment. *Arch. Path.* 71: 237, 1961.

SUNDERMAN, F. W. and KINCAID, J. F.: Nickel poisoning. 2, Studies on patients suffering from acute exposure to vapors of nickel carbonyl. *J. Am. Med. Assoc.* 155: 889, 1954.

(130) Nicotine. *See* Pesticides Section

(131) Nitric Acid

aqua fortis, hydrogen nitrate

Harmful Effects

Local Very corrosive. Capable of producing severe burns, ulcers and necrosis of skin, mucous membranes and eyes. Prolonged exposure to vapor may cause yellowing of skin and erosion of teeth.

Route of Entry Inhalation of vapor.

Systemic Inhalation may cause irritation of entire respiratory tract. Pulmonary edema may result. Pulmonary fibrosis has been reported to follow inhalation.

Special Diagnostic Test

None.

Recommended Threshold Limit

10 parts per million parts of air by volume or 25 milligrams per cubic meter of air.

Potential Occupational Exposures

Aircraft workers
Ammonium nitrate makers
Bleachers
Brass cleaners
Bright-dip workers
Cellulose nitrate makers
Drug makers
Dye makers
Electroplaters
Etchers
Explosive makers
Jewelers
Laboratory workers, chemical
Lithographers
Mirror makers
Nitration workers
Nitric acid workers
Nitrobenzene makers
Nitro-compound workers
Ore flotation workers
Organic chemical synthesizers
Photoengravers
Rocket fuel handlers
Rock phosphate acidulators
Steel etchers
Sulfuric acid makers

(132) Nitrobenzene

nitrobenzol, essence of mirbane, oil of mirbane, oil of bitter almonds

Harmful Effects

Local Nitrobenzene may produce contact dermatitis through primary irritation or allergic hypersensitization.

Route of Entry Inhalation of vapor or percutaneous absorption of liquid.

Systemic Nitrobenzene converts hemoglobin to methemoglobin, resulting in headache, dizziness, shortness of breath, and a bluish-discoloration of the lips (blue-lip), mucous membranes, and skin. Central nervous system depression, anemia, and liver damage may occur with acute or chronic intoxication.

Special Diagnostic Tests

Analysis of urine for nitrophenol, and of blood for methemoglobin. *See* Salmowa et al., 1963, and Von Oettingen, 1941.

Recommended Threshold Limit

1 part per million parts of air by volume or 5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Aniline makers
Azobenzene makers
Benzidine makers
Explosive workers
Glue makers
Ink makers
Lacquer makers
Metal polish makers
Nitrobenzene workers
Organic chemical synthesizers
Petroleum refinery workers
Quinoline makers
Shoe polish makers
Soap makers
Stainers
Stain makers
Vanillin makers

References

SALMOWA, J.; PIOTROWSKI, J., AND NEUHORN, U.: Evaluation of exposure to nitrobenzene; absorption of nitrobenezene vapour through lungs and excretion of p-nitrophenol in urine. *Brit. J. Indust. Med.* 20: 41, 1963.

VON OETTINGEN, W. F.: The aromatic amino and nitro compounds, their toxicity and potential dangers. A review of the literature. Pub. Health Bull. No. 271. U.S. Government Printing Office, Washington, D.C., 1941.

(133) Nitroethane. *See* Nitroparaffins

(134) Nitrogen Oxides

The most common oxides of nitrogen include nitrous oxide (N_2O, dinitrogen monoxide), nitric oxide (NO, nitrogen monoxide), nitrogen dioxide (NO_2) which usually consists of an equilibrium mixture of nitrogen dioxide and nitrogen tetroxide (N_2O_4, dinitrogen tetroxide). Nitrogen trioxide (N_2O_3, dinitrogen trioxide) dissociates into nitric oxide and nitrogen dioxide. Nitrogen pentoxide (N_2O_5, dinitrogen pentoxide), upon contact with air, decomposes into nitrogen dioxide and oxygen.

(1) Nitrous Oxide

dinitrogen monoxide, nitrogen monoxide, factitious air, hyponitrous acid anhydride, laughing gas

Harmful Effects

Local None.

Route of Entry Inhalation of gas.

Systemic Mild anesthetic action.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Aerosol packagers, food
Dental technicians
Dentists
Medical technicians
Nitrous oxide workers
Nurses
Physicians
Rocket fuel makers

(2) Nitric Oxide

At ordinary temperatures, nitric oxide combines with atmospheric oxygen to form nitrogen dioxide.

(3) Nitrogen Tetroxide

dinitrogen tetroxide

Nitrogen tetroxide is an equilibrium mixture of nitrogen tetroxide and nitrogen dioxide.

Harmful Effects

Local Nitrogen tetroxide is an extremely corrosive liquid and may cause severe burns.

Route of Entry Inhalation of gas.

Systemic Effects are due to inhalation of the emitted nitrogen dioxide gas.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Nitrogen dioxide) 5 parts per million parts of air by volume or 9 milligrams per cubic meter of air.

Potential Occupational Exposure

Nitrogen tetroxide workers Rocket fuel makers.

(4) Nitrogen Dioxide

Harmful Effects

Local Very irritating to eyes and mucous membranes. Prolonged low-level exposures may produce yellowish to brown staining of the teeth and skin.

Route of Entry Inhalation of gas.

Systemic Exposure to high concentrations may produce immediate coughing and chest pain. When lower concentrations are inhaled, there may be only mild signs of bronchial irritation followed by a 5- to 12-hour symptom free period. Subsequently, the onset of signs and symptoms of acute pulmonary edema may be noted. Death often results within 24 hours. If the acute episode is survived, sequelae such as bronchiectasis or emphysema may develop.

Methemoglobinemia, generally of a mild degree, may be produced by exposure to nitrous fumes containing small amounts of nitric oxide.

Nitrogen dioxide may be formed from fresh green silage in amounts which, when restrained in the confines of a silo, may constitute a serious health hazard. The name *silo-filler's disease* has been used to designate the lung condition caused by exposure to nitrogen dioxide evolved in this way.

The following events characterize this disease: Exposure to the gases contained within a recently filled silo has been followed almost immediately by malaise, cough, dyspnea, chest pain, chills, fever, nausea, and vomiting.

These signs and symptoms may last from several days to several weeks. In some cases, complete resolution may occur while in others there may be a progression to severe pulmonary insufficiency and death. A latent period of 2 or 3 weeks between the initial onset of signs and symptoms and the final progression to potentially fatal pulmonary insufficiency has been described.

The chest roentgenogram may reveal a picture varying from one of a diffuse, patchy, confluent infiltration to one of numerous, uniformly scattered, nodular densities ranging in size from 1 to 5 mm in diameter. These roentgenographic patterns may undergo partial or complete clearing as the disease resolves. However, in some cases, there may remain roentgenographic evidence of pulmonary fibrosis.

Pulmonary function studies done after recovery from the initial acute episode may reveal striking variations from the expected normal values. Findings suggestive of obstructive pulmonary emphysema have been reported. These changes include an increase in residual volume, a decreased maximum breathing capacity, and some prolongation of the timed vital capacity. Changes in the diffusion capabilities of the lung do not seem to be significant.

Histologic sections of biopsy or necropsy specimens of lung tissue acquired during the initial acute stages of disease usually indicate extensive bronchopneumonia. During later stages, however, the histopathologic picture is frequently one of bronchiolitis fibrosa obliterans. No significant pathogenic microorganisms have been consistently identified in patients with this disease.

Special Diagnostic Test

None.

Recommended Threshold Limit

5 parts per million parts of air by volume or 9 milligrams per cubic meter of air.

Potential Occupational Exposures

Brass cleaners
Braziers
Bright-dip workers
Bronze cleaners
Copper cleaners
Cotton bleachers
Electric arc welders
Electroplaters
Flour bleachers
Nitrate workers
Nitric acid makers
Nitrogen dioxide workers
Oxidized cellulose (cellulosic acid) compound makers
Pipe fitters
Plasma torch operators
Raw silk bleachers
Rocket fuel makers
Silo fillers
Sulfuric acid makers

References

CLANCY, P. J.; WATSON, S. L., AND REARDAN, J. B.: Nitrogen tetroxide exposure in the missile industry. *J. Occup. Med.* 4: 691, 1962.

GRAYSON, R. R.: Silage gas poisoning; nitrogen dioxide pneumonia, a new disease in agricultural workers. *Ann. Int. Med.* 45: 393, 1956.

LA FLECHE, L. R.; BOLVIN, C., AND LEONARD, C.: Nitrogen dioxide, a respiratory irritant. *Canad. Med. Assoc. J.* 84: 1438, 1961.

LEIB, G. M. P.; DAVIS, W. N.; BROWN, T., AND MC QUIGGAN, M.: Chronic pulmonary insufficiency secondary to silo-filler's disease. *Am. J. Med.* 24: 471, 1958.

LOWRY, T. AND SCHUMAN, L. M.: "Silo fillers disease," a syndrome caused by nitrogen dioxide. *J. Am. Med. Assoc.* 162: 153, 1956.

OFFICE OF DIRECTOR, DEFENSE RESEARCH AND ENGINEERING, DEPARTMENT OF DEFENSE: *The Handling and Storage of Liquid Propellants.* U.S. Government Printing Office, Washington, D.C., 1961.

RAFIL, S. AND GODWIN, M. C.: Silo filler's disease; relapse following latent period. *Arch. Path.* 72: 424, 1961.

(135) Nitroglycerin

nitroglycerol, glyceryl trinitrate, trinitroglycerol

Harmful Effects

Local May cause contact dermatitis because of allergic hypersensitivity or primary irritation or both.

Route of Entry Inhalation of dust or vapor; ingestion of dust; percutaneous absorption of liquid.

Systemic Powerful vasodilatation with resultant flushing of skin and throbbing headache. Blood pressure may be lowered. Visual acuity may be diminished, or total temporary blindness may occur. Methemoglobin is formed but only in small amounts. Transitory mental aberration may occur. Massive exposures may cause loss of consciousness due to peripheral vascular dilatation. It is uncertain as to whether long-term exposure to nitroglycerin has an adverse effect upon the cardiovascular system. Toxic effects of nitroglycerin are accentuated by alcohol ingestion.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Nitroglycerin with ethylene glycol dinitrate) 0.2 part per million parts of air by volume or 2 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Drug makers
Dynamite makers
Missile technicians
Nitroglycerin workers
Rocket fuel makers
Shell fillers
Smokeless powder makers

References

BARSOTTI, M.: Attacks of stenocardia in workers engaged in the production of dynamites with nitroglycol. *Med. Lavoro* 45: 544, 1954.

FORSSMAN, S.; MASRELIEZ, N.; JOHANSSON, G.; SUNDELL, G.; WILANDER, O., AND BOSTROM, G.: The health of workers with nitro-compounds in three Swedish explosives factories. *Arch. Gewerbepath.* 16: 157, 1958.

LAWS, G. C.: The effects of nitroglycerin upon those who manufacture it. *J. Am. Med. Assoc.* 31: 793, 1898.

RABINOWITCH, I. M.: Acute nitroglycerin poisoning. *Canad. Med. Assoc. J.* 50: 199, 1944.

VON OETTINGEN, W. F.: The effects of aliphatic nitrous and nitric acid esters on the physiological functions with special reference to their chemical constitution. Nat. Inst. Health Bull. No. 186. U.S. Government Printing Office, Washington, D.C., 1946.

YEE, H. T.; FOSDICK, L. B., AND BOURNE, H. G., JR.: Nitroglycerin and nitroglycol exposure in an explosives plant. *Am. Indust. Hyg. Assoc. J.* 20: 45, 1959.

(136) Nitromethane. *See* Nitroparaffins

(137) Nitroparaffins

The nitroparaffins include nitromethane, nitroethane, and nitropropane.

Harmful Effects

Local The nitroparaffins are mild irritants to eyes and upper respiratory tract. Nitromethane is mildly irritating to skin.

Route of Entry Inhalation of vapor.

Systemic High concentrations may produce light narcosis and irritation of central nervous system. Liver and kidney damage have been observed in animal experiments. Only one notable industrial exposure has been reported, with symptoms of anorexia, nausea, vomiting, diarrhea, and occipital headache from inhalation of 2-nitropropane.

Special Diagnostic Test

None.

Recommended Threshold Limit

Nitromethane, 100 parts per million parts of air by volume or 250 milligrams per cubic meter of air.

Nitroethane, 100 parts per million parts of air by volume or 310 milligrams per cubic meter of air.

2-Nitropropane, 25 parts per million parts of air by volume or 90 milligrams per cubic meter of air. Same value for 1-Nitropropane.

Potential Occupational Exposures

Alkyd resin makers
Artificial resin makers
Cellulose acetate workers
Cellulose acetobutyrate workers
Cellulose acetopropionate workers
Dye makers
Fat processors
Nitrocellulose workers

Nitroethane workers
Nitromethane workers
Nitropropane workers
Organic chemical synthesizers
Rocket fuel makers
Stainers
Stain makers
Vinyl resin makers
Wax makers

Reference

SKINNER, J. B.: Toxicity of 2-nitropropane. *Indust. Med.* 16: 441, 1947.

(138) Nitrophenols (Ortho-, Meta- and Para-)

Harmful Effects

Local Unknown.

Routes of Entry Percutaneous absorption of liquid; inhalation of vapor.

Systemic No industrial cases of poisoning have been recorded. Exposure to vapors may produce methemoglobinemia. Paranitrophenol is the most toxic of the isomers.

Special Diagnostic Test

Analysis of urine for paranitrophenol. *See* Mountain et al., 1951.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Aminophenol makers
Drug makers
Dye makers
Explosive makers
Fungicide makers
Indicator makers, chemical
Nitroanisole makers
Nitrophenol workers
Organic chemical synthesizers
Photographic chemical makers
Textile makers

References

MOUNTAIN, J. T.; ZLOTOLOW, H., AND O'CONOR, G. T.: Determination of paranitrophenol in urine in parathion poisoning cases. *Indust. Health Monthly* (USPHS) 11: 88, (June) 1951.

VON OETTINGEN, W. F.: The aromatic amino and nitro compounds, their toxicity and potential dangers. A review of the literature. Pub. Health Bull. No. 271. U.S. Government Printing Office, Washington, D.C., 1941.

(139) Nitropropane. *See* Nitroparaffins

(140) Osmium and Compounds

Metallic osmium is considered to be nontoxic.

Harmful Effects

Local Upon heating in air osmium produces osmium tetroxide fume (osmic acid) which is irritating to mucous membranes and eyes. Certain

osmium salts, the chloride for example, may exhibit a caustic effect upon skin.

Route of Entry Inhalation of vapor or fume.

Systemic Inhalation of osmium tetroxide fume has been reported to produce extreme lung irritation, frequently progressing to bronchopneumonia. Asthma-like symptoms have also been reported to occur following inhalation of vapors of certain osmium compounds.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Osmium tetroxide) 0.002 milligram per cubic meter of air.

Potential Occupational Exposures

Alloy makers
Ammonia makers, synthetic
Electric contact makers
Histology technicians
Incandescent lamp makers
Machine bearing makers
Organic chemical synthesizers
Osmium workers
Pen point makers
Phonograph needle makers
Platinum hardeners

Reference

MC LAUGHLIN, A. I. G.; MILTON, R., AND PERRY, K. M. A.: Toxic manifestations of osmium tetroxide. *Brit. J. Indust. Med.* 3: 183, 1946.

(141) Oxalic Acid

dicarboxylic acid, ethane-di-acid, ethanedioic acid

Harmful Effects

Local Corrosive action on skin and mucous membranes may produce ulceration.

Route of Entry Inhalation of mist.

Systemic The calcium-complexing action of oxalate depresses level of ionized calcium in body fluids, producing severe disturbances of heart as well as muscle twitching, cramps, and central nervous system depression. Renal injury is frequently produced by acute poisoning but is rarely the cause of death.

Special Diagnostic Test

Determination of blood calcium levels. *See* Gleason et al., 1957.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Automobile radiator cleaners
Bleachers
Bleach makers
Celluloid makers
Ceramic makers
Coal washers
Cream of tartar makers
Dextrin makers
Drug makers
Dye makers
Dyers
Formic acid makers
Glycerine makers
Hydrocyanic acid makers
Ink makers
Ink remover makers
Laundry workers
Leather bleachers
Lithographers
Metal polish makers
Methanol makers
Organic chemical synthesizers
Oxalic acid workers
Paint remover makers
Paint removers
Paper makers
Photoengravers
Photographic workers
Pigment makers
Rayon bleachers
Rubber makers
Rust remover makers
Rust removers
Stain removers
Stearin makers
Straw hat bleachers
Tannery workers
Tartaric acid makers
Textile dyers
Textile printers
Varnish remover makers
Varnish removers
Wood bleachers
Wood cleaners
Wood cleanser makers

Reference

GLEASON, M. N.; GOSSELIN, R. E., AND HODGE, H. C.; *Clinical Toxicology of Commercial Products; Acute Poisoning (Home and Farm)*. Williams & Wilkins Co., Baltimore, 1957.

(142) Ozone

Harmful Effects

Local Irritant to eyes and mucous membranes.

Route of Entry Inhalation of gas.

Systemic Pulmonary edema and hemorrhage may result from severe exposure. Less severe exposure may produce headache, malaise, shortness of breath, and drowsiness.

Special Diagnostic Test

None.

Recommended Threshold Limit

0.1 part per million parts of air by volume or 0.2 milligram per cubic meter of air.

Potential Occupational Exposures

Air treaters
Arc cutters
Arc welders, argon shielded
Arc welders, electric
Arc welders, heliarc
Arc workers, electric
Bactericide makers
Electroplaters
Food preservers (cold storage)
Hydrogen peroxide makers
Industrial waste treaters
Odor controllers
Oil bleachers
Organic chemical synthesizers
Ozone workers
Photoengravers
Photographers
Plasma torch operators
Sewage gas treaters
Textile bleachers
Ultraviolet lamp workers
Water treaters
Wax bleachers

Reference

STOKINGER, H. E.: Ozone toxicity. A review of the literature through 1953. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 9: 367, 1954.

(143) Pentanone. *See* Ketones

(144) Perchloroethylene

tetrachloroethylene, carbon dichloride, ethylene tetrachloride

Harmful Effects

Local Repeated contact with liquid causes a dry, scaly, and fissured dermatitis. High concentrations produce eye and nose irritation.

Routes of Entry Inhalation of vapor; of lesser importance, percutaneous absorption of liquid.

Systemic Primary systemic effect is narcosis, with symptoms of headache, dizziness, nausea, incoordination, and somnolence. Repeated exposures to high concentrations can produce a mild hepatitis.

Special Diagnostic Test

Analysis of blood for tetrachloroethylene. *See* Stewart, Gay et al., 1961.

Recommended Threshold Limit

100 parts per million parts of air by volume or 670 milligrams per cubic meter of air.

Potential Occupational Exposures

Cellulose ester processors
Cellulose ether processors
Degreasers
Detergent makers
Dope processors
Drug makers (anthelmintics)
Dry cleaners
Electroplaters

Fumigant workers
Gum processors
Heat transfer workers
Metal degreasers
Organic chemical synthesizers
Paraffin processors
Perchloroethylene workers
Printers
Rubber workers
Soap workers
Solvent workers
Tar processors
Vacuum tube makers
Wax makers
Wool scourers

References

COLER, H. R. AND ROSSMILLER, H. R.: Tetrachlorethylene exposure in a small industry. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 227, 1953.

QUERIES AND MINOR NOTES: Toxicity of tetrachloroethylene. *J. Am. Med. Assoc.* 131: 1468, 1946.

STEWART, R. D.; ERLEY, D. S.; SCHAFFER, A. W., AND GAY, H. H.: Accidental vapor exposure to anesthetic concentrations of a solvent containing tetrachloroethylene. *Indust. Med. & Surg.* 30: 327, 1961.

STEWART, R. D.; GAY, H. H.; ERLEY, D. S.; HAKE, C. L., AND SCHAFFER, A. W.: Human exposure to tetrachloroethylene vapor. Relationship of expired air and blood concentrations to exposure and toxicity. *Arch. Environ. Health* 2: 516, 1961.

(145) Perchloromethyl Mercaptan. *See* Mercaptans

(146) Phenol

carbolic acid, phenic acid, phenylic acid, phenyl hydrate, hydroxybenzene, monohydroxybenzene

Harmful Effects

Local A primary irritant possessing strong corrosive properties for all tissues of body.

Route of Entry Inhalation of mist or vapor; percutaneous absorption of mist, vapor, or liquid.

Systemic Acute poisoning is mainly characterized by central nervous system manifestations including tinnitus, vertigo, tremor, excitement, and convulsions. Pneumonia often follows. Chronic phenol poisoning is characterized by headache, fatigue, cough, anorexia, insomnia, nervousness, paresthesias, weight loss, and cachexia. Renal and hepatic damage frequently follow phenol intoxication.

Special Diagnostic Tests

Analysis of urine and blood for phenol. *See* Von Oettingen, 1958.

Recommended Threshold Limit

5 parts per million parts of air by volume or 19 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Battery makers, dry
Coal tar workers
Disinfectant makers
Drug makers
Dye makers
Dyers
Etchers
Explosive workers
Gas workers, illuminating
Gas purifiers
Herbicide makers
Lampblack makers
Lubricating oil processors
Paint makers
Paint remover makers
Paint removers
Pentachlorophenol makers
Perfume makers
Phenol workers
Photographic material workers
Picric acid makers
Resin makers
Rubber reclaimers
Rubber workers
Stillmen, carbolic acid
Surgical dressing makers
Textile printers
Varnish makers
Weed killers
Wood preservers

References

EVANS, S. J.: Acute phenol poisoning. *Brit. J. Indust. Med.* 9: 227, 1952.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(147) Phenylhydrazine

hydrazinobenzene

Harmful Effects

Local Eczematous contact dermatitis from primary irritation as well as allergic sensitivity.

Route of Entry Inhalation of vapor or mist; percutaneous absorption.

Systemic Phenylhydrazine hemolyses red blood cells and is a slight methemoglobin former. It also causes injury to liver, kidneys, and heart. Systemic findings and symptoms include anemia, leukopenia, hepatitis, headache, weakness, dizziness, anorexia, gastritis, diarrhea, hematuria, and albuminuria.

Special Diagnostic Test

None.

Recommended Threshold Limit

5 parts per million parts of air by volume or 22 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Analytical chemists
Antipyrine makers
Drug makers
Dye makers
Nitron makers
Organic chemical synthesizers
Phenylhydrazine workers

Reference

VON OETTINGEN, W. F.: The aromatic amino and nitro compounds, their toxicity and potential dangers. A review of the literature. Pub. Health Bull. No. 271. U.S. Government Printing Office, Washington, D.C., 1941.

(148) Phosgene

carbonyl chloride, carbon oxychloride, combat gas, D-Stoff, chloroformyl chloride

Harmful Effects

Local Contact dermatitis from primary irritation, conjunctival and upper respiratory tract irritation.

Route of Entry Inhalation of gas.

Systemic Acute exposure produces pulmonary edema frequently preceded by a latent period of several hours' duration. Death may result from respiratory or cardiac failure.

Special Diagnostic Test

None.

Recommended Threshold Limit

1 part per million parts of air by volume or 4 milligrams per cubic meter of air.

Potential Occupational Exposures

Chlorinated compound makers
Dye makers
Firemen
Glass makers
Organic chemical synthesizers
Phosgene workers
Resin makers

(149) Phosphine

hydrogen phosphide, phosphoretted hydrogen

Harmful Effects

Local High concentrations of gas are irritating to eyes, nose and skin.

Route of Entry Inhalation of gas.

Systemic Acute effects are secondary to central nervous system depression, irritation of lungs, and damage to kidneys and other organs and include weakness, fatigue, hypotension, bradycardia, headache, dizziness, fainting, drowsiness, thirst, abdominal pain, nausea, vomiting, diarrhea, dyspnea, bronchitis, pulmonary edema, albuminuria, hematuria, tremors, staggering gait, convulsions, coma and death. In chronic poisoning there may be disturbances in sight, speech, and motor functions and effects seen in chronic phosphorus poisoning such as nonhemolytic anemia, brittle teeth, and necrosis of the lower jaw.

218-695 O-66-14

Special Diagnostic Test

None.

Recommended Threshold Limit

0.3 part per million parts of air by volume or 0.4 milligram per cubic meter of air.

Potential Occupational Exposures

Acetylene generator workers
Acetylene workers
Aluminum phosphide workers
Calcium carbide workers
Cement workers
Ferrosilicon workers
Firemen
Grain fumigators
Metal alloy workers
Metal refiners
Metal slag workers
Organic chemical synthesizers
Phosphine workers
Pyrites sulfuric acid workers
Rustproofers
Sodium phosphide workers
Sulfuric acid tank cleaners
Welders
Zirconium diphosphide makers

Reference

HARGER, R. N. AND SPOLYAR, L. W.: Toxicity of phosphine, with a possible fatality from this poison. *A.M.A. Arch. Indust. Health* 18: 497, 1958.

(150) Phosphorus and Compounds

Elemental phosphorus exists in two forms, a red form which is nontoxic, and a yellow or white which is very toxic. Frequently the red is contaminated with small amounts of the yellow.

Harmful Effects

Local Skin contact with yellow phosphorus results in production of severe burns. In addition, the following phosphorus compounds are reported to be potent irritants of skin, eyes, and mucous membranes of nose, throat, and respiratory tract:

Phosphorus trichloride
Phosphorus pentachloride
Phosphorus oxychloride
Phosphorus tribromide
Phosphorus pentabromide
Phosphorus trisulfide
Phosphorus pentasulfide
Phosphorus sesquisulfide
Phosphoric acid

Routes of Entry Ingestion and percutaneous absorption of dust; inhalation of dust or fume.

Systemic Ingestion of yellow phosphorus produces severe poisoning, beginning with local gastrointestinal irritation, progressing to systemic poisoning. Shock may ensue rapidly. If death is not immediate, patient

may succumb later to liver, kidney, or heart failure brought about by direct action of phosphorus on these organs.

Inhalation of fumes produced by the phosphorus compounds listed above may cause irritation of pulmonary tissues with resultant acute pulmonary edema.

Chronic phosphorus poisoning is result of continued absorption of small amounts of yellow phosphorus. This form of intoxication is characterized by periostitis with suppuration, ulceration, necrosis, and severe deformity of the lower jaw. *See* Phosphine.

Special Diagnostic Test

Roentgenographic examination of lower jaw to detect possible necrosis of mandible. *See* Patty, 1958.

Recommended Threshold Limit

Phosphorus (yellow), 0.1 milligram per cubic meter of air.
Phosphoric acid, 1 milligram per cubic meter of air.
Phosphorus pentachloride, 1 milligram per cubic meter of air.
Phosphorus pentasulfide, 1 milligram per cubic meter of air.
Phosphorus trichloride (gas), 3 milligrams per cubic meter of air or 0.5 part per million parts of air by volume.

Potential Occupational Exposures

Phosphorus (white or yellow)

Bronze alloy makers
Electroluminescent coating makers
Fertilizer makers
Fireworks makers
Incendiary makers
Metallic phosphide makers
Metal refiners
Munitions workers
Pesticide workers
Phosphoric acid makers
Phosphoric anhydride makers
Phosphorus workers
Rat poison workers
Red phosphorus makers
Semiconductor makers
Smoke bomb makers

Phosphoric acid

Activated carbon makers
Animal feed makers
Ceramic makers
Dental cement makers
Detergent makers
Drug makers
Electropolishers
Engravers
Fertilizer makers
Flavoring syrup makers
Foundry workers
Gelatin makers
Lithographers
Metal cleaners
Phosphate makers
Phosphoric acid workers
Photoengravers
Polish makers
Rubber latex makers
Rust inhibitor makers

Rustproofers
Soft drink makers
Sugar refiners
Water treaters
Wax makers
Yeast makers

Phosphorus trichloride

Agricultural chemical makers
Chlorinated compound makers
Dye makers
Gasoline additive makers
Phosphorus oxychloride makers
Phosphorus trichloride workers
Plasticizer makers
Saccharin makers
Surfactant makers

Phosphorus pentachloride

Acetylcellulose makers
Agricultural chemical makers
Chlorinated compound makers
Organic chemical synthesizers
Phosphorus oxychloride makers
Phosphorus pentachloride workers

Phosphorus pentasulfide

Agricultural chemical makers
Flotation agent makers
Insecticide makers
Lubricating oil additive makers
Match makers
Organic chemical synthesizers
Phosphorus pentasulfide workers
Rubber chemical makers

Phosphorus oxychloride

Agricultural chemical makers
Chlorinated compound makers
Drug makers
Gasoline additive makers
Hydraulic fluid makers
Organic chemical synthesizers
Organic phosphate makers
Phosphorus oxychloride workers
Plasticizer makers

References

CALEY, J. P. AND KELLOCK, I. A.: Acute yellow phosphorus poisoning with recovery. *Lancet* 1: 539, 1955.

HEIMANN, H.: Chronic phosphorus poisoning. *J. Indust. Hyg. and Toxicol.* 28: 142, 1946.

PATTY, F. A. (EDITOR): *Industrial Hygiene and Toxicology.* 2nd ed. Vol. 1. Interscience Publishers, New York, 1958.

RUBITSKY, H. J. AND MYERSON, R. M.: Acute phosphorus poisoning. *Arch. Int. Med.* 83: 164, 1949.

(151) Phthalic Anhydride

phthalic acid anhydride

Harmful Effects

Local Phthalic anydride in pure state is not an irritant, but after contact with water, the caustic phthalic acid is formed. Local irritation may produce conjunctivitis and contact dermatitis. Contact dermatitis may also

be caused by allergic hypersensitivity. Irritation of upper respiratory tract is manifested by epistaxis, atrophy of the nasal mucous membranes, loss of sense of smell, and hoarseness.

Route of Entry Inhalation of fume or dust.

Systemic Bronchitis, emphysema, asthma, and urticaria may occur.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Alcohol denaturant makers
Alizarin dye makers
Alkyd resin makers
Anthranilic acid makers
Anthraquinone makers
Automobile finish makers
Benzoic acid makers
Cellulose acetate plasticizer makers
Dacron fiber makers
Diethylphthalate makers
Dimethylphthalate makers
Drug makers
Dye makers
Enamel makers
Eosin makers
Erythrosin makers
Herbicide makers
Indigo makers
Insecticide makers
Isophthalic acid makers
Methyl aniline purifiers
Mylar plastic makers
Organic chemical synthesizers
Phenolphthalein makers
Photographic film makers
Phthalamide makers
Phthalate ester makers
Phthalein makers
Phthalic anhydride workers
Plasticizer makers
Repellent makers
Resin makers
Sulfathalidine makers
Terephthalic acid makers
Vat dye makers
Vinyl plasticizer makers

Reference

MERLEVEDE, E. AND ELSKENS, J.: The toxicity of phthalic anhydride, maleic anhydride and the phthalates. *Arch. belges med. sociale, hyg., med. travail et med. legale* 15: 445, 1957. (Abst., *Bull. Hyg.* 33: 1151, 1958)

(152) Picric Acid

picronitric acid, trinitrophenol, nitroxanthic acid, carbazotic acid, phenol trinitrate

Harmful Effects

Local Contact dermatitis from either primary irritation or allergic hypersensitivity, yellow coloring of skin and hair, conjunctivitis, keratitis, and yellow vision.

Routes of Entry Percutaneous absorption from dust, inhalation of dust, and ingestion.

Systemic Headache, vertigo, convulsions, gastroenteritis, sometimes hepatitis and hemorrhagic nephritis.

Special Diagnostic Test

Analysis of urine for picric acid. *See* Von Oettingen, 1958.

Recommended Threshold Limit

0.1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Battery makers
Copper etchers
Disinfectant makers
Drug makers
Dye makers
Dyers
Explosive makers
Fireworks makers
Forensic chemists
Glass makers, colored
Histology technicians
Match makers
Picrate makers
Picric acid workers
Shell fillers
Tannery workers
Textile dyers
Textile printers

References

VON OETTINGEN, W. F.: The aromatic amino and nitro compounds, their toxicity and potential dangers. A review of the literature. Pub. Health Bull. No. 271. U.S. Government Printing Office, Washington, D.C., 1941.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(153) Platinum and Compounds

Harmful Effects

Local Metallic platinum is nontoxic. Platinum salts may act as skin irritants and skin sensitizers to produce contact dermatitis; irritation of nose and throat has been reported to follow exposure to these salts.

Route of Entry Inhalation of dust or mist.

Systemic Sodium chloroplatinate (platinic sodium chloride) has been implicated as the etiologic agent responsible for syndrome called *platinosis.* This condition consists of mild chemical irritation and chronic inflammation of entire respiratory tract associated, in some cases, with allergic manifestations affecting skin. In more severe cases, symptoms of typical bronchial asthma may become evident.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Soluble salts) 0.002 milligram per cubic meter of air.

Potential Occupational Exposures

Alloy makers
Ceramic workers
Dental alloy makers
Drug makers
Electronic equipment makers
Electroplaters
Glass makers
Ink makers, indelible
Jewelry makers
Laboratory ware makers
Laboratory workers, chemical
Microscopists
Mirror makers
Platinum workers
Spark plug makers
Zinc etchers

Reference

ROBERTS, A. E.: Platinosis. A five-year study of the effects on employees in a platinum laboratory and refinery. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 549, 1951.

(154) Propyl Acetate

Harmful Effects

Local May be irritating to skin and mucous membranes of upper respiratory tract.

Route of Entry Inhalation of vapor.

Systemic No industrial poisonings have been reported. In high concentrations vapors may depress central nervous system.

Special Diagnostic Test

None.

Recommended Threshold Limit

200 parts per million parts of air by volume or 840 milligrams per cubic meter of air.

Potential Occupational Exposures

Flavoring makers
Lacquer makers
Nitrocellulose workers
Organic chemical synthesizers
Perfume makers
Propyl acetate workers
Resin makers
Varnish makers

(155) n-Propyl Alcohol

1-propanol, propylic alcohol

Harmful Effects

Local Vapors are mildly irritating to conjunctiva and mucous membranes of upper respiratory tract.

Route of Entry Inhalation of vapor.

Systemic No cases of industrial poisoning have been recorded. Vapor can produce mild central nervous system depression.

Special Diagnostic Test

None.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Brake fluid makers
Cosmetic makers
Disinfectant makers
Dope processors
Drug makers
Ink makers, printing
Lacquer makers
Metal degreasers
Nitrocellulose workers
Organic chemical synthesizers
Polish makers
Propionaldehyde makers
Propionic acid makers
n-Propyl acetate makers
n-Propyl alcohol workers
n-Propylated urea makers
Resin makers
Soap makers
Solvent makers
Vegetable oil processors
Wax makers
Window cleaning fluid makers

Reference

HENSON, E. V.: The toxicology of some aliphatic alcohols; part 1. *J. Occup. Med.* 2: 442, 1960.

(156) Propylene Dichloride

1,2-dichloropropane, propylene chloride

Harmful Effects

Local Repeated or prolonged contact with liquid can produce a dry, scaly, fissured dermatitis. May be irritating to eyes and other mucous membranes.

Route of Entry Inhalation of vapor.

Systemic Produces marked narcosis. May cause fatty degeneration of liver, kidneys and heart.

Special Diagnostic Test

None.

Recommended Threshold Limit

75 parts per million parts of air by volume or 350 milligrams per cubic meter of air.

Potential Occupational Exposures

Cellulose plastic makers
Dry cleaners
Dry cleaning fluid makers
Fat processors
Fumigant workers
Gum processors
Metal degreasers
Oil processors
Organic chemical synthesizers
Propylene dichloride workers
Rubber makers
Scouring compound makers
Solvent workers
Stain removers
Wax makers

Reference

HEPPEL, L. A.; NEAL, P. A.; HIGHMAN, B., AND PORTERFIELD, V. T.: Toxicology of 1,2-dichloropropane (propylene dichloride). 1, Studies on effects of daily inhalations. *J. Indust. Hyg. & Toxicol.* 28: 1, 1946.

(157) Pyrethrum. *See* Pesticides Section

(158) Pyridine

Harmful Effects

Local Liquid and vapor are irritating to eyes, nose, and throat. Repeated or prolonged contact with liquid can produce a dry, scaly, fissured dermatitis. Photosensitization dermatitis can occur.

Routes of Entry Inhalation of vapor and percutaneous absorption of liquid.

Systemic Acute exposure produces flushing of the face and narcotic effects of nausea, vomiting, dizziness, and drowsiness. Effects of chronic exposure include headache, nervousness, insomnia, and other neurologic disturbances, but these have also been reported following acute and subacute exposures.

Special Diagnostic Test

None.

Recommended Threshold Limit

5 parts per million parts of air by volume or 15 milligrams per cubic meter of air.

Potential Occupational Exposures

Acrylonitrile makers
Adhesive workers
Alcohol denaturant makers
Anhydrous salt processors
Denatured alcohol makers
Drug makers
Dyers
Explosive workers
Furniture polishers
Gas mantle makers
Gilders
Lacquerers

Lacquer makers
Latex workers
Niacin makers
Organic chemical synthesizers
Paint makers
Pencil makers
Pyridine workers
Rubber chemical makers
Rubber makers
Rust inhibitor workers
Solvent workers
Styrene makers
Sulfapyridine makers
Textile dyers
Vitamin makers
Waterproofing makers

Reference

BALDI, G.: Occupational pathology from pyridine. *Med. Lavoro* 44: 244, 1953.

(159) Quinone

benzoquinone, chinone

Harmful Effects

Local Quinone in solid form, solutions, or vapor phase can produce contact dermatitis from primary irritation. Condensation of quinone vapors on eyes produces conjunctivitis, lacrimation, photophobia, corneal stains, ulcerations, and opacities.

Route of Entry Inhalation of vapor.

Systemic None has been reported from inhalation or ingestion in humans. In animal studies, ingestion or subcutaneous injection of quinone has produced convulsions, respiratory difficulties, hypotension, and asphyxia.

Special Diagnostic Test

Analysis of urine for hydroquinone. *See* Von Oettingen, 1958.

Recommended Threshold Limit

0.1 part per million parts of air by volume or 0.4 milligram per cubic meter of air.

Potential Occupational Exposures

Dye makers
Gelatin makers
Hydrogen peroxide makers
Hydroquinone makers
Laboratory workers, chemical
Photographic film developers
Protein fiber makers
Quinone workers
Tanners

References

STERNER, J. H.; OGLESBY, F. L., AND ANDERSON, B.: Quinone vapors and their harmful effects. 1, Corneal and conjunctival injury. *J. Indust. Hyg. & Toxicol.* 29: 60, 1947.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(160) Radon. *See* Uranium and Compounds

(161) Selenium and Compounds

Harmful Effects

Local Compounds of selenium are potent skin and mucous membrane irritants. Selenium, selenium oxide, selenic acid, selenic acid anhydride, selenium sulfide, potassium selenite, sodium selenite, selenium bromide, hydrogen selenide, methyl selenide, selenious acid, and selenium oxychloride produce degrees of dermatitis varying from erythema to severe burns with vesiculation. These compounds may produce nose, throat, and eye irritation with sneezing, nasal congestion, anosmia, coughing, perspiration, lacrimation, palpebral edema, and conjunctivitis.

Route of Entry Inhalation of dust or vapor; percutaneous absorption of liquid.

Systemic Acute systemic poisoning in industry is rare since these compounds are sufficiently irritant to compel the workers to leave an area of high exposure. Nervousness, dizziness, weakness, nausea, vomiting, diarrhea, abdominal pain, somnolence, dyspnea, garlic-like odor of the breath, convulsions, and death may result from severe exposure. Pulmonary edema may be produced by the more volatile selenium compounds, such as selenium oxide and hydrogen selenide. Percutaneous absorption of a selenium sulfide compound used in certain shampoo mixtures has been reported to produce loss of hair in a few persons and, in one case, symptoms compatible with systemic toxicity.

Chronic exposure is characterized by nausea, vomiting, possible liver damage, nervousness, tremor, metallic taste, garlic-like odor of the breath, pallor, dizziness, and fatigue.

Special Diagnostic Test

Analysis of urine for selenium. *See* Elkins, 1959.

Recommended Threshold Limits

Hydrogen selenide, 0.05 part per million parts of air by volume or 0.2 milligram per cubic meter of air.

Selenium compounds (as Se), 0.1 milligram per cubic meter of air.

Potential Occupational Exposures

Arc light electrode makers
Bacteriologists
Barbers
Brass founders
Cement workers
Ceramic makers
Copper smelters
Drug makers
Electric rectifier makers
Electroplaters
Glass makers
Hairdressers

Ink makers
Lead smelters
Leather workers
Lime workers
Lubricating oil makers
Microscopists
Organic chemical synthesizers
Paint makers
Paper makers
Pesticide makers
Phosphor makers
Photoelectric cell makers
Photographic chemical makers
Pigment makers
Plastic workers
Pyrite roasters
Rubber makers
Seed germination testers
Selenium workers
Semiconductor makers
Shampoo makers
Stainless steel makers
Stenching agent makers
Sulfuric acid makers
Textile workers
Xerographic plate makers
Zinc smelters

References

BUCHAN, R. F.: Industrial selenosis. *Occup. Med.* 3: 439, 1947.

CERWENKA, E. A., JR. AND COOPER, W. CHARLES: Toxicology of selenium and tellurium, and their compounds. *Arch. Environ. Health* 3: 189, 1961.

CLINTON, M., JR.: Selenium fume exposure. *J. Indust. Hyg. & Toxicol.* 29. 225, 1947.

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

GLOVER, J. R.: Some medical problems concerning selenium in industry. *Trans. Assoc. Indust. Med. Officers* 4: 94, 1954.

GROVER, R. W.: Diffuse hair loss associated with selenium (Selsun) sulfide shampoo. *J. Am. Med. Assoc.* 160: 1397, 1956.

RANSONE, J. W.; SCOTT, N. M., JR., AND KNOBLOCK, E. C.: Selenium sulfide intoxication. *New Eng. J. Med.* 264: 384, 1961.

(162) Silver and Compounds

Harmful Effects

Local Localized industrial argyria (argyrism) is caused by implantation of silver particles in skin and is manifested as small bluish-black spots, usually on hands and forearms. Silver nitrate is irritating to skin and mucous membranes and can temporarily discolor skin.

Routes of Entry Inhalation of dust; ingestion of solutions or dust.

Systemic Industrial argyria from ingestion of silver compounds has been reported, but is no longer seen. It resembled the bluish-gray discoloration of eyes and skin seen in generalized argyria from therapeutic ingestion or injection of silver salts. Depth of color in argyria is greater in those areas exposed to light.

When silver or its salts are inhaled in industrial exposures, much of the silver is deposited in elastic tissue of lungs (pulmonary argyria), but eventually the bluish-gray discoloration appears in eyes and skin. Bron-

chitis and emphysema have been described in workers with pulmonary argyria, but a cause and effect relationship has not been demonstrated. Except for its cosmetic disfigurement, argyria is generally considered to be benign.

Special Diagnostic Tests

Analysis of blood for excessive amounts of silver is helpful only during exposure. Examination of skin with ultraviolet lamp and of cornea with slit lamp. *See* Blumberg and Carey, 1934; Harker and Hunter, 1935, and Holden, 1950.

Recommended Threshold Limit

(Tentative) 0.05 milligram per cubic meter of air.

Potential Occupational Exposures

Algicide makers
Alloy makers
Artificial rain makers
Bactericide makers
Battery makers
Bearing metal makers
Brazing rod makers
Ceramic makers
Chemical equipment makers
Copper refiners
Cutlery makers
Dental alloy makers
Drug makers
Electric conductor makers
Electric equipment makers
Electronic workers
Electrotype makers
Food product equipment makers
Gas mask makers
Glass makers
Glass polish makers
Gold refiners
Hair dye makers
Ink makers, indelible
Ivory etchers
Jewelry makers
Lead refiners
Metal inlayers
Mirror makers
Optical workers
Organic chemical synthesizers
Paint makers
Photographic chemical makers
Photographic film makers
Silver bromide makers
Silver engravers
Silver finishers
Silver nitrate makers
Silver platers
Silver polishers
Silver reclaimers
Silversmiths
Silver workers
Solder workers, hard
Water treaters

References

BLUMBERG, H. AND CAREY, T. N.: Argyremia; detection of unsuspected and obscure argyria by the spectrographic demonstration of high blood silver. *J. Am. Med. Assoc.* 103: 1521, 1934.

BROWNING, E.: *Toxicity of Industrial Metals.* Butterworths, London, 1961.

HARKER, J. M. AND HUNTER, D.: Occupational argyria. *Brit. J. Dermat.* 47: 441, 1935. Examination with slit lamp.

HEIMANN, H.: Toxicity of metallic silver. *Indust. Bull.* (N.Y. State Dept. Labor) 22: 81, 1943.

HILL, W. R. AND PILLSBURY, D. M.: *Argyria.* Williams & Wilkins Co., Baltimore, 1939.

HOLDEN, R. F., JR.: Observations in argyria. *J. Lab. & Clin. Med.* 36: 837, 1950.

MONTANDON, M. A.: Argyrose des voies respiratoires. *Arch. Mal. Prof.* 20: 419, 1959.

(163) Sodium and Potassium Hydroxides

Aqueous solution of sodium hydroxide (caustic soda or caustic alkali) or potassium hydroxide (caustic potash or caustic alkali) is known as lye; the sodium hydroxide solution is also referred to as soda lye. Sodium hydroxide added to calcium oxide produces soda lime (*See* Calcium Oxide). Water added to calcium oxide (lime or quicklime) produces calcium hydroxide or slaked lime. Washing soda (soda ash or sal soda) is sodium carbonate combined with 10 molecules of water. Baking soda is sodium bicarbonate. Chloride of lime (which see) is a mixture of calcium chloride, calcium hypochlorite and calcium hydroxide.

Harmful Effects

Local Both compounds exert an extremely corrosive action on skin, eyes and mucous membranes.

Route of Entry Inhalation of dust or mist.

Systemic Systemic effects are due entirely to local tissue injury. Extreme pulmonary irritation may result from inhalation of dust or mist.

Special Diagnostic Test

None.

Recommended Threshold Limit

Sodium hydroxide, 2 milligrams per cubic meter of air.

Potassium hydroxide, not established.

Potential Occupational Exposures

Bleachers
Bleach makers
Bronzers
Degreasers
Detergent makers
Electroplaters
Enamelers
Engravers
Etchers
Furniture polishers
Housekeepers
Laboratory workers, chemical
Laundry workers
Lithographers
Match makers
Mercerizers
Oxalic acid makers
Paint removers
Paper makers
Perfume makers
Petroleum refinery workers
Photoengravers
Potassium hydroxide workers
Printers
Printing ink makers
Pulp makers

Rayon makers
Rubber reclaimers
Soap makers
Sodium hydroxide workers
Textile bleachers
Varnish removers
Vegetable oil refiners

(164) Styrene

cinnamene, cinnamenol, cinnamol, phenethylene, phenylethylene, styrene monomer, styrol, styrolene, vinylbenzene

Harmful Effects

Local Styrene and its vapor are irritating to eyes, nose, and throat. Styrene liquid is a low-grade cutaneous irritant, and repeated contact with skin will produce a dry, scaly, and fissured dermatitis.

Route of Entry Inhalation of vapor.

Systemic Stryrene sickness with symptoms of headache, fatigue, nausea, vomiting, anorexia, dizziness, and drowsiness has occurred from narcotic effect of vapor. Respiratory tract irritation by high vapor concentrations is manifested by cough. No chronic systemic effects have been reported.

Special Diagnostic Test

Determination of urinary hippuric acid excretion has been suggested; however, it is not specific. *See* Gerarde, 1960.

Recommended Threshold Limit

100 parts per million parts of air by volume or 420 milligrams per cubic meter of air.

Potential Occupational Exposures

Adhesive makers
Boat makers
Emulsifier agent makers
Insulator makers
Organic chemical synthesizers
Petroleum refinery workers
Plastic luggage makers
Polyester resin laminators
Polystyrene makers
Potting compound workers
Protective coating makers
Rubber makers
Sports car body makers
Styrene workers
Swimming pool makers
Varnish makers

References

CARPENTER, C. P.; SHAFFER, C. B.; WEIL, C. S., AND SMYTH, H. F., JR.: Studies on the inhalation of 1,3-butadiene; with a comparison of its narcotic effect with benzol, toluol, and styrene, and a note on the elimination of styrene by the human. *J. Indust. Hyg. & Toxicol.* 26: 69, 1944.

GERARDE, H. W.: *Toxicology and Biochemistry of Aromatic Hydrocarbons.* Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1960.

ROGERS, J. C. AND HOOPER, C. C.: An industrial hygiene problem in the plastics field. *Modern Sanit.* 8: 19, (Aug.) 1956.

WILSON, R. H.: Health hazards encountered in the manufacture of synthetic rubber. *J. Am. Med. Assoc.* 124: 701, 1944.

(165) Sulfur Dioxide

sulfurous anhydride, sulfurous oxide

Harmful Effects

Local Gaseous sulfur dioxide is irritant to conjunctiva and mucous membranes of upper respiratory tract. High exposure may produce laryngeal edema and death from asphyxiation. Liquid sulfur dioxide is skin irritant. Corneal injury with blindness has resulted from liquid splashes into eyes.

Route of Entry Inhalation of gas.

Systemic Severe acute symptoms are unusual since gas is sufficiently irritant to compel the workers to flee. Inhalation of high concentrations may produce bronchitis, pneumonitis, pulmonary edema, and death.

Studies of chronic sulfur dioxide exposure in humans have indicated no appreciable danger to health. Nasopharyngitis, fatigue, altered sense of taste and smell, and dyspnea on exertion have been said to result from long continued low exposures.

Special Diagnostic Test

None.

Recommended Threshold Limit

5 parts per million parts of air by volume or 13 milligrams per cubic meter of air.

Potential Occupational Exposures

Beet sugar bleachers
Boiler water treaters
Brewery workers
Diesel engine operators
Diesel engine repairmen
Disinfectant makers
Disinfectors
Firemen
Flour bleachers
Food bleachers
Foundry workers
Fruit bleachers
Fumigant makers
Fumigators
Furnace operators
Gelatin bleachers
Glass makers
Glue bleachers
Grain bleachers
Ice makers
Meat preservers
Oil bleachers
Oil processors
Ore smelter workers
Organic sulfonate makers
Paper makers
Petroleum refinery workers
Preservative makers
Protein makers, food
Protein makers, industrial
Refrigeration workers
Straw bleachers

Sugar refiners
Sulfite makers
Sulfur dioxide workers
Sulfuric acid makers
Sulfuryl chloride makers
Tannery workers
Textile bleachers
Thermometer makers, vapor pressure
Thionyl chloride makers
Wicker ware bleachers
Wine makers
Wood pulp bleachers
Wool bleachers

References

ANDERSON, A.: Possible long term effects of exposure to sulfur dioxide. *Brit. J. Indust. Med.* 7: 82, 1950.

KEHOE, R. A.; MACHLE, W. F.; KITZMILLER, K., AND LEBLANC, T. J.: On the effects of prolonged exposure to sulfur dioxide. *J. Indust. Hyg.* 14: 159, 1932.

(166) Sulfuric Acid

oil of vitriol, spirit of vitriol, hydrogen sulfate

Harmful Effects

Local Sulfuric acid is irritant to conjunctiva and mucous membranes of the upper respiratory tract. The acid may also produce erosion of teeth, usually the incisors. Liquid may produce severe burns and ulceration of skin.

Route of Entry Inhalation of vapor.

Systemic Systemic effects are not well recognized. Human experimental studies have revealed that rapid shallow respiration may occur following exposure to low concentrations of sulfuric acid mist below the taste-odor-irritation threshold. Pulmonary fibrosis, bronchiectasis, and emphysema have been reported from acute exposure to fuming sulfuric acid and sulfuric acid mist.

Special Diagnostic Test

None.

Recommended Threshold Limit

1 milligram per cubic meter of air.

Potential Occupational Exposures

Aluminum sulfate makers
Ammonium sulfate makers
Battery makers, storage
Cellulose workers
Copper sulfate makers
Detergent makers
Drug makers
Dye makers
Electroplaters
Explosive makers
Fertilizer makers
Food processors
Fur processors
Galvanizers
Glue makers
Jewelers
Laboratory workers, chemical
Metal cleaners

218-695 O-66-15

Paint makers
Paper makers
Petrochemical workers
Petroleum refinery workers
Phenol makers
Phosphate workers
Phosphoric acid makers
Pigment makers
Rayon workers
Rubber workers
Steel workers
Sulfuric acid workers
Textile workers

References

AMDUR, M. O.; SILVERMAN, L., AND DRINKER, P.: Inhalation of sulfuric acid mist by human subjects. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 305, 1952.

GOLDMAN, A. AND HILL, W. T.: Chronic bronchopulmonary disease due to inhalation of sulfuric acid fumes. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 205, 1953.

MALCOLM, D. AND PAUL, E.: Erosion of the teeth due to sulfuric acid in the battery industry. *Brit. J. Indust. Med.* 18: 63, 1961.

(167) Sulfur Monochloride

sulfur chloride, sulfur subchloride

Harmful Effects

Local Sulfur monochloride liquid may cause severe skin burns and its vapors are very irritating to eyes and mucous membranes of nose, throat, and trachea.

Route of Entry Inhalation of vapor.

Systemic Although this compound is almost certainly capable of producing severe lung irritation, very few serious cases of industrial exposure have been reported. This is probably because the pronounced irritant effects of sulfur monochloride serve as an immediate warning signal when concentration of the gas approaches a hazardous level.

Special Diagnostic Test

None.

Recommended Threshold Limit

1 part per million parts of air by volume or 6 milligrams per cubic meter of air.

Potential Occupational Exposures

Carbon tetrachloride makers
Drug makers
Dyers
Gold extractors
Insecticide makers
Organic chemical synthesizers
Rubber cement makers
Rubber makers
Rubber substitute makers
Sugar juice purifiers
Sulfur dye makers
Sulfur monochloride workers
Textile dyers
Textile finishers
Vegetable oil processors
Vulcanized oil makers
Vulcanizers
Wood hardeners

(168) Tellurium

aurum paradoxum, metallum problematum

Harmful Effects

Local No local effects have been reported.

Routes of Entry Inhalation of dust or fume; percutaneous absorption from dust.

Systemic Exposure to dust or fume may produce a persistent garlic-like odor of breath (due to methyl telluride), suppression of perspiration, metallic taste, nausea, anorexia, and somnolence.

Special Diagnostic Test

Analysis of tellurium in urine and feces. *See* Steinberg et al., 1942.

Recommended Threshold Limit

0.1 milligram per cubic meter of air.

Potential Occupational Exposures

Alloy makers
Ceramic makers
Copper alloy makers
Copper refinery workers
Electronic workers
Enamel makers
Foundry workers
Glass makers
Iron makers
Lead refinery workers
Porcelain makers
Rubber makers
Semiconductor makers
Silverware makers
Stainless steel makers
Tellurium lead alloy makers
Tellurium workers
Thermoelectric device makers
Vulcanizers

References

CERWENKA, E. A., JR., AND COOPER, W. CHARLES: Toxicology of selenium and tellurium, and their compounds. *Arch. Environ. Health* 3: 189, 1961.

GLASS, P. K.: Toxicity of tellurium. United States Atomic Energy Commission AECU-374. The Commission, Washington, D.C., 1948.

QUERIES AND MINOR NOTES: Effect of tellurium on health. *J. Am. Med. Assoc.* 155: 1548, 1954.

STEINBERG, H. H.; MASSARI, S. C.; MINER, A. C., AND RINK, R.: Industrial exposure to tellurium; atmospheric studies and clinical evaluation. *J. Indust. Hyg. & Toxical.* 24: 183, 1942.

(169) Tetrachloroethane

sym.-tetrachloroethane, acetylene tetrachloride

Harmful Effects

Local Repeated or prolonged contact with this low grade primary irritant can produce a scaly and fissured dermatitis.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.
Systemic Most toxic of all of chlorinated hydrocarbons. Early effects are caused by its narcotic action. Later, liver damage may be severe resulting in acute yellow atrophy of this organ. Fatty degeneration of kidneys and myocardium may be produced.

Special Diagnostic Test

None.

Recommended Threshold Limit

5 parts per million parts of air by volume or 35 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Biologists
Cellulose acetate workers
Denatured alcohol workers
Ethyl chloride makers
Ethylene dichloride makers
Fat processors
Fumigant makers
Fumigators
Gasket makers
Herbicide workers
Insecticide workers
Lacquer workers
Metal cleaners
Metal degreasers
Mineralogists
Oil processors
Organic chemical synthesizers
Paint makers
Paint remover workers
Phosphorus processors
Photographic film makers
Resin makers
Rubber makers
Rust remover workers
Soil treaters
Solvent workers
Sulfur processors
Tetrachloroethane workers
Trichloroethylene makers
Varnish workers
Waxers
Wax makers

Reference

VON OETTINGEN, W. F.: The halogenated aliphatic, olefinic, cyclic, aromatic, and aliphatic-aromatic hydrocarbons including the halogenated insecticides; their toxicity and potental dangers. Pub. Health Service Pub. No. 414. U.S. Government Printing Office, Washington, D.C., 1955.

(170) Tetraethyl Lead

TEL

Harmful Effects

Local Liquid may penetrate the skin without producing appreciable local injury.
Routes of Entry Percutaneous absorption of liquid, inhalation of vapor.
Systemic Signs and symptoms resulting from tetraethyl lead intoxication differ from those of inorganic lead poisoning. Central nervous system

effects predominate. Symptoms are usually delayed for a few hours to several days following exposure. Insomnia, headaches, nightmares; nervousness, irritability, and vague gastrointestinal symptoms may appear early. If the illness is severe, patients frequently experience episodes of maniacal behavior. Fatigue, weakness, weight loss, muscular pains, tremors, slow pulse, and low blood pressure also characterize the acute illness. Prognosis will depend upon severity of exposure. Many deaths have been recorded in literature. Those patients who recover generally exhibit no sequelae.

Special Diagnostic Tests

Analysis of blood and urine for lead. *See* Fleming et al., 1960.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Storage tank cleaners
Tetraethyl lead blenders
Tetraethyl lead makers
Tetraethyl lead mixers
Tetraethyl lead workers

References

ADVISORY COMMITTEE ON TETRAETHYL LEAD TO SURGEON GENERAL OF PUBLIC HEALTH SERVICE: Public health aspects of increasing tetraethyl lead content in motor fuel. Pub. Health Service Pub. No. 712. U. S. Government Printing Office, Washington, D.C., 1959.

CREMER, J. E. AND CALLAWAY, S.: Further studies on the toxicity of some tetra and trialkyl lead compounds. *Brit. J. Indust. Med.* 18: 277, 1961.

FLEMING, A. J.; D'ALONZO, C. A., AND ZAPP, J. A. (EDITORS): *Modern Occupational Medicine.* 2nd ed. Lea & Febiger, Philadelphia, 1960.

KEHOE, R. A.: Tetraethyl lead, the disposition of an international chemical hazard. In *Proceedings Thirteenth International Congress on Occupational Health,* New York, July 25–29, 1960. U.S. Executive Committee of the Congress (L. Wade, M.D., chairman), New York, 1961.

VARIOUS AUTHORS: *Lead Symposium,* February 25–27, 1963. University of Cincinnati, Cincinnati, Ohio, 1963.

(171) Tetramethyl Lead

TML

Harmful Effects

Local Same as tetraethyl lead, which see.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid. TML is more volatile than TEL and therefore may present more of an inhalation hazard.

Systemic Similar to tetraethyl lead as indicated by animal experimentation, but is less readily absorbed through intact skin.

Special Diagnostic Tests

Same as tetraethyl lead.

Recommended Threshold Limit

Not established.

Potential Occupational Exposures

Storage tank cleaners
Tetramethyl lead blenders
Tetramethyl lead makers
Tetramethyl lead mixers
Tetramethyl lead workers

References

CREMER, J. E. AND CALLAWAY, S.: Further studies on the toxicity of some tetra and trialkyl lead compounds. *Brit. J. Indust. Med.* 18: 277, 1961.

DETREVILLE, R. T. P.; WHEELER, H. W., AND STERLING, T.: Occupational exposure to organic lead compounds. The relative degree of hazard in occupational exposure to air-borne tetraethyl lead and tetramethyl lead. *Arch. Environ. Health* 5: 532, 1962.

KEHOE, R. A.; CHOLAK, J.; SPENCE, J. A., AND HANCOCK, W.: Potential hazard of exposure to lead. 1, Handling and use of gasoline containing tetramethyl lead. *Arch. Environ. Health* 6: 239, 1963.

KEHOE, R. A.; CHOLAK, J.; MCILHINNEY, J. G.; LOFQUIST, G. A., AND STERLING, T. D.: Potential hazard of exposure to lead. 2, Further investigations in the preparation, handling, and use of gasoline containing tetramethyl lead. *Arch. Environ. Health* 6: 255, 1963.

VARIOUS AUTHORS: *Lead Symposium*, February 25–27, 1963. University of Cincinnati, Cincinnati, Ohio, 1963.

(172) Tetramethylthiuram Disulfide

thiram, bis-(dimethylthiocarbamyl) disulfide, thirad, thiuram, TMTD

Harmful Effects

Local Irritation of skin and mucous membranes of eyes and upper respiratory tract. Allergic contact dermatitis.

Route of Entry Inhalation of dust.

Systemic No systemic poisoning has been reported in United States. On basis of animal experiments and reports in foreign literature, bronchitis, liver, and kidney damage may be expected from exposure to high concentrations. Intolerance to alcohol has been observed, manifested by flushing of face, palpitation, rapid pulse, dizziness, and hypotension.

Special Diagnostic Test

None.

Recommended Threshold Limit

5 milligrams per cubic meter of air.

Potential Occupational Exposures

Bacteriostat makers, soap
Dock workers
Fungicide workers
Insecticide workers
Japanese beetle repellent makers
Lubricating oil blenders
Rat repellent makers
Rubber makers, heat resistant
Seed disinfectors
Soap makers
Tetramethylthiuram disulfide workers
Vulcanizers

References

FINULLI, M. AND MAGISTRETTI, M.: Antabuse-like intoxication in workmen employed in the manufacture of the synthetic agricultural chemical TMTD (tetramethylthiouram disulfide). *Med. Lavoro* 52: 132, 1961.

LAVARINO, A. AND MASOERO, A.: Acute poisoning by tetramethylthiuram disulfide. *Rass. med. ind.* 24: 458, 1955. (*Indust. Hyg. Digest*, Abst. No. 883, July 1956)

SCHULZ, K. H. AND HERRMANN, W. P.: Tetramethylthiuram disulfide, a thiourea derivative, as an agent provocative of dermatitis in dock laborers. *Berufsdermatosen* 6: 130, 1958. (*Indust. Hyg. Digest*, Abst. No. 162, February 1958)

(173) Tetryl

trinitrophenylmethylnitramine, nitramine, tetranitromethylaniline, pyrenite, picrylmethylnitramine, picrylnitromethylamine

Harmful Effects

Local Tetryl is a potent sensitizer, and allergic contact dermatitis is common. Contact may stain skin and hair yellow or orange; workers with such stains have been referred to as canaries. Tetryl dust is sometimes irritating to eyes and nose, causing conjunctivitis, sneezing, and epistaxis.

Route of Entry Inhalation of dust.

Systemic Cough is a common sympton among workers initially exposed to large amounts of dust, but chest roentgenograms reveal no pulmonary disease. Systemic intoxication is practically never encountered. In the few cases of liver damage that have been reported, exposure was massive. Tetryl workers are frequently exposed to trinitrotoluene and other explosives, making it difficult to establish the specific agent producing the systemic symptoms.

Special Diagnostic Test

Webster's reagent, a dilute solution of sodium hydroxide in ethyl alcohol, is discolored dark brown by tetryl on skin. *See* Norwood, 1943.

Recommended Threshold Limit

1.5 milligrams per cubic meter of air.

Potential Occupational Exposures

Ammunition makers
Detonator makers
Explosive workers
Indicator makers, chemical
Tetryl workers

References

BERGMAN, B. B.: Tetryl toxicity; a summary of ten years' experience. *A.M.A. Arch Indust. Hyg. & Occup. Med.* 5: 10, 1952.

HARDY, H. L. AND MALOOF, C. C.: Evidence of systemic effect of tetryl; with summary of available literature. *Arch. Indust. Hyg. & Occup. Med.* 1: 454, 1950.

NORWOOD, W. D.: Trinitrotoluene (TNT); its effective removal from the skin by a special liquid soap. *Indust. Med.* 12: 206, 1943.

(174) Thallium and Compounds

Harmful Effects

Local Some thallium salts may produce skin irritation.

Routes of Entry Inhalation of dust and fume. Ingestion and percutaneous absorption of dust.

Systemic Thallium may act as a *cumulative poison;* that is, repeated small doses which would individually produce little or no effect may be stored in body until a harmful or even lethal dose accumulates. Acute effects include severe gastroenteritis, abdominal pain, and collapse. Subacute or chronic effects include nausea, vomiting, leg and abdominal cramping, paresthesia of lower limbs, irritability, anorexia, stomatitis, dry scaly skin, metallic taste, garlic-like foul breath, visual disturbances, convulsions, delayed loss of hair, and kidney damage.

Special Diagnostic Test

Analysis of urine or tissues for thallium. *See* Winn et al., 1952, and Jacobs, 1962.

Recommended Threshold Limit

(Soluble compounds) 0.1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Alloy makers
Artificial diamond makers
Carbon disulfide testers
Chlorinated compound makers
Depilatory makers
Drug makers
Dye makers
Fireworks makers
Flotation workers
Gem makers
Glass makers, high refractive index
Incandescent lamp makers
Indicator makers, chemical
Infrared instrument makers
Insecticide workers

Match makers
Optical glass makers
Ore upgraders
Organic chemical synthesizers
Ozone testers
Photoelectric cell makers
Rodenticide workers
Textile workers
Thallium workers

References

JACOBS, M. B.: The determination of thallium in urine. *Am. Indust. Hyg. Assoc. J.* 23: 411, 1962.

RICHESON, E. M.: Industrial thallium intoxication. *Indust. Med. & Surg.* 27: 607, 1958.

TRUHAUT, R.: The toxicology of thallium. *J. Occup. Med.* 2: 334, 1960.

WINN, G. S.; GODFREY, E. L., AND NELSON, K. W.: Polarographic procedure for urinary thallium. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 14, 1952.

(175) Thorium and Compounds

Harmful Effects

Local Thorium nitrate may produce a primary contact dermatitis.

Routes of Entry Ingestion of liquid; inhalation of dust or gas.

Systemic Thorium compounds have not been reported as causing systemic poisoning in industry.

Mesothorium (radium-228), a radioactive decay product of thorium, produced malignant tumors in radium dial painters.

Thorotrast, a thorium dioxide suspension used formerly in radiographic contrast studies, has been reported to cause malignancy and hematopoietic changes several years following injection; however, a cause and effect relationship has not been established.

Special Diagnostic Tests

Analysis of blood and urine for thorium, breath for thoron, or feces for thorium X. *See* Fairhall, 1957, and Von Oettingen, 1958.

Recommended Threshold Limit

Thorium-232, 10^{-11} microcurie per cubic centimeter of air using *total body* as organ of reference. *See* National Committee on Radiation Protection, 1959, p. 83.

Potential Occupational Exposures

Alloy makers, magnesium
Ceramic makers
Crucible makers
Gas mantle makers
Glass makers
Incandescent lamp makers
Luminous pigment workers
Metal refiners
Nuclear reactor workers
Organic chemical synthesizers
Sunlamp makers
Thorium workers
Vacuum tube makers

References

ALBERT, R.; KLEVIN, P.; FRESCO, J.; HARLEY, J.; HARRIS, W., AND EISENBUD, M.: Industrial hygiene and medical survey of a thorium refinery. *A.M.A. Arch. Indust. Health* 11: 234, 1955.

BAKER, W. H.; BULKLEY, J. B.; DUDLEY, R. A.; EVANS, R. D.; M'CLUSKEY, H. B.; REEVES, J. D., JR.; RYDER, R. H.; SALTER, L. P., AND SHANAHAN, M. M.: Observations on the late effects of internally deposited mixtures of mesothorium and radium in twelve dial painters. *New Eng. J. Med.* 265: 1023, 1961.

FAIRHALL, L. T.: *Industrial Toxicology.* 2nd ed. Williams & Wilkins Co., Baltimore, 1957.

NATIONAL COMMITTEE ON RADIATION PROTECTION: Maximum permissible body burdens and maximum permissible concentrations of radionuclides in air and in water for occupational exposure. National Bureau of Standards Handbook 69. Issued June 5, 1959. U.S. Government Printing Office, Washington, D.C., 1959.

ROBERTS, J. C. AND CARLSON, K. E.: Hepatic duct carcinoma seventeen years after injection of thorium dioxide. *A.M.A. Arch. Path.* 62: 1, 1956.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(176) Tin and Compounds

stannum

Harmful Effects

Local Inorganic salt, tin tetrachloride, may act as skin and mucous membrane irritant. Certain organo-tins, especially of the tri-butyl series are potent skin irritants.

Routes of Entry Inhalation of dust. Ingestion or percutaneous absorption of organo-tins.

Systemic Prolonged inhalation of small amounts of tin oxide dust may result in production of pseudo-nodulation in lung which may be easily seen on chest roentgenogram. This condition is referred to as *stannosis* and is considered to be nonprogressive and nondisabling. Inorganic tin compounds are relatively nontoxic and are not generally thought of as important industrial hazards. Organo-tin compounds have been reported, upon ingestion, to cause acute cerebral edema often resulting in death. These compounds, it is thought, may be absorbed through intact skin.

Special Diagnostic Test

Analysis of tissue for abnormal amounts of tin. *See* Gonzales et al., 1958.

Recommended Threshold Limit

(Tentative)

Tin (inorganic compounds), 2 milligrams per cubic meter of air.

Tin (*organic compounds, as Sn*), 0.1 milligram per cubic meter of air; should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Babbitt metal (tin, copper, antimony) makers
Bactericide workers
Brass (essentially copper and zinc) founders
Britannia metal (tin, copper, antimony) makers
Bronze (tin, copper) founders
Ceramic makers
Dye makers
Dyers
Fungicide workers
Paper makers, sensitized
Perfume makers
Pewter (tin with lead, brass or copper) makers
Pigment makers
Plasticizer makers
Plastic workers
Putty makers
Rodenticide workers
Solder makers
Sugar processors
Textile makers
Textile printers
Tin ore smelters
Tin platers
Tin workers
Type metal (lead, antimony, tin) makers

References

BARNES, J. M. AND STONER, H. B.: Toxic properties of some dialkyl and trialkyl tin salts. *Brit. J. Indust. Med.* 15: 15, 1958.

GONZALES, T. A.; VANCE, M.; HELPERN, M., AND UMBERGER, C. J.: *Legal Medicine; Pathology and Toxicology*. 2nd ed. Appleton-Century-Crofts, New York, 1954.

LEWIS, C. E.: The toxicology of organometallic compounds; part 1. *J. Occup. Med.* 2: 183, 1960.

LYLE, W. H.: Lesions of the skin in process workers caused by contact with butyl tin compounds. *Brit. J. Indust. Med.* 15: 193, 1958.

PENDERGRASS, E. P. AND PRYDE, A. W.: Benign pneumoconiosis due to tin oxide. A case report with experimental investigation of the radiographic density of the tin oxide dust. *J. Indust. Hyg. & Toxicol.* 30: 119, 1948.

ROBERTSON, A. J.: Pneumoconiosis due to tin oxide. *In* King, E. J. and Fletcher, C. M. (editors): *Industrial Pulmonary Diseases*. Symposium, Postgraduate Medical School of London, 18–20 September 1957 and 25–27 March 1958. J. & A. Churchill, London, 1960.

ROBERTSON, A. J.; RIVERS, D.; NAGELSCHMIDT, G., AND DUNCUMB, P.: Stannosis; benign pneumoconiosis due to tin dioxide. *Lancet* 1: 1089, 1961.

(177) Titanium and Compounds

Harmful Effects

Local High concentrations of titanium dioxide dust may produce irritation of respiratory tract. Titanium tetrachloride has a highly corrosive action upon skin and mucous membranes.

Route of Entry. Inhalation of dust or fume.

Systemic Titanium dioxide fume may produce metal fume fever. Titanium tetrachloride fume, when inhaled, produces severe lung irritation often resulting in pulmonary edema.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Titanium dioxide) 15 milligrams per cubic meter of air.

Potential Occupational Exposures

Abrasive makers
Cemented carbide makers
Ceramic makers
Cermet makers
Corrosion inhibitor makers
Cosmetic makers
Electrode makers
Electronic equipment makers
Flameproofers
Foundry workers
Gem makers
Glass makers
Incandescent lamp makers
Ink makers
Lacquer makers
Linoleum makers
Nuclear steel makers
Painters
Paint makers
Paper makers
Pearl makers
Pigment makers
Porcelain enamel makers
Rayon makers
Refractory material makers
Resin makers
Rubber makers
Shoe whitener makers
Smoke screen makers
Steel workers
Surfactant makers
Surgical instrument makers
Tannery workers
Titanium alloy makers
Titanium metal refiners
Titanium workers
Vacuum tube makers
Varnishers
Waterproofing makers
Welding rod makers

Reference

LAWSON, J. J.: The toxicity of titanium tetrachloride. *J. Occup. Med.* 3: 7, 1961.

(178) Toluene

toluol, methylbenzene, phenylmethane, methylbenzol

Harmful Effects

Local Liquid or vapor is primary irritant of skin, eyes, and mucous membranes of upper respiratory tract. Small corneal vacuoles have been produced by mixture of substances containing toluene.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid leading to systemic toxicity is improbable.

Systemic Primary effect of both acute and chronic exposures is central nervous system depression. Symptoms and signs include headache, dizziness, weakness, fatigue, paresthesia, disturbance of coordination and equilibrium, insomnia, and loss of consciousness. Onset and severity of symptoms will depend upon degree and type of exposure. Hematologic effects are not prominent; however, temporary and slight lymphocytosis has occasionally been observed.

Special Diagnostic Tests

Analysis of urine for hippuric acid, and of blood for toluene. *See* Von Oettingen, 1958, and Gerarde, 1960.

Recommended Threshold Limit

200 parts per million parts of air by volume or 750 milligrams per cubic meter of air

Potential Occupational Exposures

Benzaldehyde makers
Benzoic acid makers
Detergent makers
Drug makers
Dye makers
Enamel makers
Explosive makers
Gasoline blenders
Gum processors
Histology technicians
Ink makers
Laboratory workers, chemical
Lacquerers
Lacquer makers
Leather workers
Oil processors
Painters
Paint makers
Paint thinner makers
Perfume makers
Pesticide workers
Petroleum refinery workers
Printers
Resin workers
Rubber cement makers
Saccharin makers
Solvent workers
Stainers
Stain makers
Tannery workers
Textile workers
Thermometer makers, vapor pressure
Toluene workers
Toluidine makers
Trinitrotoluene makers
Varnish makers
Vinyltoluene makers
Wax makers

References

GERARDE, H. W.: *Toxicology and Biochemistry of Aromatic Hydrocarbons.* Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1960.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(179) Tolylene Diisocyanate

2,4-toluene diisocyanate, TDI

Harmful Effects

Local TDI vapor is highly irritating to eyes, nose, and throat, and produces conjunctivitis and coryza-like symptoms. Although TDI liquid is mildly irritating to skin, dermatitis is rare. Continued contact may darken and harden skin.

Route of Entry Inhalation of vapor.

Systemic Pulmonary irritation, and in some cases pulmonary sensitization, may cause nonproductive cough, wheezing, shortness of breath, and tightness of chest. Diagnoses of bronchitis and bronchial asthma are frequently made.

Special Diagnostic Test

None.

Recommended Threshold Limit

(2,4-isomer) 0.02 part per million parts of air by volume or 0.14 milligram per cubic meter of air.

Potential Occupational Exposures

Abrasion resistant rubber makers
Adhesive workers
Aircraft builders
Insulation workers
Isocyanate resin workers
Lacquer workers
Mine tunnel coaters
Organic chemical synthesizers
Plastic foam makers
Plasticizer workers
Polyurethane foam makers
Polyurethane sprayers
Ship burners
Ship welders
Spray painters
Textile processors
Tolylene diisocyanate workers
Upholstery makers
Wire coating workers

References

BERGTHOLDT, C. P. I.: Recent welding practices at naval facilities. *Arch. Environ. Health* 2: 257, 1961.

BRUGSCH, H. G. AND ELKINS, H. B.: Toluene diisocyanate (TDI) toxicity. *New Eng. J. Med.* 268: 353, 1963. 31 references.

JOHNSTONE, R. T.: Toluene 2,4-diisocyanate; clinical features. *Indust. Med. & Surg.* 26: 33, 1957.

MUNN, A.: Experiences with diisocyanates. *Trans. Assoc. Indust. Med. Officers* 9: 134, 1960.

WILSON, R. H. AND WILSON, G. L.: Toxicology of diisocyanates. *J. Occup. Med.* 1: 448, 1959.

ZAPP, J. A.: Hazards of isocyanates in polyurethane foam plastic production. *A.M.A. Arch. Indust. Health* 15: 324, 1957.

(180) Toxaphene. *See* Pesticides Section

(181) Trichloroethylene

ethinyl trichloride, ethylene trichloride, trichloroethene

Harmful Effects

Local Liquid or high concentration of vapor may irritate eyes. Repeated contact with liquid or high vapor concentrations can produce a dry, scaly, and fissured dermatitis.

Routes of Entry Inhalation of vapor; of lesser importance, percutaneous absorption of liquid.

Systemic Trichloroethylene has a narcotic effect on central nervous system. In acute intoxications from low concentrations, manifestations include drowsiness, giddiness, dizziness, vertigo, fatigue, headache, exhilaration, nausea, vomiting, and incoordination. A characteristic symptom is intolerance toward alcohol. High vapor concentrations also have a narcotic effect and can produce unconsciousness, convulsions, coma, and death from respiratory paralysis. Death can occur from primary cardiac failure, ventricular fibrillation, and anoxia secondary to tachypnea and impaired alveolar ventilation. Reported cases of pulmonary edema may have been due to phosgene and hydrochloric acid, which are liberated when trichloroethylene is decomposed by heat.

A great variety of chronic effects have been attributed to trichloroethylene, such as liver damage, neuritis, and neurotic symptoms. Indication of liver damage is usually limited to abnormal liver function tests, but cases of acute yellow atrophy have been reported. The latter may have been due to contaminants or decomposition products. Injury to optic and trigeminal nerves has been reported. Neurotic symptoms are more difficult to evaluate and are doubted by some investigators.

Special Diagnostic Tests

Determination of urinary metabolites, particularly trichloroacetic acid and trichloroethanol. *See* Seto and Schultze, 1956, and Souček and Vlachová, 1960.

Recommended Threshold Limit

100 parts per million parts of air by volume or 520 milligrams per cubic meter of air.

Potential Occupational Exposures

Anesthetic gas makers
Caffeine processors
Cleaners
Coating makers
Degreasers
Disinfectant makers
Drug makers
Dry cleaners
Dye makers
Dyers

Electronic equipment cleaners
Electroplaters
Fat processors
Fumigant workers
Galvanizers
Gas purifiers
Gas workers, illuminating
Glass cleaners
Glue workers
Heat transfer workers
Lacquerers
Lacquer makers
Leather workers
Mechanics
Metal burnishers
Metal cleaners
Metal polishers
Metal scourers
Oil processors
Optical lens cleaners
Organic chemical synthesizers
Painters
Paint makers
Paint remover makers
Paper cup makers
Perfume makers
Petroleum refinery workers
Photographic plate cleaners
Polish makers
Printers
Resin workers
Rubber cementers
Rubber workers
Shoe workers
Soap makers
Solvent workers
Stainers
Stain makers
Textile cleaners
Tobacco denicotinizers
Trichloroethylene workers
Vacuum tube makers
Varnishers
Varnish makers
Veterinarians
Wax makers
Wool scourers

References

BARDODĚJ, Z., AND VYSKOČIL, J.: The problem of trichoroethylene in occupational medicine. Trichloroethylene metabolism and its effect on the nervous system evaluated as a means of hygienic control. *A.M.A. Arch. Indust. Health* 13: 581, 1956.

HARGARTEN, J. J.; HETRICK, G. H., AND FLEMING, A. J.: Industrial safety experience with trichloroethylene. *Arch. Environ. Health* 3: 461, 1961.

KLEINFELD, M., AND TABERSHAW, I. R.: Trichloroethylene toxicity. Report of five fatal cases. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 10: 134, 1954.

SETO, T. A. AND SCHULTZE, M. O.: Determination of trichloroethylene, trichloroacetic acid and trichloroethanol in urine. *Anal. Chem.* 28: 1625, 1956.

SOUČEK, B. AND VLACHOVÁ, D.: Excretion of trichloroethylene metabolites in human urine. *Brit. J. Indust. Med.* 17: 60, 1960.

WILLIAMS, J. W.: The toxicity of trichloroethylene. *J. Occup. Med.* 1: 549, 1959.

(182) Tricresyl Phosphate

TCP, tritolyl phosphate

The ortho-derivative is the most toxic of the three isomers of tricresyl phosphate. The meta- and para-isomers are relatively inactive, but may contain the ortho-isomer as a contaminant unless special precautions were taken during manufacture.

Harmful Effects

Local Contact dermatitis.

Routes of Entry Inhalation of vapor or mist; ingestion, percutaneous absorption of liquid.

Systemic Neurologic effects are caused by inhibition of cholinesterase as well as by demyelination and include polyneuritis and flaccid or spastic paralysis of extremities, usually the lower limbs. Recovery from paralysis may not be complete. There may be nystagmus, dysarthria, and accommodation difficulties.

Special Diagnostic Test

Cholinesterase activity of plasma. *See* Elkins, 1959.

Recommended Threshold Limit

(Triorthocresyl phosphate) 0.1 milligram per cubic meter of air.

Potential Occupational Exposures

Gasoline additive makers
Gasoline blenders
Hydraulic fluid workers
Lead scavenger makers
Lubricant additive workers
Nitrocellulose workers
Plasticizer workers
Polystyrene makers
Polyvinyl chloride makers
Solvent workers
Surgical instrument sterilizers
Tricresyl phosphate workers
Waterproofing makers

References

BIDSTRUP, P. L. AND BONNELL, J. A.: Anticholinesterases. Paralysis in man following poisoning by cholinesterase inhibitors. *Chem. & Indust.* (London) 24: 674, 1954. (Abst., *A.M.A. Arch. Indust. Health* 11: 178, 1955)

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

HUNTER, D.; PERRY, K. M. A., AND EVANS, R. B.: Toxic polyneuritis arising during the manufacture of tricresyl phosphate. *Brit. J. Indust. Med.* 1: 227, 1944.

TABERSHAW, I. R. AND KLEINFELD, M.: Manufacture of tricresyl phosphate and other alkyl phenyl phosphates; an industrial hygiene study. 2, Clinical effects of tricresyl phosphate. *A.M.A. Arch. Indust. Health* 15: 541, 1957.

(183) Trinitrotoluene

TNT, sym.-trinitrotoluol, methyltrinitrobenzene

Harmful Effects

Local Contact dermatitis from allergic hypersensitization. May stain skin a light yellow color and discolor hair to a reddish blond.

Routes of Entry Inhalation of dust, fume, or vapor. Ingestion of dust or percutaneous absorption from dust.

218-695 O-66-16

Systemic Gastrointestinal symptoms often occur first and include nausea, vomiting, and anorexia. Severe liver injury may follow and progress to acute yellow atrophy and death. Oxygen-carrying capacity of the blood is reduced through two mechanisms, namely, red blood corpuscle hemolysis, and formation of methemoglobin. Cyanosis, especially of lips, is a common finding. Breathlessness, weakness, and malaise may be present. Aplastic anemia has been reported to follow exposure to trinitrotoluene.

Special Diagnostic Test

Qualitative and quantitative analyses of urine for trinitrotoluene and its metabolites. *See* Von Oettingen, 1958. The Webster test can be used to detect trinitrotoluene on skin or in clothing. *See* Norwood, 1943.

Recommended Threshold Limit

1.5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Potential Occupational Exposures

Demolition workers
Dye intermediate makers
Explosive fillers
Photographic chemical makers
Trinitrotoluene workers

References

MCCONNELL, W. J.; FLINN, R. H., AND BRANDT, A. D.: Occupational diseases in government-owned ordnance explosives plants; observations on their prevalence and control during World War II. *Occup. Med.* 1: 551, 1946.

MCCONNELL, W. J. AND FLINN, R. H.: Summary of twenty-two trinitrotoluene fatalities in World War II. *J. Indust. Hyg. & Toxicol.*, 28: 76, 1946.

NORWOOD, W. D.: Trinitrotoluene (TNT); its effective removal from the skin by a special liquid soap. *Indust. Med.* 12: 206, 1943.

VON OETTINGEN, W. F.: *Poisoning, A Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(184) Turpentine

gum turpentine; oil of turpentine; spirit of turpentine; turps; gum spirit, derived from pine resin; wood turpentine, derived from pine stumps or sulfate wood pulp waste

Harmful Effects

Local Liquid may produce contact dermatitis from primary irritation as well as allergic hypersensitivity. High concentrations of vapor are irritating to eyes, nose, and throat.

Routes of Entry Inhalation of vapor; percutaneous absorption of liquid.

Systemic Headache, anorexia, gastritis, anxiety, excitement, mental confusion, tinnitus, bronchitis, and toxic nephritis.

Special Diagnostic Test

None.

Recommended Threshold Limit

100 parts per million parts of air by volume or 560 milligrams per cubic meter of air.

Potential Occupational Exposures

Art glass workers
Belt dressing makers
Camphor makers
Drug makers
Furniture polishers
Furniture polish makers
Ink makers
Insecticide makers
Lacquerers
Lacquer makers
Leather polish makers
Lithographers
Oil additive makers
Paint workers
Pine oil makers
Resin makers
Rubber reclaim workers
Rubber workers
Shoe polish makers
Solvent workers
Stainers
Stain makers
Stove polishers
Stove polish makers
Turpentine workers
Varnish workers
Wax makers

(185) Uranium and Compounds

Although uranium and its salts are highly toxic materials, poisoning attributable to their use in industry has not been a serious problem in this country.

Harmful Effects

Local Principal skin hazard in handling uranium metal is exposure of hands to beta radiation.

Route of Entry Inhalation of fume, dust or gas. The following uranium salts are reported to be capable of penetrating intact skin.

Uranyl nitrate
Uranyl fluoride
Uranium pentachloride
Uranium trioxide
Sodium diuranate
Ammonium diuranate
Uranium hexafluoride

Systemic Uranium and its salts, when absorbed into body, are highly toxic and may cause hepatic degeneration and chronic nephritis. Uranium

hexafluoride fumes, when inhaled, may produce a severe chemical pneumonitis. Prolonged inhalation of significant quantities of uranium, its salts, or its decay product, radon gas, may play an important role in causation of lung cancer.

Special Diagnostic Test

Analysis of urine for uranium. *See* Elkins, 1959.

Recommended Threshold Limit

Uranium (soluble compounds), 0.05 milligram per cubic meter of air.
Uranium (insoluble compounds), 0.25 milligram per cubic meter of air.

Potential Occupational Exposures

Atomic bomb workers
Ceramic makers
Glass makers
Hydrogen bomb workers
Nuclear reactor workers
Photographic chemical makers
Pigment makers
Uranium alloy makers
Uranium hexafluoride makers
Uranium millers
Uranium miners
Uranium paint makers
Uranium processors
Uranium workers
Vanadium millers
Vanadium miners

References

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

VOEGTLIN, C. AND HODGE, H. C.: *Pharmacology and Toxicology of Uranium Compounds*. McGraw-Hill Book Co., New York, 1949.

(186) Vanadium

Harmful Effects

Local Irritant to mucous membranes of eyes, nose, throat, and upper respiratory tract. Vanadium tetrachloride has been reported to be a skin irritant. Greenish discoloration of tongue is common among vanadium workers but is of no known toxicologic significance.

Route of Entry Inhalation of dust.

Systemic Reduction of serum cholesterol levels. Pulmonary irritation, possibly pneumonitis.

Special Diagnostic Test

Analysis of urine for vanadium. *See* Lewis, 1959, part 1.

Recommended Threshold Limit

Vanadium pentoxide dust, 0.5 milligram per cubic meter of air.
Vanadium pentoxide fume, 0.1 milligram per cubic meter of air.

Potential Occupational Exposures

Alloy makers
Boiler cleaners
Ceramic makers
Dye makers
Dyers
Ferrovanadium workers
Glass makers
Ink makers
Organic chemical synthesizers
Petroleum refinery workers
Photographic chemical makers
Textile dye workers
Uranium millers
Vanadium alloy makers
Vanadium millers
Vanadium miners
Vanadium workers

References

LEWIS, C. E.: The biological actions of vanadium. 1, Effects upon serum cholesterol levels in man. *A.M.A. Arch. Indust. Health* 19: 419, 1959.

LEWIS, C. E.: The biological effects of vanadium. 2, The signs and symptoms of occupational vanadium exposure. *A.M.A. Arch. Indust. Health* 19: 497, 1959.

TALVITIE, N. A. AND WAGNER, W. D.: Studies in vanadium toxicology. 2, Distribution and excretion of vanadium in animals. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 9: 414, 1954.

(187) Vinyl Chloride

chloroethylene, chlorethene, monochloroethylene

Harmful Effects

Local Liquid is irritating to skin and eyes.

Route of Entry Inhalation of gas.

Systemic Gas is central nervous system depressant and produces dizziness and disorientation. Two deaths from occupational exposure to vinyl chloride have been recorded. No characteristic findings were noted at autopsy.

Special Diagnostic Test

None.

Recommended Threshold Limit

500 parts per million parts of air by volume or 1,300 milligrams per cubic meter of air.

Potential Occupational Exposures

Organic chemical synthesizers
Polyvinyl resin makers
Rubber makers
Vinyl chloride workers

References

DANZIGER, H.: Accidental poisoning by vinyl chloride. Report of two cases. *Canad. Med. Assoc. J.* 82: 828, 1960.

HARRIS, D. K.: Health problems in the manufacture and use of plastics. *Brit. J. Indust. Med.* 10: 255, 1953.

(188) Warfarin. *See* Pesticides Section

(189) Xylene

xylol, dimethylbenzene

Harmful Effects

Local Xylene and its concentrated vapor are irritating to eyes, nose, and throat. Repeated contact of liquid with skin will produce a dry, scaly, and fissured dermatitis.

Routes of Entry Inhalation of vapor and, to a small but unimportant extent, percutaneous absorption of liquid.

Systemic Acute toxicity of inhaled xylene vapor is due to its vasodilatory and narcotic effects. Symptoms include flushing of face, headache, fatigue, confusion, paresthesias, dizziness, sleepiness, and unconsciousness. Chronic xylene poisoning probably does not occur.

Special Diagnostic Test

None.

Recommended Threshold Limit

200 parts per million parts of air by volume or 870 milligrams per cubic meter of air.

Potential Occupational Exposures

Adhesive workers
Aviation gasoline workers
Bacteriologists
Benzoic acid makers
Brake lining makers
Catgut sterilizers
Color printers
Drug makers
Dye makers
Enamel workers
Histology technicians
Ink makers
Lacquerers
Lacquer makers
Leather makers
Lithographers
Microscopists
Organic chemical synthesizers
Painters
Pathologists
Pesticide workers
Petroleum refinery workers
Phthalic anhydride makers
Polyethylene terephthalate film makers
Protective coating workers
Quartz crystal oscillator makers
Resin makers
Rubber cement makers
Rubber workers
Silk finishers
Solvent workers
Stainers
Stain makers
Terephthalic acid makers
Vitamin makers
Xylene workers

(190) Zinc and Compounds

Harmful Effects

Local Zinc chloride is extremely irritating to skin and may produce extensive ulceration; very irritating to eyes, nose, and throat. Perforation of nasal septum may be produced. Zinc chromate, zinc cyanide and zinc sulfate may cause dermatitis.

Route of Entry Inhalation of fume, dust, or vapor.

Systemic Inhalation of zinc chloride fumes may produce severe pneumonitis. Certain smoke-screening compounds produce upon ignition essentially zinc chloride and aluminum oxide. When inhaled, the zinc chloride in extremely high concentrations of finely divided particles will produce a chemical irritation of the upper respiratory tract; in the concentrations usually met with among military personnel, an insidious chemical pneumonitis has been reported to occur.

When metallic zinc is heated to a temperature near its boiling point, very finely divided zinc oxide fume is produced. Inhalation of freshly formed fumes may produce a brief, self-limiting illness known variously as *zinc chills, metal fume fever, brass chills,* and *brass founder's fever.* This condition is characterized by chills, fever, nausea, vomiting, muscular pain, dryness of mouth and throat, headache, fatigue, and weakness. There may also be a slight leukocytosis. These signs and symptoms usually abate in 12 to 24 hours with complete recovery following. Immunity from this condition is rapidly acquired if exposure occurs daily but is quickly lost during holidays or over weekends. Certain other metallic oxide fumes may cause this condition. These include the oxides of nickel, copper, magnesium, cadmium, iron, mercury, tungsten and titanium.

Special Diagnostic Test

None.

Recommended Threshold Limit

(Zinc oxide fume) 5 milligrams per cubic meter of air.

Potential Occupational Exposures

Zinc

Alloy makers
Arc welders, electric
Brass foundry workers
Braziers
Bronze foundry workers
Electric fuse makers
Electroplaters
Galvanizers
Metal cutters
Metalizers
Metal sprayers
Printing plate makers
Roofing makers
Zinc smelters
Zinc workers

Zinc compounds

Activated carbon makers
Adhesive makers
Antistatic agent makers
Battery makers, dry
Candle makers
Ceramic makers
Cosmetic makers
Crepe makers
Dental cement makers
Dentifrice makers
Deodorant workers
Disinfectant makers
Dye makers
Electroplaters
Embalmers
Embalming fluid workers
Enamel makers
Feed additive makers
Fungicide workers
Galvanizers
Gelatin makers
Glass etching agent makers
Glue makers
Illuminating gas workers
Ink makers
Insecticide workers
Iron copper-platers
Linoleum makers
Lithopone makers
Lumber fireproofers
Magnesium oxide cement makers
Match makers
Mercerizers
Metal etchers
Metal platers
Microscopists
Military personnel
Organic chemical synthesizers
Painters
Paint makers
Paper makers
Petroleum refinery workers
Pigment makers, steel
Polish makers, steel
Railroad tie preservers
Rayon makers
Rubber makers
Seed treaters
Silk makers
Smoke screen makers
Soap makers
Solder flux makers
Taxidermists
Textile crimpers
Textile fireproofers
Textile makers
Textile mordanters
Textile sizers
Textile weighters
Varnish makers
Wood preservative workers

References

JOHNSON, F. A. AND STONEHILL, R. B.: Chemical pneumonitis from inhalation of zinc chloride. *Dis. Chest* 40: 619, 1961.

MORRIS, G. E.: Toxic hazards; metal fume fever. *New Eng. J. Med.* 260: 1091, 1959.

ROHRS, L. C.: Metal-fume fever from inhaling zinc oxide. *A.M.A. Arch. Indust. Health* 16: 42, 1957.

(191) Zirconium Compounds

Harmful Effects

Local Certain of the zirconium salts used in antiperspirant preparations have been reported to produce granulomatous lesions in axilla.

Route of Entry Inhalation of dust or fume.

Systemic No systemic effects in workers have been reported.

Special Diagnostic Test

None.

Recommended Threshold Limit

Zirconium compounds (as Zr), 5 milligrams per cubic meter of air.

Potential Occupational Exposures

Abrasive makers
Alloy makers
Arc lamp makers
Ceramic workers
Cermet makers
Crucible makers
Deodorant makers
Drug makers
Dye makers
Enamel makers
Explosive makers
Foundry workers
Furnace lining makers
Gem makers
Glass makers
Incandescent lamp makers
Lacquer makers
Lubricant makers
Metallurgists
Nuclear reactor workers
Paint makers
Phosphor makers
Photographic illuminant makers
Pigment makers
Polish makers
Pottery makers
Rayon spinneret makers
Refractory material makers
Steel makers
Tannery workers
Textile waterproofers
Vacuum tube makers
Varnish makers
Waterproofers
Zirconium workers

References

REED, C. E.: A study of the effects on the lung of industrial exposure to zirconium dusts. *A.M.A. Arch. Indust. Health* 13: 578, 1956.

SHEARD, C., JR.; CORMIA, F. E.; ATKINSON, S. C., AND WORTHINGTON, E. L.: Granulomatous reactions to deodorant sticks. *J. Am. Med. Assoc.* 164: 1085, 1957.

General References, Chemical Hazards Section

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION: *Hygienic Guide Series.* The Association, Detroit, various years.

AMERICAN PETROLEUM INSTITUTE: *API Toxicological Reviews.* The Institute, New York, various years.

AMERICAN STANDARDS ASSOCIATION: *American Standard Maximal Acceptable Concentrations.* The Association, New York, various years.

BROWNING, E.: *Toxicity of Industrial Organic Solvents.* Chemical Publishing Co., New York, 1953.

BROWNING, E.: *Toxicity of Industrial Metals.* Butterworths, London, 1961.

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

FAIRHALL, L. T.: *Industrial Toxicology.* 2nd ed. Williams & Wilkins Co., Baltimore, 1957.

FLEMING, A. J.; D'ALONZO, C. A., AND ZAPP, J. A. (EDITORS): *Modern Occupational Medicine.* 2nd ed. Lea & Febiger, Philadelphia, 1960.

GLEASON, M. N.; GOSSELIN, R. E., AND HODGE, H. C.: *Clinical Toxicology of Commercial Products; Acute Poisoning (Home & Farm).* Williams & Wilkins Co., Baltimore, 1957. Supp. 1959.

HAMILTON, A. AND HARDY, H. L.: *Industrial Toxicology.* Paul B. Hoeber, New York, 1949.

HUNTER, D.: *The Diseases of Occupations.* 3rd ed. Little, Brown and Co., Boston, 1962.

JOHNSTONE, R. T. AND MILLER, S. E.: *Occupational Diseases and Industrial Medicine.* W. B. Saunders Co., Philadelphia, 1960.

KIRK, R. E. AND OTHMER, D. F. (EDITORS): *Encyclopedia of Chemical Technology.* 15 vols. and 2 supplement vols. Interscience Publishers, New York, 1947–1960.

MALKINSON, F. D.: Percutaneous absorption of toxic substances in industry. *A.M.A. Arch. Indust. Health* 21: 87, 1960.

MANUFACTURING CHEMISTS ASSOCIATION: *Chemical Safety Data Sheets.* The Association, Washington, D.C., various years.

NATIONAL SAFETY COUNCIL: *Data Sheets.* The Council, Chicago, various years.

PATTY, F. A. (EDITOR): *Industrial Hygiene and Toxicology.* Vol. 2. Interscience Publishers, New York, 1949. 2nd ed., 1963.

ROSE, A. AND ROSE, E. (EDITORS): *The Condensed Chemical Dictionary.* 6th ed. Reinhold Publishing Corp., New York, 1961.

SAX, N. I.: *Dangerous Properties of Industrial Materials.* Reinhold Publishing Corp., New York, 1957.

SCHWARTZ, L.; TULIPAN, L., AND BIRMINGHAM, D. J.: *Occupational Diseases of the Skin.* 3rd ed. Lea & Febiger, Philadelphia, 1957.

SHELL CHEMICAL CORPORATION: *Industrial Hygiene Bulletin.* Toxicity Data Sheets. The Corporation, New York, various years.

SMYTH, H. F., JR.: Improved communication. Hygienic standards for daily inhalation. *Am. Indust. Hyg. Assoc. Quart.* 17: 129, 1956.

STECHER, P. G.; FINKEL, M. J.; SIEGMUND, O. H., AND SZAFRANSKI, B. M.: *The Merck Index of Chemicals and Drugs.* 7th ed. Merck & Co., Rahway, N.J.; 1960.

• section VII

PESTICIDES

ROY L. GIBSON, M.D., AND THOMAS H. MILBY, M.D.

The beneficial effects of pesticides in disease control and crop production are well recognized. In many areas of the world the chemical eradication of insect and rodent vectors has partially or completely eliminated such diseases as malaria, yellow fever, dengue, and plague. Populations tend to increase with the disappearance of such potentially lethal diseases, and the current population explosion is in part the result of these pest control measures.

Growing populations require more land for habitation and food for consumption; therefore, optimum crop production becomes a necessity. The shrinking-world phenomenon forces the public health worker and others to become acquainted not only with an increasing variety of pests of all kinds but also with the new pesticides which are being developed to cope with the problem. Pesticides popular today may be of little use tomorrow because of the development of pest resistance to these chemicals. Thus, man is confronted with thousands of these products, many of which may impose a human liability if exposure is sufficient and uncontrolled.

For the purpose of this presentation, the pesticides are divided into five major groups that denote their principal proposed use. These groups include insecticides, rodenticides, fungicides, herbicides, and fumigants. The insecticides are further categorized according to chemical configuration into chlorinated hydrocarbons, phosphate esters, and miscellaneous insecticides.

For clarity and to facilitate presentation, the pesticides are generally designated by the term or terms most commonly appearing in the medical literature. Pesticide solvents such as kerosine, naphtha, toluene and xylene, which are not presented here, are included in the section on Chemical Hazards.

No attempt is made to present the clinical effects of all pesticides currently used. Such information may be found in more extensive treatises on the subject. Specific toxicologic information is lacking for many of these compounds, and others are not of sufficient toxicity or use to pose significant health problems.

Hazardous exposures may occur in both occupational and nonoccupational pursuits. Manufacturers, spray pilots, crop workers, farmers, nursery workers, seed-treaters, soil fumigators, exterminators, and others engaged in the development, manufacture, distribution, and use of these chemicals are subject to intoxication if proper precautions are not observed. Such nonoccupational activities as gardening, yard maintenance, and camping expose millions more to these agents. An understanding of their harmful effects is therefore essential.

Insecticides

Chlorinated Hydrocarbons

All chlorinated hydrocarbon insecticides, in general, produce similar physiologic effects in man, and consequently their clinical characteristics can be discussed collectively. However, they vary in their ability to cause clinical symptoms by virtue of differences in absorption and excretion. Intoxication may result from ingestion, inhalation, or percutaneous absorption. Headache, loss of appetite, nausea, vomiting, dizziness, tremors, convulsions, and coma occur following excessive exposure.

The insecticide or its derivatives can often be demonstrated in urine, stomach contents or body tissues, especially fat. Some of the chemicals in this group—for example, chlordane and benzene hexachloride—have been reported to cause dermatitis either by primary irritation or hypersensitization.

The chlorinated hydrocarbons of greatest significance and their threshold limit values, in milligrams per cubic meter of air, follow.

Aldrin	0.25 mg. per cu. m.*
Benzene hexachloride	Not established.
Chlordane	0.5 mg. per cu. m.
DDT (chlorophenothane)	1 mg. per cu. m.*
Dieldrin	0.25 mg. per cu. m.*
Endrin	0.1 mg. per cu. m. (tentative)*
Heptachlor	0.5 mg. per cu. m. (tentative)
Lindane (gamma-benzene hexachloride)	0.5 mg. per cu. m.
Methoxychlor	15 mg. per cu. m.
Terpene polychlorinates	Not established.
Toxaphene (chlorinated camphene, 60 percent).	0.5 mg. per cu. m.

*Should be reduced when also absorbed percutaneously.

Phosphate Esters

The phosphate ester insecticides are characterized by the similarity of their structural relationship—they are phosphoric acid derivatives—and by their identical mechanism of toxic action. They differ widely, however, in inherent toxicity and, to some extent, in rate of absorption and excretion.

These compounds are readily absorbed through the intact skin. They may also be absorbed following ingestion or inhalation of contaminated materials.

The organic phosphorus compounds act as irreversible inhibitors of cholinesterase, thereby allowing the accumulation of large amounts of acetylcholine. When a critical level of cholinesterase depletion is reached, usually about 20 percent of normal, symptoms and signs of poisoning become manifest. Symptoms may include blurred vision, weakness, nausea, headache, abdominal cramps, chest discomfort, and diarrhea. Signs may include miosis, muscle twitching, salivation, sweating, tearing, cyanosis, convulsions, and coma. Peripheral nerve damage has been reported as an occasional sequela of poisoning by certain organic phosphates.

Diagnosis is based upon the following criteria: a definite history of exposure of six hours or less before the onset of symptoms, clinical evidence of diffuse parasympathetic stimulation, and depression of plasma and red blood cell cholinesterase. The urinary excretion of p-nitrophenol may be useful in the diagnosis of parathion or EPN intoxication.

The more commonly encountered phosphate ester insecticides together with their threshold limit values, in milligrams per cubic meter of air, are grouped according to toxicity in the following list.

Highly toxic:

Dematon (Systox[R])	0.1 mg. per cu. m.* (tentative)
Ethyl p-nitrophenyl thionobenzene phosphonate (EPN).	0.5 mg. per cu. m.*
Guthion[R]	Not established.
Hexaethyl tetraphosphate (HETP)	Not established.
Methyl parathion	Not established.
Octamethyl pyrophosphoramide (OMPA).	Not established.
Parathion	0.1 mg. per cu. m.*
Phosdrin[R]	0.1 mg. per cu. m.*
Pyrazoxon[R]	Not established.
Tetraethyl dithionopyrophosphate (TEDP).	0.2 mg. per cu. m.*
Tetraethyl pyrophosphate (TEPP)	0.05 mg. per cu. m.*
Thimet[R]	Not established.
Trithion[R]	Not established.

Moderately toxic:

Diazinon[R]	Not established.
Dimethyldichlorvinyl phosphate (DDVP).	1 mg. per cu. m.* (tentative)
Ethion	Not established.

*See footnote at end of table.

Slightly toxic:

Chlorthion[R]	Not established.
Dipterex[R]	Not established.
Malathion	15 mg. per cu. m.*

*Should be reduced when also absorbed percutaneously.

Miscellaneous Insecticides

Although the newer synthetic pesticides previously discussed in this section are becoming increasingly popular, the following compounds continue to find significant usage.

Lead arsenate and arsenite—These compounds enter the body by inhalation, ingestion, or percutaneous absorption. Signs and symptoms of poisoning are similar to those characteristic of lead or arsenic intoxication. Acute symptoms includes nausea, vomiting, abdominal pain, diarrhea, muscle cramps, excitation, and disorientation. Chronic poisoning is manifested by anorexia, weakness, weight loss, pallor, colic, diarrhea, peripheral neuritis, hepatitis, and nephritis. A vesicular dermatitis has frequently been reported. The carcinogenic hazard from chronic arsenic exposure cannot be ignored. The recommended threshold limit value for lead arsenate is 0.15 milligram per cubic meter of air.

Nicotine—This extremely toxic alkaloid is capable of producing nervous system stimulation followed by severe nervous system depression. The effects may result from ingestion, inhalation, or rapid percutaneous absorption of the material. Analysis for urinary nicotine may aid in the diagnosis. The recommended threshold limit value is 0.5 milligram per cubic meter of air which should be reduced when also absorbed percutaneously.

Pyrethrum—This material is not particularly toxic; however, primary contact dermatitis and allergic skin and pulmonary reactions have occurred following minimal exposure to the dust. The recommended threshold limit value is 5 milligrams per cubic meter of air.

Rotenone—This plant extract is more toxic than pyrethrum but, as normally used, is not excessively hazardous. Contact dermatitis and numbness of the oral mucous membranes may follow sufficient exposure. The recommended threshold limit value is 5 milligrams per cubic meter of air.

Rodenticides

Rodenticides of first importance include sodium fluoroacetate, strychnine, thallium sulfate, and warfarin. For information on rodenticides containing arsenic, barium, cyanide and phosphorus, reference may be made to the appropriate chemical in the section on Chemical Hazards.

Sodium fluoroacetate (Compound 1080)—This material may be absorbed through the skin and the respiratory and gastrointestinal tracts. Clinical manifestations include nausea, apprehension, cardiac irregularities, and con-

vulsions followed by central nervous system depression. The recommended threshold limit value is 0.05 milligram per cubic meter of air which should be reduced when also absorbed percutaneously.

Strychnine—Severe convulsions without loss of consciousness are characteristic of strychnine poisoning. Death is usually a result of asphyxia or involvement of vital brain centers. The compound may be identified in the urine soon after ingestion. The recommended threshold limit value is 0.15 milligram per cubic meter of air.

Thallium sulfate—Intoxication may follow ingestion or skin absorption. Acute poisoning is characterized by severe gastroenteritis following a latent period of 12 to 24 hours. Other effects may include liver and kidney damage, encephalopathy, neuritis, ataxia and alopecia. Recovery is slow. Thallium may be demonstrated in the urine. The recommended threshold limit value for soluble compounds of thallium is 0.1 milligram per cubic meter of air which should be reduced when also absorbed percutaneously.

Warfarin—Intoxication occurs following chronic ingestion of warfarin, and the following signs and symptoms due to inhibition of prothrombin formation and capillary fragility are observed: pallor, bleeding gums, nose bleeds, petechial rash, bruises, blood in the urine and stools, and shock. Laboratory studies reveal a prolonged prothrombin time. The recommended threshold limit value for warfarin is 0.1 milligram per cubic meter of air.

Fungicides

The fungicides are a heterogeneous group of chemicals and, with the major exception of the dithiocarbamates, have been in use for many years. Many of the fungicides—formaldehyde, furfural, phenol, tetramethylthiuram disulfide and compounds of boron, chromium, copper, mercury, tin and zinc—some of which are also used as herbicides and insecticides, are discussed in the section on Chemical Hazards.

The dithiocarbamates include ferbam (ferric dimethyldithiocarbamate), ziram (zinc dimethyldithiocarbamate), maneb (manganous ethylene bisdithiocarbamate), nabam (disodium ethylene bisdithiocarbamate) and zineb (zinc ethylene bisdithiocarbamate). Their chief adverse effects are irritation of the skin, eyes, and upper respiratory tract.

Threshold limit values, in milligrams per cubic meter of air, have been recommended for the following fungicides as shown.

Ferbam	15 mg. per cu. m.
Formaldehyde	6 mg. per cu. m.
Organic mercurials	0.01 mg. per cu. m.*
Pentachlorophenol (PCP)	0.5 mg. per cu. m.*
Sodium dichromate	0.1 mg. per cu. m. (as CrO_3)

*Should be reduced when also absorbed percutaneously.

Herbicides

Herbicides, or weed killers, may be classified as pesticide chemicals. They can kill plants on contact, or they can be translocated; that is, absorbed by one part of the plant and carried to other parts where they exert their primary toxic effect. Most of the commonly used herbicides—ammonium sulfamate, dalapon, phenoxyacetic acid derivatives, carbamate derivatives, petroleum oils, sodium borate, Crag[R] herbicide—have a low toxicity and have caused little difficulty among users.

Some herbicides pose more serious problems; for example, the central nervous system effects of maleic hydrazide or the methemoglobinemia and central nervous system depression of sodium chlorate. Pentachlorophenol, a metabolic stimulant, has been responsible for several deaths because of hyperthermia. Amino triazol has produced cancer in experimental animals, but there have been no untoward effects reported in man.

Herbicides with cutaneous effects include trichloroacetic acid, a corrosive irritant of the skin aud mucous membranes; maleic hydrazide, a producer of allergic contact dermatitis; pentachlorophenol, a producer of a primary irritant type of contact dermatitis; and creosote, a primary irritant and photosensitizer.

Reference may be made to chemicals in the section on Chemical Hazards for the toxicity of the following herbicides: Arsenic trioxide and sodium arsenate (see Arsenic), copper sulfate (see Copper and Compounds), creosote compounds (see Cresol and Phenol), dinitrophenols (see Dinitrophenol), kerosine, and phenylmercuric acetate (see Mercury and Compounds).

Threshold limit values, in milligrams per cubic meter of air, have been recommended for the following herbicides as shown.

Ammonium sulfamate (Ammate[R])	15 mg. per cu. m.
Crag[R] herbicide	15 mg. per cu. m.
2,4-D (2,4-dichlorophenoxyacetic acid)	10 mg. per cu. m.
Pentachlorophenol (PCP)	0.5 mg. per cu. m.*
Phenylmercuric acetate (PMA)	0.01 mg. per cu. m.* (for organic mercury)
2,4,5–T (2,4,5-trichlorophenoxyacetic acid).	10 mg. per cu. m. (tentative)

*Should be reduced when also absorbed percutaneously.

Fumigants

Fumigants are pesticides which may be applied in the solid, liquid, or gaseous state. A combination of high volatility with high pest toxicity is generally desired; however, compounds with low volatility may be preferred for soil fumigation. The possibility of excessive exposures exists wherever

fumigants are used, as in fumigating grains, soils, clothes, furs, homes, warehouses, barns, ships, mills, freight cars, and greenhouses.

Each of the following compounds has found use as a fumigant. Because they have other industrial applications as well, they are discussed individually in the section on Chemical Hazards.

Acrylonitrile
Carbon Disulfide
Carbon Tetrachloride
p-Dichlorobenzene (see Chlorinated Benzenes)
Dioxane
Ethylene Dibromide
Ethylene Dichloride
Ethylene Oxide
Hydrogen Cyanide
Methyl Bromide (see Bromine and Compounds)
Methylene Chloride
Methyl Formate
Naphthalene
Perchloroethylene
Propylene Dichloride
Sulfur Dioxide
Tetrachloroethane
Trichloroethylene

References

ARTERBERRY, J. D.; DURHAM, W. F.; ELLIOTT, J. W., AND WOLFE, H. R.: Exposure to parathion. Measurement by blood cholinesterase level and urinary p-nitrophenol excretion. *Arch. Environ. Health* 3: 476, 1961.

ASSOCIATION OF AMERICAN PESTICIDE CONTROL OFFICIALS: *Pesticide Chemicals Official Compendium.* A. B. Heagy, University Post Office, College Park, Md., 1962. Loose-leaf service.

COMMITTEE ON PESTICIDES: Outlines of information on pesticides. Part 1. Agricultural fungicides. *J. Am. Med. Assoc.* 157: 237, 1955.

COMMITTEE ON TOXICOLOGY: Occupational dieldrin poisoning. *J. Am. Med. Assoc.* 172: 2077, 1960.

COMMUNICABLE DISEASE CENTER, PUBLIC HEALTH SERVICE: Clinical memoranda on economic poisons. Pub. Health Service Pub. No. 476. U.S. Government Printing Office, Washington, D.C., 1956.

DIVISION OF BIOLOGY AND AGRICULTURE, NATIONAL RESEARCH COUNCIL–NATIONAL ACADEMY OF SCIENCES: *Handbook of Toxicology.* Vol. 3, Insecticides. Vol. 5, Fungicides. W. B. Saunders Co., Philadelphia, 1959.

DURHAM, W. F.; GANES, T. B., AND HAYES, W. J. JR.: Paralytic and related effects of certain organic phosphorus compounds. *A.M.A. Arch. Indust. Health* 13: 326, 1956.

DURHAM, W F. AND HAYES., W. J., JR.: Organic phosphorus poisoning and its therapy; with special reference to modes of action and compounds that reactivate inhibited cholinesterase. *Arch. Environ. Health* 5: 21, 1962.

FREAR, D. E. H. (EDITOR): *Pesticide Index.* College Science Publishers, State College, Pa., 1961.

GAINES, T. B.: The acute toxicity of pesticides to rats. *Toxicol. Appl. Pharmacol.* 2: 88, 1960.

GOLZ, H. H. AND SHAFFER, C. B.: *Toxicological Information on Cyanamide Insecticides.* American Cyanamid Co., New York, 1960.

HAYES, W. J., JR.: The toxicity of dieldrin to man; report on a survey. *Bull. WHO* 20: 891, 1959.

HAYES, W. J., JR.: Pesticides in relation to public health. *Ann. Rev. Entomol.* 5: 379, 1960.

218-695 O-66-17

HAYES, W. J., JR.; DIXON, E. M.; BATCHELOR, G. S., AND UPHOLT, W. M.: Exposure to organic phosporus sprays and occurrence of selected symptoms. *Pub. Health Rep.* 72: 787, 1957.

HAYES, W. J., JR.; DURHAM, W. F., AND CUETO, C., JR.: The effect of known repeated oral doses of chlorophenothane (DDT) in man. *J. Am. Med. Assoc.* 162: 890, 1956.

HEARN, C. E. D.: Trithion poisoning. *Brit. J. Indust. Med.* 18: 231, 1961.

HEATH, D. F.: *Organophosphorus Poisons; Anticholinesterases and Related Compounds.* Pergamon Press, London and New York, 1961.

HOOGENDAM, I.; VERSTEEG, J. P. J., AND DE VLIEGER, M.: Electroencephalograms in insecticide toxicity. *Arch. Environ. Health* 4: 86, 1962.

LUNDGREN, K. D. AND SWENSSON, A.: A survey of results of investigations on some organic mercury compounds used as fungicides. *Am. Indust. Hyg. Assoc. J.* 21: 308, 1960.

LYLE, W. H.: Lesions of the skin in process workers caused by contact with butyl tin compounds. *Brit. J. Indust. Med.* 15: 193, 1958.

MCGEE, L. C.; REED, H. L., AND FLEMING, J. P.: Accidental poisoning by toxaphene. *J. Am. Med. Assoc.* 149: 1124, 1952.

NATIONAL ARCHIVES OF THE UNITED STATES: Federal Register, the primary source of administrative law. U.S. Government Printing Office, Washington, D.C. This periodical appearing daily publishes the summaries of pesticide tolerances and exemptions prepared by the Food and Drug Administration.

O'BRIEN, R. O.: *Toxic Phosphate Esters; Chemistry, Metabolism and Biologic Effects.* Academic Press, New York, 1960.

ORTLEE, M. F.: Study of man with prolonged intensive occupational exposure to DDT. *A.M.A. Arch. Indust. Health* 18: 433, 1958.

PETTY, C. S.: Organic phosphate insecticide poisoning; residual effects in two cases. *Am. J. Med.* 24: 467, 1958.

PRESIDENT'S SCIENCE ADVISORY COMMITTEE: Use of pesticides; a report. U.S. Government Printing Office, Washington, D.C., (May 15) 1963. 25 pp. Classifies pesticides and presents material on their distribution and persistence in the environment, their biologic effect, and their toxicity. Four pages of recommendations.

PRINCI, F.: Toxicology, diagnosis, and treatment of chlorinated hydrocarbon insecticide intoxications. *A.M.A. Arch. Indust. Health* 16: 333, 1957.

QUERIES AND MINOR NOTES: Toxicity of 2,4-dichlorophenoxyacetic acid (weed spray). *J. Am. Med. Assoc.* 162: 1269, 1956.

WITTER, R. F.: Measurement of blood cholinesterase; a critical account of methods of estimating cholinesterase with reference to their usefulness and limitations under different conditions. *Arch. Environ. Health* 6: 537, 1963.

WOLFE, H. R.; DURHAM, W. F., AND ARMSTRONG, J. F.: Health hazards of the pesticides endrin and dieldrin. *Arch. Environ. Health* 6: 458, 1963.

WOLFSIE, J. H.: Blood cholinesterase activity. Practical considerations in routine testing programs. *A.M.A. Arch. Indust. Health* 16: 403, 1957.

• section VIII

PLASTICS AND SYNTHETIC RESINS

MARCUS M. KEY, M.D.

Plastics were so named because at some stage in their manufacture they were soft enough to be formed into various shapes. Synthetic resins were named for their similarity to natural resins, such as rosin. Generally the words are interchangeable, although, for a specific plastic or resin, it is customary to use one in preference to the other; thus, epoxy resin rather than epoxy plastic.

There are a number of basic chemical or family groups of plastics and resins, several of which have been arbitrarily chosen for presentation. Each group may be classified as either thermosetting or thermoplastic. Thermosetting resins cure or harden under heat and cannot be reshaped subsequently. Examples are alkyds, allyls, aminos, diisocyanates, epoxies, phenolics, and polyesters. Thermoplastic resins can be softened by heat and reshaped repeatedly. Examples are acrylics, cellulosics, fluorocarbons, nylons, polyethylenes, polystyrenes, and vinyls.

Plastics and resins are made by polymerization or condensation. In polymerization, a large number of identical molecules, called monomers, unite to form a larger molecule, called a polymer. In condensation, a number of molecules, not necessarily of the same composition, unite to form a molecule dissimilar in composition to the components, with the liberation of water or other simple substances.

In addition to monomers and condensate components, many other materials are used in compounding plastics and resins; namely, accelerators, catalysts, copolymers, dyes, fillers (asbestos, diatomite, glass fiber, mica, quartz, sand and many other substances), mold lubricants, pigments, plasticizers, solvents, stabilizers, and ultraviolet absorbers.

The major hazard from the manufacture, curing, and processing of plastics and resins is contact dermatitis which may be due either to primary irritation or to allergic sensitization. As a rule, completely condensed or polymerized resins do not cause dermatitis. Frequent offenders are the catalysts,

low molecular weight polymers, monomers, and uncured condensate components. Accelerators, plasticizers, solvents, and other components may also cause dermatitis.

In addition to dermatitis, the manufacture, curing, and processing of some plastics and resins may be accompanied by systemic reactions such as bronchial asthma from tolylene diisocyanate and polymer fume fever from polytetrafluoroethylene. Most of the components that produce systemic effects are included in the section on Chemical Hazards under the following headings:

Acetic Acid
Acetone (See Ketones)
Allyl Alcohol
Ammonia
Amyl Acetate
Cresol
Epichlorohydrin
Ethyl Acetate
Formaldehyde
Furfural
Hexamethylenetetramine
Ketones
Methyl Chloride
Organo-tin Compounds (See Tin)
Phenol
Phosgene
Phthalic Anhydride
Styrene
Tolylene Diisocyanate
Tricresyl Phosphate
Vinyl Chloride

In the following 14 major family groups, material is presented with regard to harmful effects, and potential occupational exposures. Because of the rapid changes in the plastics industry, it is understandable that the potential occupational exposures as given may require periodic revision. Basic information on chemical technology and production technics may be secured by referring to the *Modern Plastics Encyclopedia* issued annually as a part of the periodical, *Modern Plastics*.

Thermosets

(1) *Alkyd Resins*

Some of the condensate materials can produce contact dermatitis; for example, phthalic and maleic anhydride.

Potential Occupational Exposures

Alkyd resin makers
Lacquer makers
Textile finishers

(2) *Allyl Resins*

Contact dermatitis has occurred from the monomers as well as their precursors, allyl alcohol and diglycochlorformate. The use of phosgene to make diglycochlorformate can produce pulmonary edema. The organic peroxide catalysts used to cure the prepolymers can produce contact dermatitis.

Potential Occupational Exposures

Aircraft part makers
Allyl prepolymer makers
Decorative laminators
Electric part makers
Missile component makers

(3) *Amino Resins*

urea-formaldehyde resins, melamine-formaldehyde resins

The most common dermatitis-producing component is formaldehyde. It may be encountered in the production of the amino resins or may be released subsequently from improperly cured urea-formaldehyde or melamine-formaldehyde crease-resistant fabrics and produce allergic contact dermatitis and conjunctivitis. Formaldehyde reaction products, for example, dimethylol urea, in textile finishes can also act as sensistizers. Hexamethylenetetramine, a formaldehyde liberator, is used as a stabilizer for urea-formaldehyde molding compounds and can also produce dermatitis.

Potential Occupational Exposures

Adhesive makers
Aerosol dispenser makers
Automobile ignition makers
Button makers
Crease-resistant textile finishers
Cutlery handle makers
Decorative tabletop makers
Dish makers
Dress makers
Foundry workers
Furniture makers
Gluers
Lace tenters
Paper treaters
Plywood makers
Shirt makers
Soap makers

(4) *Diisocyanate Resins*

polyurethane resins

The diisocyanates, usually tolylene diisocyanate and diphenyl-methane diisocyanate, are irritating to the skin, and some of the tertiary amine catalysts are strong alkaline irritants, but dermatitis in polyurethane foam production is rare. Tolylene diisocyanate is irritating to the eyes and respiratory tract, and can produce allergic bronchial asthma. Cutaneous spills of exothermally reacting polyurethane foam can produce burns.

Potential Occupational Exposures

Adhesive makers
Aircraft part makers
Boat makers
Crash pad fillers
Cushion makers
Float makers
Freezer makers
Life preserver makers
Printing roll makers
Refrigerator makers

(5) *Epoxy Resins*

Both epichlorohydrin and bisphenol, used to make the epoxy resins, can produce dermatitis. The liquid epoxy resins are moderate irritants and sensitizers. The solid epoxy resins are less likely to affect the skin, but their solvents, such as ketones, esters and ethers, frequently produce excessive drying, scaling, and fissuring of the skin. The resins may also contain plasticizers such as dibutyl phthalate and tricresyl phosphate, and reactive diluents (various glycidyl ethers), all of which can produce contact dermatitis. The most frequent dermatitis-producing components are the aliphatic polyamine curing agents; for example, diethylenetriamine and triethylenetetramine. These are highly alkaline, primary irritants and sensitizers. Aromatic amine curing agents such as metaphenylenediamine, and anhydride curing agents such as phthalic anhydride, can also produce contact dermatitis. Glass fiber used in making laminates frequently produces a mechanical irritation of the skin accompanied by considerable pruritus. Cured epoxy resins, especially if heat-cured, are usually inert, but subsequent tooling operations may be associated with dermatitis from glass fiber laminate particulates or possibly from resin-decomposition products.

Inhalation of the vapors of aliphatic polyamines can produce asthma-like symptoms and urticaria. The amines and their vapors are irritating to the conjunctiva.

Potential Occupational Exposures

- Adhesive makers
- Aircraft panel makers
- Appliance sprayers
- Automobile body repairmen
- Automobile prototype makers
- Body solder makers
- Brick masons
- Cement patchers
- Electric equipment makers
- Electricians
- Electron microscopists
- Gluers
- Highway maintenance workers
- Laminators
- Paint makers
- Paint sprayers
- Pattern makers
- Tank coaters
- Tile setters
- Tool and die makers

(6) *Phenolic Resins*

phenol-formaldehyde resins, cashew-nut-shell-liquid formaldehyde resins

Contact dermatitis can result from any of the phenolic components such as phenol, cresol, resorcinol, and cashew nut shell liquid; aldehyde components such as formaldehyde, paraformaldehyde, and furfural; or catalysts such as ammonia and hexamethylenetetramine. The most frequent offenders are formaldehyde and hexamethylenetetramine, which are irritants as well as

sensitizers. Mineral oil, which surrounds the resin in the curing ovens, can produce folliculitis. Irritation of the eyes and upper respiratory tract are occasionally produced by formaldehyde vapors from the various operations associated with mixing, extrusion, molding, laminating, and casting.

Potential Occupational Exposures

Abrasive wheel makers
Brake lining makers
Cabinet makers
Decorative laminators
Electric circuit printers
Electric component makers
Foundry workers
Glass wool insulation makers
Headphone makers
Lacquer makers
Luggage makers
Missile blast tube makers
Nose cone makers
Particle board makers
Plywood makers
Resin extrusion workers
Sandpaper makers
Shell molders
Sports equipment makers
Television cabinet makers
Toilet seat makers
Toy makers
Varnish makers

(7) *Polyester Resins*

Phthalic or maleic anhydride used in the manufacture of polyester monomer can produce dermatitis. The polyester monomer, modifiers such as styrene and methyl methacrylate, accelerators such as cobalt naphthenate and dimethyl aniline, catalysts such as organic peroxides, and plasticizers such as tricresyl phosphate and dibutyl phthalate can also produce contact dermatitis. The glass fiber used for lamination produces a pruritic, mechanical dermatitis.

Symptoms of headache, nausea, vomiting, and anorexia from styrene inhalation are occasionally encountered. Dimethyl aniline is a central nervous system depressant and may be absorbed through the skin as well as by inhalation.

Potential Occupational Exposures

Adhesive makers
Artificial limb makers
Automobile body repairmen
Automobile glass fiber body makers
Boat makers
Body solder makers
Chair makers
Electric equipment makers
Fishing rod makers
Food wrapper makers
Geodesic dome makers
Glass fiber sheeting makers
Ignition part makers
Lampshade makers
Laundry tub makers
Luggage makers
Pipe makers
Swimming pool makers
Translucent panel makers

Thermoplastics

(8) *Acrylic Resins*

The most widely used monomer, methyl methacrylate, is a sensitizer. The catalyst benzoyl peroxide can also produce dermatitis. Inhalation of methyl methacrylate vapor can produce irritability, headache, anorexia, somnolence, and hypotension. Solvents used to make solution polymers can produce a dry, scaly, and fissured dermatitis.

Potential Occupational Exposures

- Acrylic emulsion makers
- Acrylic molding bead makers
- Acrylic resin casters
- Acrylic solution polymer makers
- Dental technicians

(9) *Cellulosics*

cellulose nitrate, cellulose acetate, cellulose acetate butyrate, and cellulose propionate

Dermatitis occurs from mineral acids and alkalis used to treat the cellulose pulp. Acetic acid used in the production of cellulose acetate has caused mucosal irritation and dental erosion. Dermatitis may occur from plasticizers such as tricresyl phosphate, dibutyl phthalate, and dimethyl phthalate, and from solvents such as alcohol, acetone, ethyl acetate, and amyl acetate in the production of celluloid or pyroxylin from scraps of cellulose nitrate or cellulose.

Potential Occupational Exposures

- Cellulose derivative makers
- Cellulose plastic compounders
- Synthetic fiber makers

(10) *Fluorocarbons*

polytetrafluoroethylene

Inhalation of freshly generated polytetrafluoroethylene (Teflon) dust, especially when heated, can produce a self-limited condition known as polymer fume fever, which is manifested by chills, fever, and aches in muscles and joints. Inhalation of thermal decomposition products of polytetrafluoroethylene can also irritate the respiratory tract and produce pulmonary edema.

Potential Occupational Exposures

- Bearing makers
- Electric insulation makers
- Gasket makers
- Machinists
- Pump diaphragm makers
- Tubing makers

(11) *Nylons*

polyamides

No harmful effects have been reported in manufacturing or processing.

(12) *Polyethylenes*

Dermatitis is rare but has occurred from contact with the resin before it has completely polymerized.

Potential Occupational Exposure

Ethylene polymer makers

(13) *Polystyrenes*

The styrene monomer is a skin irritant and produces systemic symptoms of headache, nausea, vomiting, and anorexia. Organic peroxide catalysts and polymerization solvents can produce contact dermatitis. Central nervous system damage resulting in dizziness, staggering gait, and death has occurred from inhalation of methyl chloride, which may be released in the fabrication of polystyrene foam.

Potential Occupational Exposures

Polystyrene foam fabricators

Styrene polymer makers

(14) *Vinyl Plastics*

polyvinyl chloride, polyvinyl acetate, polyvinyl alcohol

Dermatitis is rarely encountered although the monomers are irritants. Plasticizers and stabilizers occasionally cause contact dermatitis.

Potential Occupational Exposures

Leather makers, artificial

Vinyl polymer makers

References

ANON.: Methylchloride intoxication. *Pub. Health Rep.* 74: 683, 1959.

BARNES, J. M. AND STONER, H. B.: Toxic properties of some dialkyl and trialkyl tin salts. *Brit. J. Indust. Med.* 15: 15, 1958.

BIRMINGHAM, D. J.: Clinical observations on the cutaneous effects associated with curing epoxy resins. *A.M.A. Arch. Indust. Health* 19: 365, 1959.

BOURNE, H. G., JR. AND SEFERIAN, S.: Formaldehyde in wrinkle-proof apparel produces tears for milady. *Indust. Med. & Surg.* 28: 232, 1959.

BOURNE, L. B.; MILNER, F. J. M., AND ALBERMAN, K. B.: Health problems of epoxy resins and amine-curing agents. *Brit. J. Indust. Med.* 16: 81, 1959.

BOURNE, L. B. AND MILNER, F. J. M.: Polyester resin hazards. *Brit. J. Indust. Med.* 20: 100, 1963.

DERNEHL, C. U.: Clinical experiences with exposures to ethylene amines. *Indust. Med.* 20: 541, 1951.

HARRIS, D. K.: Some hazards in the manufacture and use of plastics. ***Brit. J. Indust. Med.*** 16: 221, 1959.

HARRIS, D. K.: Polytetrafluoroethylene (Fluon, Teflon, PTFE). 15 references. ***Lancet*** 1: 587, 1962.

HINE, C. H.; KODAMA, J. K.; ANDERSON, H. H.; SIMONSON, D. W., AND WELLINGTON, J. S.: The toxicology of epoxy resins. *A.M.A. Arch. Indust. Health* 17: 129, 1958.

HODGSON, G.: The effects on the skin of some new adhesives and laminating materials. *Brit. J. Dermat.* 72: 95, 1960.

HOVDING, G.: Contact eczema due to formaldehyde in resin finished textiles. *Acta dermat.-venereol.* 41: 194, 1961.

JOYNER, R. E. AND PEGUES, W. L.: A health hazard associated with epoxy resin-concrete dust. *J. Occup. Med.* 3: 211, 1961.

KEY, M. M.: Some unusual allergic reactions in industry. *Arch. Dermat.* 83: 3, 1961.

KODAMA, J. K.; GUZMAN, R. J.; DUNLAP, M. K.; LOQUVAM, G. S.; LIMA, R., AND HINE, C. H.: Some effects of epoxy compounds on the blood. *Arch. Environ. Health* 2: 50, 1961.

LEA, W. A., JR.; BLOCK, W. D., AND CORNISH, H. H.: The irritating and sensitizing capacity of epoxy resins. *A.M.A. Arch. Dermat.* 78: 304, 1958.

LYLE, W. H.: Lesions of the skin in process workers caused by contact with butyl tin compounds. *Brit. J. Indust. Med.* 15: 193, 1958.

MALTEN, K. E.: *Occupational eczema in working with synthetic substances, in particular unsaturated polyester resins and ethoxyline resins.* Thesis, University of Amsterdam, 1956. 140 pages. (Abst., *Excerpta Med.*, sec. 13, vol. 10, p. 321, 1956)

MALTEN, K. E. AND ZIELHUIS, R. L.: *Plastics, their Toxicological and Dermatological Industrial Hazards.* Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1963.

MORRIS, G. E.: Condensation plastics, their dermatological and chemical aspects. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 5: 37, 1952.

MORRIS, G. E.: Vinyl plastics, their dermatological and chemical aspects. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 535, 1953.

MORRIS, G. E.: Epoxy resins, their uses and chemical and dermatological aspects. *A.M.A. Arch. Dermat.* 76: 757, 1957.

MORRIS, G. E.: Allergic rhinitis acquired during the processing of epoxy resins. *Ann. Allergy* 17: 74, 1959.

MUNN, A.: Experiences with diisocyanates. *Trans. Assoc. Indust. Med. Officers* 9: 134, 1960.

SCHWARTZ, L.; TULIPAN, L., AND BIRMINGHAM, D. J.: *Occupational Diseases of the Skin.* 3rd ed. Lea & Febiger, Philadelphia, 1957.

STEPHENSON, R. W. AND FOSDICK, L. B.: Hazards in the use of isopolyesters as maintenance coatings. *Am. Indust. Hyg. Assoc. J.* 21: 522, 1960.

TUBICH, G. E.; DAVIS, I. H., AND BLOOMFIELD, B. D.: Occupational health studies of the shell molding process. In *Transactions, 21st Annual meeting, American Conference of Governmental Industrial Hygienists*, 1959. Sec.-Treas., 1014 Broadway, Cincinnati 2, Ohio.

VARIOUS AUTHORS: *Modern Plastics, Encyclopedia Issue for 1964.* This is vol. 41, no. 1A of *Modern Plastics.* Plastics Catalogue Corp., Bristol, Conn., 1963.

WILSON, R. H. AND M'CORMICK, W. E.: Plastics, the toxicology of synthetic resins. *A.M.A. Arch. Indust. Health* 21: 536, 1960.

ZAPP, J. A., JR.: Toxic and health effects of plastics and resins. *Arch. Environ. Health* 4: 335, 1962.

ZIELHUIS, R. L.: Systemic toxicity from exposure to epoxy resins, hardeners, and styrene. *J. Occup. Med.* 3: 25, 1961.

• section IX

PHYSICAL HAZARDS

MARCUS M. KEY, M.D.; THOMAS H. MILBY, M.D.; DUNCAN A. HOLADAY, M.A., AND ALEXANDER COHEN, PH. D.

The hazards considered are those associated with exposure to the following agents:

(1) Ultraviolet, Infrared, and Microwave Radiation
(2) Ionizing Radiation
(3) Abnormal Air Pressure
(4) Abnormal Temperature
(5) Defective Illumination
(6) Noise and Vibration

Some of the hazards are encountered only in specific occupational situations, while others may be present in numerous working environments.

The physical agents discussed usually produce tissue injury by imparting energy in a harmful form to the tissue. This energy may be great and applied to the whole surface of the body such as in the instance of increased air pressure. On the other hand the energy may be small but applied at the cellular level such as noise or at the intracellular level as is the case with ionizing radiation.

Although a thorough investigation of the effect on man of devices employing the laser (light amplification by stimulated emission of radiation) has not been reported, the subject is of sufficient occupational health importance to merit brief mention. With the use of electric energy, the laser creates a high light intensity having acute biologic significance when the direct or optically reflected rays impinge on biologic tissue. Much work is being done on the development of devices, or in attempts to improve their efficiency and mode of operation, in such diverse fields as tracking and ranging (radar), communication, high temperature research, micromeasurement, microsurgery, biologic tissue removal or stimulation, high resolution spectroscopy, and microwelding. Sufficient experimental work on animals has been performed to indicate that persons working with laser light sources should be cognizant of the potentially hazardous nature of their occupation.

References

SOLON, L. R.; ARONSON, R., AND GOULD, G.: Physiological implications of laser beams. *Science* 134: 1506, 1961.

ZARET, M. M.; BREININ, G. M.; SCHMIDT, H.; RIPPS, H.; SIEGEL, I. M., AND SOLON, L. R.: Ocular lesions produced by an optical maser (laser). *Science* 134: 1525, 1961.

(1) Ultraviolet, Infrared, and Microwave Radiation

The continuum of energy extending from the long wave lengths of radio to the short wave lengths of cosmic radiation has been designated the electromagnetic spectrum. In between are television, microwaves, infrared rays, visible light rays, ultraviolet rays, X-rays, and gamma rays. The visible or light waves vary in length from 4,000 to 7,600 Angstrom units, with the ultraviolet rays below the 4,000 Å lower limit and the infrared and microwaves, respectively, above the 7.600 Å upper limit. (10 Å equal 1 millimicron or one millionth of a millimeter.) Ultraviolet and infrared rays, and microwaves, are invisible radiation. Because of their physical characteristics, alpha particles, beta particles, protons, neutrons, and positrons are classified under corpuscular (or particulate) radiations rather than under the radiations of the electromagnetic wave type. An electromagnetic wave, as the name implies, involves both an electric field and a magnetic field, each being measurable. All electromagnetic oscillations in free space travel with the speed of light, and cease locally when the source is turned off or removed. The power density is inversely proportional to the square of the distance from the source.

Ultraviolet Radiation

The sunburn spectrum of sunlight is in the ultraviolet zone, 2,900–3,100 Angstrom units, with a peak at about 3,000. Repeated exposures of fair-skinned individuals to the sunburn spectrum results in actinic skin. This is manifested by a dry, brown, inelastic and wrinkled skin. On the face, there are telangiectases, and on the nape, the neck movements produce lines in an angular pattern. Sailor's, farmer's, or fisherman's skin are names given to actinic skin, which bespeak their occupational origin. Oil field, pipeline, and construction workers also develop this condition. Actinic skin is not harmful in itself, but is a warning to susceptible individuals who tan poorly that certain conditions may develop such as senile keratoses, squamous cell epitheliomas and basal cell epitheliomas.

The ultraviolet light generated during welding can cause keratoconjunctivitis and sunburn of the exposed skin. The welder's helper, whose protection is more likely to be deficient, more often suffers the damage. Some of the newer welding processes, such as inert-gas, metal-arc welding with consumable electrodes, produce high intensities of ultraviolet light, and consequently have caused a marked increase in frequency and severity of eye and skin burns.

Photosensitizing agents have action spectra which are frequently in the ultraviolet range. Many plants such as figs, limes, parsnips, and pink-rot celery carry photosensitizing chemicals. These are believed to be furocoumarins and psoralens. The signs upon contact are those of an exaggerated sunburn, and blisters are frequently present. The most important industrial photosensitizer is coal tar, with an action spectrum in the visible light range. The increased incidence of skin cancer in coal tar workers is not only due to carcinogens, but also to repeated bouts of photosensitization.

Ultraviolet light can also act in a nonspecific manner similar to trauma in producing lesions of herpes simplex and chronic discoid lupus erythematosus.

Incandescent and fluorescent lamps used for general lighting purposes emit little or no ultraviolet radiation and are generally considered harmless. A rare reaction to fluorescent lighting has been observed in individuals who are sensitive to visible light. This sensitivity is manifested by urticaria (urticaria solaris) or by erythema and edema (erythema solare perstans) of exposed areas.

Potential occupational exposures—Occupations potentially associated with ultraviolet radiation exposures include the following.

Aircraft workers
Barbers
Bath attendants
Brick masons
Burners, metal
Cattlemen
Construction workers
Cutters, metal
Drug makers
Electricians
Farmers
Fishermen
Food irradiators
Foundry workers
Furnace workers
Gardeners
Gas mantle makers
Glass blowers
Glass furnace workers
Hairdressers
Herders
Iron workers
Lifeguards
Lithographers
Metal casting inspectors
Miners, open pit
Nurses
Oil field workers
Pipeline workers
Plasma torch operators
Railroad track workers
Ranchers
Road workers
Seamen
Skimmers, glass
Steel mill workers
Stockmen
Stokers
Tobacco irradiators
Vitamin D preparation makers
Welders

Infrared Radiation

The action of infrared rays is thermal, and fortunately such action gives a warning in the skin. However, in the eye there is no warning, and damage

may be produced by amounts of energy which will not burn the skin. The classic eye lesion after many years of exposure is posterior cataract, sometimes called glassblower's cataract, but this has not been reported in the United States. Mild exposures to infrared can cause eye fatigue and headaches. On the skin, infrared radiation produces a thermal burn.

Both infrared and ultraviolet radiation are present in some industrial exposures—for example, oxacetylene and electric welding; cupola, open-hearth, and electric furnace operations and foundry pouring; and glass blowing.

Potential occupational exposures—Occupations potentially associated with infrared radiation exposures include the following.

- Bakers
- Blacksmiths
- Braziers
- Chemists
- Cloth inspectors
- Cooks
- Dryers, lacquer
- Electricians
- Firemen, stationary
- Foundry workers
- Furnace workers
- Gas mantle hardeners
- Glass blowers
- Glass furnace workers
- Heat treaters
- Iraser operators (infrared amplification by stimulated emission of radiation)
- Iron workers
- Kiln operators
- Motion picture machine operators
- Plasma torch operators
- Skimmers, glass
- Solderers
- Steel mill workers
- Stokers
- Welders

Microwave Radiation (radar)

Microwave radiation or microwave energy includes electromagnetic frequencies ranging from about 300 megacycles to more than 30,000 megacycles per second. This form of energy is normally propagated in the atmosphere from rotating antennas associated with search radar and from stationary types associated with tracking radar, radio relay links, and television transmitters. Industrial and medical apparatus utilizing the heating effect of this energy include radio frequency ovens and diathermy devices.

This energy, when propagated, is categorized into two discrete modes known as continuous wave (CW) and Pulsed. The CW mode is associated with communication transmitting devices such as radio relay and television. The Pulsed mode is associated with radar, and industrial and medical equipment. From a biologic standpoint, the Pulsed mode is considered to be the more significant, due primarily to the high power intensities and energy distribution.

Microwave energy, because it is frequently referred to as microwave radiation, is often confused with ionizing radiation. This is unfortunate since there are no important similarities between the two energies so far as biologic effects are concerned.

Microwave radiation is thought to be similar to infrared radiation in that it causes localized heating of the skin, but penetration is deeper. There is laboratory evidence based on direction of stationary pulsed beams at animals, that microwave radiation can cook underlying muscle, produce cataracts, and cause death by hyperthermia. There have been a few reports of harmful human effects such as transient superficial heating of the skin, heating of a steel fracture plate, and development of cataracts, but there have been no proved reports of more serious human injury or death from radar exposure.

The main reasons for the observed response in man include—

(1) The human body has an efficient heat regulating system capable of resisting microwave heat better than laboratory animals.

(2) The larger field intensities of microwave energy are associated with radar transmitters whose antennas are mounted on towers, either as stationary objects aimed at a point near the horizon or as rotating or rocking objects aimed both below and above the horizon. In the case of stationary antennas, the concentrated beam of energy cannot normally searchlight unprotected humans on the ground. In the case of rotating or rocking antennas, any searchlighting is of an intermittent nature, a situation which provides the worker an opportunity to lose heat during nonexposure periods.

(3) Work is generally performed in open areas making possible the loss of body heat to the cooler surrounding air.

(4) Existence of adequate health and safety programs to minimize the hazards of microwave radiation.

It is not to be inferred that the problems associated with microwave hazards require only minimum attention. Indeed, caution should be observed and specific controls adopted as dictated by the type of radar set used. The possibility of the introduction in the near future of more powerful radar units introduces additional concern. The lack of conclusive information on the existence or nonexistence of nonthermal effects might be accounted for by the absence of appropriate detection techniques.

There is no generally acceptable maximum safe exposure level, but an average power density of 0.01 watt per square centimeter for all frequencies has been suggested as an arbitrary maximum ambient power level. It should be borne in mind that high field strengths can be associated with low average power. Even though no field-strength dependent effects have been demonstrated, the possibility of such effects should not be overlooked, and caution should be exercised by those working in pulsed fields of high peak, but low average, power.

Potential occupational exposures—Occupations potentially associated with microwave radiation exposures include the following.

Air crewmen	Drug sterilizers
Chemists	Food sterilizers

Furniture veneering operators
Maser operators (microwave amplification by stimulated emission of radiation)
Microtherm operators
Microwave development workers
Microwave diathermy operators
Microwave testers
Missile launchers
Radar mechanics
Radar operators
Radio frequency oven maintenance workers
Radio frequency oven operators

References

BAER, R. L. (EDITOR): *Allergic Dermatoses Due to Physical Agents.* New York University Press, New York, 1956.

BARRON, C. I. AND BARAFF, A. A.: Medical considerations of exposure to microwaves (radar). *J. Am. Med. Assoc.* 168: 1194, 1958.

BERGTHOLDT, C. P. L.: Recent welding practices at naval facilities. *Arch. Environ. Health* 2: 257, 1961.

BIRMINGHAM, D. J.; KEY, M. M.; TUBICH, G. E., AND PERONE, V. B.: Phototoxic bullae among celery harvesters. *Arch. Dermat.* 83: 73, 1961.

BLUM, H. F.: Ultraviolet radiation and cancer. *In* Hollaender, A. (editor): *Radiation Biology.* Vol. 2. McGraw-Hill Book Co., New York, 1955.

COMBES, F. C.: *Coal Tar and Cutaneous Carcinogenesis in Industry.* Charles C. Thomas, Springfield, Ill., 1954.

DEICHMANN, W. B. AND STEPHENS, F. H., JR.: Microwave radiation of 10 microwatts per square centimeter, and factors that influence biological effects at various power densities. *Indust. Med. & Surg.* 30: 221, 1961.

FERRY, J. J.: Ultraviolet emission during inert-arc welding. *Am. Indust. Hyg. Assoc. Quart.* 15: 73, 1954.

GOLDMAN, D. E.: Short wave electromagnetic radiation as a hazard to personnel. Lecture and review series 60–6. Naval Medical Research Institute, Bethesda, Md., 1960.

GRIMM, R. C. AND KUSNETZ, H. L.: The plasma torch. *Arch. Environ. Health* 4: 295, 1962.

KLABER, R.: Phyto-photo-dermatitis. *Brit. J. Derm. & Syph.* 54: 193, 1942.

KNAUF, G. M.: Microwave exposure and missile propellants as occupational health problems. *Am. J. Pub. Health* 50: 364, 1960.

KNAUF, G. M.: It is a pleasure to acknowledge the comments on microwaves (and lasers) of Colonel Knauf, Deputy Director, Aerospace Medicine, Office of Manned Space Flight, National Aeronautics and Space Administration, Washington, D.C. Communication of October 4, 1962.

MCGRAE, J. D.; KIERLAND, R. R., AND PERRY, H. O.: Sunlight and skin. *Proc. Staff Meet., Mayo Clin.* 37: 389, 1962.

MEICHEN, F. W.: Modern welding and health hazards. *Trans. Assoc. Indust. Med. Officers* 10: 39, 1960.

MORGAN, W. E.: Microwave radiation hazards. *A.M.A. Arch. Indust. Health* 21: 570, 1960.

NATIONAL SAFETY COUNCIL: Radar hazards. Data sheet 481. The Council, Chicago, 1959.

QUAN, K. C.: Hazards of microwave radiations. A review. *Indust. Med. & Surg.* 29: 315, 1960.

REES, R. B. (EDITOR): *Dermatoses due to Environmental and Physical Factors.* Charles C. Thomas, Springfield, Ill., 1962.

VARIOUS AUTHORS: *Biological Effects of Microwave Radiation.* Proceedings of fourth annual (1960) tri-service conference. Vol. 1. Peyton, M. F. (editor). Plenum Press, New York, 1961.

(2) Ionizing Radiation

Man has always been exposed to ionizing radiation. Thus the nature of the hazard itself is not new. The change that has occurred in recent times is in the size of the hazard. Ionizing radiation refers to (1) electrically charged or neutral particles, or (2) electromagnetic radiation which will interact with gases, liquids, or solids to produce ions. A large number of such particles are known to nuclear physicists, most of which will not usually be important factors in occupational exposures to ionizing radiation.

Depending upon their physical characteristics, the numerous kinds of ionizing radiation may be classified either under the corpuscular (or particulate) radiations or the electromagnetic wave type of radiation. The first type contains particles such as alpha particles, beta particles, neutrons, and protons; these particles have mass and are electrically charged with the exception of the neutron, which carries no charge. The second, or electromagnetic wave type, has associated with it uncharged electromagnetic vibrations such as gamma rays and X-rays.

The several types of ionizing radiation determined by these different particles and rays vary in their penetrative powers, and also with regard to the number of ions left in their tracks as they move through tissue. Biologic effect varies, among other factors, with ion density; that is to say, with the number of ions produced per unit length of track.

Ionizing radiations are produced naturally by radioactive decay processes or artificially by such devices as high energy accelerators. A radioactive nucleus is one that spontaneously changes to a lower energy state, emitting particles and gamma rays in the process. The particles commonly emitted are alpha particles and beta particles. High energy accelerators can produce all of the above particles and, additionally, protons and X-rays.

Alpha Particles

An alpha particle is the nucleus of a helium atom. It is a heavy particle with a weight of four mass units, and since it consists of two protons, two neutrons, and no electrons, it carries two positive charges. Alpha particles interact readily with matter to produce ions and usually have energies of from 4 to 8 million electron volts (Mev). They will travel a few centimeters in air and up to 60 microns in tissue. Their high energy and short path mean that they produce a dense track of ionization along their path and thus can produce serious biologic damage in the tissues with which they interact. Alpha particles will not penetrate the cornified layer of the skin and thus are not an external hazard. However, if alpha-emitting elements are taken into

218-695 O-66-18

the body, they create serious exposure problems. Examples of such elements are radium, radiothorium, and polonium.

Beta Particles

A beta particle is either a negatively charged electron or a positively charged positron, created by nuclear processes in which a neutron changes to a proton and an electron, or a proton changes to a neutron and a positron. Beta particles produced by radioactive decay have energies ranging from essentially zero to a definite maximum which is characteristic for each element. One of the most energetic naturally produced beta particles (E_{max} = 3.1 Mev) occurs in the RaC to RaC″ transition. Artificial beta particles produced by accelerators such as Van de Graaff machines or betatrons may have energies up to 100 Mev.

Beta particles interact much less readily with matter than alpha particles and will travel up to a few centimeters in tissue or many meters in air. Exposure to external sources of beta particles is potentially hazardous, and beta-emitting elements which are taken into the body create internal hazards.

Protons

A proton is the nucleus of a hydrogen atom and is relatively heavy with a single positive charge. Protons are produced by high-energy accelerators usually with energies of a few Mev. They are quite effective in producing tissue ionization, and their path length is somewhat longer than the path of alpha particles of equivalent energy.

Gamma Rays and X-rays

Gamma rays and X-rays are electromagnetic radiations with similar properties. X-rays, in general, have longer wave lengths, lower frequencies and therefore lower energies, than gamma rays. Gamma rays are produced by nuclear processes, while X-rays result from the interaction of high-speed electrons with the atoms. Naturally produced gamma rays have energies ranging from a few kiloelectron volts (Kev) to a few Mev, while high-energy accelerators can produce gamma rays of a few hundred Mev. X-ray energies form a continuous spectrum from the very low energies (or frequencies) in the upper ultraviolet range up to the energies of the high speed electrons that are producing the X-rays, often about 100 Kev. Gamma rays and highly penetrating X-rays produce a low ion density in the matter with which they interact. They are primarily an external hazard, and their biologic effects are better known than those of any of the other ionizing radiations. Examples of gamma emitters used in industry are cobalt-60 and iridium-192. In addition to being generated by X-ray tubes, X-rays may be encountered during the manufacture and use of klystron tubes and electron microscopes.

Neutrons

Neutrons are uncharged nuclear particles, which, together with protons, compose the nuclei of all elements except hydrogen-1. They have approximately the same mass as a proton, but since they are electrically neutral they react with matter in a different manner. Neutrons lose energy by direct collisions with nuclei or by entering a nucleus and initiating a nuclear reaction. The biologic effects of neutrons are primarily caused by charged particles and secondarily emitted gamma rays produced by these collisions or reactions.

Neutrons are produced by nuclear reactors or from sources such as radium-beryllium or polonium-beryllium mixtures. They also are produced by high-energy accelerators such as cyclotrons, with energies ranging from thermal (0.03 electron volt) to several Mev. The relative biologic effectiveness of neutrons is dependent on the energies of the neutrons.

Biologic Effects

The fact that ionizing radiation can cause biologic damage has been well documented over a period of years, and limits for occupational exposure to external radiation and for concentrations of radioactive isotopes in air and in water have been recommended by the National Committee on Radiation Protection and published in National Bureau of Standards Handbooks 59 and 69. For external radiation, the data are based on records of human exposure. For internal exposure, sufficient records are available for radium only. The values for other bone-seeking elements have been assigned by comparison with radium and from the results of animal experiments. For most of the other isotopes the exposure limit values were calculated, using the concept that the dose to the critical organ should not exceed 100 millirems per week.

Methods of preventing over-exposure to either external or internal radiation have been developed and should be followed carefully to prevent damage to the workers. Pertinent material may be found in National Bureau of Standards handbooks listed in Handbook 69.

The biologic effects of ionizing radiation may be divided into the somatic and genetic, the somatic including such effects as acute and chronic radiodermatitis, acute and chronic radiation syndrome, skin cancer, leukemia, cataracts, sterility, and shortening of life span. The genetic effects resulting from occupational exposures are unknown. Moreover, a mutation produced by radiation is probably similar to one effected by a mutagenic chemical or to one occurring spontaneously.

The effects of occupational exposure to ionizing radiation are usually localized, with production of acute or chronic radiodermatitis. Generalized exposure to penetrating ionizing radiation, and the resulting acute radiation syndrome, are rare in industry and are usually associated with an accident. Chronic occupational exposure to low levels of ionizing radiation is also rare

but may produce leukopenia and anemia. Other rare effects from occupational exposure to ionizing radiation include cataracts among cyclotron operators, bone sarcoma among radium dial painters, and cancer of the lung among pitchblende miners.

A commonly recognized effect of ionizing radiation is acute or chronic radiodermatitis. Mild acute exposures are most common in the applications of radiotherapy, while chronic exposures are most common in industry. Acute radiodermatitis, regardless of the type of ionizing radiation involved, presents the same clinical appearance, which is graded into three degrees of damage to the skin and its appendages, thus:

1st degree: Erythema after several hours to several days, followed by hyperpigmentation and temporary alopecia.

2nd degree: Erythema and edema followed by vesicles or bullae formation, superficial ulceration, permanent alopecia, permanent loss of nails and glands (sweat glands may regenerate), and atrophic, telangiectatic scarring.

3rd degree: Erythema and deep edema, followed by necrosis and slough; healing is slow and difficult or never occurs, leaving an ulcer; underlying tissues such as cartilage and bone may be involved.

Chronic radiodermatitis results from third degree radiodermatitis or from repeated suberythemal exposures. The cutaneous effects of ionizing radiation, like those of ultraviolet radiation, are cumulative. Grossly, four clinical effects are seen in chronic radiodermatitis: atrophy, telangiectases, hypopigmentation, and hyperpigmentation. If the fingers are involved, and they frequently are, the skin markings of the finger tips may lose their ridges, but this may also be seen in workers handling heavy and rough metal parts, as well as in pottery workers. The finger nails may become fragile and show longitudinal striations. Eventually, there may develop ulcerations, senile keratoses, and squamous cell epitheliomata. Basal cell epitheliomata can also develop in areas of chronic radiodermatitis, though less commonly than the squamous cell type.

Accidental whole body radiation with doses greater than 100 roentgens usually results in the acute radiation syndrome. Initial symptoms are nausea, vomiting, weakness, and shock. Death during this stage has been called a central nervous system death. Following a latent period of two days to two weeks, later symptoms begin with malaise and fever. There are hemorrhagic lesions of the skin, and by the third week epilation occurs. Painful ulcerations occur in the mouth and throat, and simultaneous ulcerations in the gastrointestinal tract produce bloody diarrhea. By this time anemia, leukopenia, and thrombocytopenia appear. Death may result from the bloody diarrhea or from severe bone marrow depression.

Early Recognition of Exposure

Several tests for the early recognition of the effects of ionizing radiation have been described. These are based on certain hematologic findings, such as leukopenia, relative lymphocytosis, presence of abnormal monocytes, increased incidence if bilobed lymphocytes, and an increase of desoxyribonucleic acid in lymphocytes. While these tests may be helpful in certain instances, the findings are not specific and should not be accepted in themselves as evidence of exposure to ionizing radiation. Nor should they be accepted as substitutes for personal and environmental monitoring devices.

Industrial Uses

Each year additional industrial and medical applications of ionizing radiation are discovered. Industrial uses include the following.

Abrasion measurements
Atomic battery fuel
Automation
Blast furnace study
Blight control
Byproduct power
Catalytic and other application to solids
Chemical processing
Coke oven operation
Crank shaft inspection
Crop storage
Density gages, cigarette
Detection of gasket leaks and other defects
Detection of voids, and defects in welds, forgings, castings and other solids
Determination of corrosion in high pressure steam lines, gas mains and acid tanks
Determination of dustiness
Determination of effectiveness of cleansers and cleansing machines
Disinfestation of products
Distillation of sea water
Ethyl bromide production
Fire alarm making
Gelatin production
Graft polymerization of plastics
Hardening of plastics
Instrument calibration
Interface labelling of liquids flowing through a pipeline
Land propulsion
Leakage rate measurements
Leak detection in buried pipeline
Liquid level gages
Location of obstruction in pneumatic tubes
Location of oil holes and other outlets in metals and other dense products
Lubricating oil production
Luminous compound manufacture
Measurement and control of thickness in rolling steel, linoleum and other products
Oil well logging
Oil well stimulation
Ore assaying
Preservation of foods
Prevention of potato sprouting
Process heat
Regulation and measurement of flow of liquids and solids

Safety controls
Silicone rubber vulcanization
Snow depth measurements
Space heat
Static elimination
Sterilization of drugs
Sterilization of medical supplies
Sulfurimeters
Thickness gages in rolling steel and in the production of metals, rubber and tire fabric, plastics and adhesives, and paper
Vacuum gages

Potential Occupational Exposures

With the widespread use of radioactive isotopes in industry and the increasing use of X-ray sources, radiation exposures may occur in a wide variety of occupations. In addition to apparent sources such as thickness gages and radiographic equipment, incidental sources occur in industry; examples are klystron tubes and radar tube-testing operations. In general, any operation where a high voltage electron beam impinges on metal will incidentally create X-rays and should be shielded.

While users of Atomic Energy Commission-produced isotopes and source material are licensed by that agency, there are no such restrictions on cyclotron-produced materials or on natural elements such as radium, polonium, and radiothorium. Such materials, therefore, might be used by groups of widely varying competence. Several States require the registration of radiation sources, but some do not. It is pertinent to note in connection with the addition in 1959 of Section 274, *Cooperation with States,* to the Atomic Energy Act of 1954 that the Atomic Energy Commission is placing emphasis on the desirability of the States assuming some of the Commission's regulatory authority. Such authority, when accepted, would enable the State to control by-product material, source material, and special nuclear material in quantities not sufficient to form a critical mass. Kentucky was the first State to accept authority, the agreement becoming effective March 26, 1962.

The following examples show the diversity of occupations potentially exposed to ionizing radiation.

Aircraft workers
Atomic energy plant workers
Biologists
Cathode ray tube makers
Ceramic workers
Chemists
Dental assistants
Dentists
Dermatologists
Drug makers
Drug sterilizers
Electron microscope makers
Electron microscopists
Electrostatic eliminator operators
Embalmers
Fire alarm makers
Food preservers
Food sterilizers
Gas mantle makers
Glass makers
High voltage television repairmen
High voltage vacuum tube makers
High voltage vacuum tube users
Industrial fluoroscope operators

Industrial radiographers
Inspectors using, and workers in proximity to, sealed gamma ray sources (cesium-137, cobalt-60 and iridium-192)
Klystron tube operators
Laboratory technicians
Liquid level gage operators
Luminous dial painters
Machinists, fabricated metal product
Military personnel
Nurses
Oil well loggers
Ore assayers
Pathologists
Petroleum refinery workers
Physicians
Physicists
Pipeline oil flow testers
Pipeline weld radiographers
Plasma torch operators
Plastic technicians
Pressman, printing
Prospectors
Radar tube makers
Radiologists
Radium laboratory workers
Radium refinery workers
Research workers
Roentgenologists
Roentgen tube makers
Shoe fitters
Television tube makers
Thickness gage operators
Thorium-aluminum alloy workers
Thorium-magnesium alloy workers
Thorium ore producers
Tile glazers
Uranium dye workers
Uranium mill workers
Uranium miners
Veterinarians
X-ray aides
X-ray diffraction apparatus operators
X-ray technicians

References

ABRAHAMS, A. P.; CARMICHAEL, P., AND KLEINFELD, M.: A study of occupational radiation exposure in New York State. *Am. Indust. Hyg. Assoc. J.* 22: 124, 1961.

ALEXANDER, P.: *Atomic Radiation and Life.* Pelican Books, A399. Penguin Books, Baltimore, 1957.

AMERICAN PUBLIC HEALTH ASSOCIATION: *Public Exposure to Ionizing Radiations.* The Association, New York, 1958.

ATOMIC ENERGY COMMISSION: *Annual Report to Congress for 1959.* U.S. Government Printing Office, Washington, D.C., 1960. Appendix 9, Amendments to the Atomic Energy Act: An act to amend the Atomic Energy Act of 1954, as amended, with respect to cooperation with States.

BACH, S. A.: Communication of October 9, 1962 from Commanding Officer, U.S. Army Medical Research Laboratory, Fort Knox, Kentucky.

BAKER, W. H.; BULKLEY, J. B.; DUDLEY, R. A.; EVANS, R. D.; MC CLUSKEY, H. B.; REEVES, J. D., JR.; RYDER, R. H.; SALTER, L. P., AND SHANAHAN, M. M.: Observations on the late effects of internally deposited mixtures of mesothorium and radium in twelve dial painters. *New Eng. J. Med.* 265: 1023, 1961.

BEERBOWER, A.: Radioisotopes. *In* Kirk, R. E. and Othmer, D. F. (editors): *Encyclopedia of Chemical Technology.* Supp. 2 (Standen, A., editor). Interscience Publishers, New York, 1960. Use of radioisotopes in industrial processes.

BEHRENS, C. F.: *Atomic Medicine.* 3rd ed. Williams & Wilkins Co., Baltimore, 1959.

BROWNING, E.: *Harmful Effects of Ionising Radiations.* Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1959.

DUNHAM, C. L.: Health hazards from ionizing radiation. *Arch. Environ. Health* 2: 144, 1961.

ELLINGER, F.: *Medical Radiation Biology.* Charles C. Thomas, Springfield, Ill., 1957.

HUEPER, W. C.: Carcinogens in the human environment. *Arch. Path.* 71: 237, 1961.

HUTCHINSON, F.: Molecular basis for action of ionizing radiations. *Science* 134: 533, 1961.

JOINT COMMITTEE REPORT, INDUSTRIAL MEDICAL ASSOCIATION: Diagnosis and compensation of radiation injury. Statement on the diagnosis and compensation of harmful effects arising as a result of work involving exposure to ionizing radiation. *J. Occup. Med.* 2: 503, 1960.

MC ALLISTER, R. G.: Potential exposures from X-ray and sealed radioisotopes. *Arch. Environ. Health* 4: 265, 1962.

MC KEE, G. M. AND CIPOLLARO, A. C.: *X-rays and Radium in the Treatment of Diseases of the Skin.* Lea & Febiger, Philadelphia, 1946.

MEDICAL RESEARCH COUNCIL: The hazards to man of nuclear and allied radiations. Presented to Parliament, June 1956. Cmd. 9780. Her Majesty's Stationery Office, London, no year. 2nd report. Cmd. 1225, 1960.

MOELLER, D. W.; TERRILL, J. G., JR., AND INGRAHAM, S. C., II: Radiation exposure in the United States. *Pub. Health Rep.* 68: 57, 1953.

MORGAN, G. W.: Current status of Atomic Energy Commission-State cooperative program. *Am. J. Pub. Health* 53: 878, 1963.

NATIONAL COMMITTEE ON RADIATION PROTECTION: Maximum permissible body burdens and maximum permissible concentrations of radionuclides in air and in water for occupational exposure. National Bureau of Standards Handbook 69. Issued June 5, 1959. U.S. Government Printing Office, Washington, D.C., 1959. Carries a list of handbooks of the Bureau.

NATIONAL COMMITTEE ON RADIATION PROTECTION: Permissible dose from external sources of ionizing radiation. National Bureau of Standards Handbook 59. Issued Sept. 24, 1954. Addendum dated April 15, 1958. U.S. Government Printing Office, Washington, D.C., 1955.

NEWELL, R. R.: Radiation hazard in industry. *J. Am. Med. Assoc.* 175: 149, 1961.

NICKSON, J. J. AND BANE, H. N.: Physiological effects of radiation. *In* Blatz, H. (editor): *Radiation Hygiene Handbook.* McGraw-Hill Book Co., New York, 1959.

NORWOOD, W. D.: Skin hazards from radiation in atomic industry, medicine and defense. *In* Schwartz, L.; Tulipan, L., and Birmingham, D. J.: *Occupational Diseases of the Skin.* 3rd ed. Lea & Febiger, Philadelphia, 1957.

THOMA, G. E., JR. AND WALD, N.: The diagnosis and management of accidental radiation injury. *J. Occup. Med.* 1: 421, 1959.

VARIOUS AUTHORS: Nine articles on ionizing radiation. *Sc. American* 201: 74, (Sept.) 1959.

WARREN, S.: Radiation protection. 44 references. *New Eng. J. Med.* 264: 705, 1961.

(3) Abnormal Air Pressure

Abnormal air pressure may be considered to be any pressure either above or below normal sea level pressure of 14.7 pounds per square inch. The normal of 14.7 pounds is equivalent to 1 atmosphere. Potential occupational exposure to more than 1 atmosphere of compressed air is associated with those who work in caissons or in tunneling operations where a compressed air environment is utilized to exclude water and to aid in supporting the structure. Potential occupational exposure is also associated with those

who dive into the depths of the sea, whether they breathe from a self-contained underwater breathing apparatus (SCUBA) or from a hose bringing air from a surface compressor. Pressure below 1 atmosphere is a potential hazard of pilots and crews of high performance aircraft in flight.

Increased Air Pressure

It has long been recognized that exposure to a compressed air environment with subsequent return to normal ambient air pressure levels may result in the production of signs and symptoms of alarming severity.

An increasingly large number of physicians throughout the country are being faced with the problems of recognizing, preventing, and treating the many consequences of compressed air exposure. This fact is due largely to the activities of two groups; namely, the skin divers using SCUBA and the tunnel builders, who because of expanding urban development must burrow deeper and longer to complete the necessary sewer and aqueduct construction.

It is well known that excessively rapid decompression of divers and compressed air workers gives rise to the formation of nitrogen bubbles in the blood stream and body tissues, and that these bubbles, if they are of sufficient size and number and if they are formed in or carried to critical areas, may give rise to one or more of the many signs and symptoms of compressed air illness or decompression sickness.

While the hazards of rapid decompression are great, one must also be mindful of the untoward effects produced by exposure to compressed air itself.

The effects of compression, that is, the effects of increasing air pressure on the body, will be considered first, followed by a discussion of the effects of decompression or the effects of returning the body to normal or sea level air pressure.

Primary Pressure Phenomena

The body can be compressed to almost 18 atmospheres without demonstrable changes attributable to the compression itself provided that air has free access to all surfaces of the body including the sinuses and middle ear spaces. If, however, the pressure is not equally distributed over all body surfaces, a pressure difference between tissues and the ambient atmosphere of less than 1 pound per square inch will produce congestion, edema, hemorrhage, and pain in the tissues exposed.

The ears, sinuses, and teeth are common sites of injury resulting from *squeeze* due to increased air pressure. About 1.5 percent of exposed individuals are subject to dental pain or barodontalgia and another 1.5 percent are subject to sinus pain or barosinusitis. Dental pain suggests the presence of small gas bubbles in the pulp or in a part of the tooth where soft tissues can be squeezed. Sinus pain is probably due to occlusion of the sinus aperture by inflamed mucous membrane or lymphatic tissue thus preventing

the requisite air from entering or leaving the sinus to effect an equalization of internal and external pressures.

If the eustachian tube is blocked by lymphoid tissue or swollen mucous membrane, a pressure difference will be created between the middle ear and the external environment. As this pressure gradient increases and as the duration of exposure is prolonged, certain progressive changes occur in the ear drum and in the middle ear. These changes, referred to as barotitis, begin with congestion of the ear drum and progress through erythema, and contraction of the drum and hemorrhage into it. This may be followed by hemorrhage into the middle ear itself. However, properly treated by expectancy, this condition will resolve itself and no sequelae will occur.

The lungs themselves may be subject to squeeze if the chest is compressed to a volume smaller than the amount of residual air of the lung, normally the amount of air left in the lungs following forced expiration. *Lung squeeze* is occasionally seen in unprotected swimmers who dive by simply holding their breath. The effect of the squeeze is to force blood and tissue fluids into the respiratory passages and alveoli. Considerable lung damage may result.

Secondary Pressure Phenomena

Narcotic action of nitrogen—The phenomena previously described are effected primarily by the formation of a pressure gradient between a body cavity and the external environment. On an entirely different basis are those pressure phenomena associated with disturbances in gaseous equilibria. At 4 atmospheres of pressure or more, the gaseous nitrogen induces a narcotic action evidenced by decreased ability to work, mood changes, and frequently, a mild to marked euphoria. The responses are, in fact, similar to those associated with alcoholic intoxication. The exact cause of this cerebral disturbance is unknown. However, it may be noted that nitrogen is highly soluble in fat, the ratio of its solubility in fat to its solubility in water being about 5 to 1. According to the Meyer-Overton hypothesis, a gas having such a relatively high ratio may act as a narcotic.

Oxygen poisoning—Inhalation of oxygen when its partial pressure exceeds 2 atmospheres, or about 30 pounds per square inch (gage) may result in the production of the signs and symptoms of oxygen poisoning.

The mechanism of oxygen toxicity is not well understood. The signs and symptoms of this intoxication may include tingling of fingers and toes, visual disturbances, acoustic hallucinations, confusion, muscle twitching, especially about the face, nausea, and vertigo. The final result of such exposure may be the epileptiform convulsion, which ceases as soon as exposure to high oxygen partial pressures is terminated. This toxic action of oxygen is greatly enhanced by exercise or by the presence of moderate amounts of carbon dioxide.

At normal atmospheric pressures, pure oxygen will irritate the throat and nasal membranes after about 12 hours of breathing time but signs and symptoms of systemic oxygen poisoning do not occur.

It should be noted that the greatest hazard in oxygen administration in chambers is the danger of fire.

Effect of carbon dioxide—Carbon dioxide enhances the toxicity of oxygen and the narcotic effect of nitrogen, and in addition a higher incidence of bends has been reported in association with a rise in the CO_2 level. The concentration of CO_2 present in the breathing media in a compressed air environment should not exceed the equivalent of 0.2 percent of one atmosphere.

Effects of Decompression

During ascent from the depths or during decompression in a chamber, two major groups of problems are introduced. The first group is a result of the property which allows a gas to expand as pressure decreases. The second is due to the tendency for dissolved nitrogen to escape from solution in the form of bubbles as pressure decreases.

The most serious effects of decompression are produced by the expansion of air in the lungs. If air is taken into the lung at a depth of 125 feet (55 pounds per square inch, gage), it will increase in volume five times when decompression to atmospheric pressure occurs. If decompression is excessively rapid and sufficient air is not exhaled, some of the pulmonary alveoli will rupture with the formation of one or more of the following: mediastinal emphysema, pneumothorax, or air embolism. The most dangerous of these conditions is the air embolism which occurs when air, expanding in the lung, is forced into the pulmonary blood vessels and then into the left side of the heart and into the arterial circulation which may quickly carry the air bubbles to the brain and produce a cerebral air embolism, a condition which may be rapidly fatal if not treated promptly by recompression.

If a given exposure to high pressure has caused a sufficient amount of nitrogen to be dissolved in the blood and in the tissues, and if decompression occurs sufficiently fast, gas bubbles will be formed. These bubbles of liberated gas create a condition of circulatory impairment and local tissue destruction which in turn are responsible for the signs and symptoms of decompression sickness.

The amount of bubble formation that will occur upon decompression depends to a large extent upon the following three factors: (1) the amount of gas dissolved in the tissues, which in turn is dependent upon the degree and duration of exposure to pressure and upon the amount of body fat in which the gas can be dissolved; (2) conditions which alter blood flow, including age, temperature, exercise, fright, and post-alcoholic state, especially if these alteration in blood flow occur during or shortly after the decompres-

sion process; and (3) the rapidity of decompression from elevated air pressure to the ambient level.

Nitrogen Bubble Formation: Signs and Symptoms

Bends—A relatively common manifestation of compressed air illness is described by Behnke as being a dull, throbbing type of pain which is gradual in onset, progressive and shifting in character, and frequently felt in the joints or deep in the muscles and bones. When the symptoms of bends occur, they do so in the first 4 to 6 hours in 80 percent of the cases, while the remainder will occur within 24 hours. Contributing to variations in susceptibility are such factors as age, obesity, defects of the lungs, heart impairments, temporary ill health, and individual predisposition.

Chokes—This rather specific type of asphyxia occurs less frequently than bends and is thought to be due to the accumulation in the large veins, the right side of the heart, and the pulmonary vessels of quantities of gas eliminated from the arterial circulation and from the extravascular tissues. The earliest evidence of impending chokes is a sensation of substernal distress felt during deep inspiration, especially during inhalation of tobacco smoke, which elicits paroxysmal coughing (Behnke's Sign). These attacks of coughing may proceed to loss of consciousness with all of the signs and symptoms of a true shock-like syndrome.

Paralysis—The most serious complication of decompression sickness is paralysis. Spastic paraplegia or monoplegia involving the lower extremities may follow improper decompression resulting in the formation of bubbles in the blood vessels and tissues of the spinal cord. Immediate and prolonged recompression usually brings about rapid recovery even following paraplegia. Cerebral involvement is very rare.

Aseptic bone necrosis—One important sequela of compressed air exposure is the development of aseptic bone necrosis. This condition is thought to be caused by the occlusion of small arteries in the bone by bubbles of nitrogen followed by infarction of the involved area. The sites of predilection for the occurrence of occlusion and necrosis, as seen in this process, are the lower femoral diaphysis, the upper tibial diaphysis, and the head and neck of the humerus and the femur. These lesions are usually multiple and tend to be bilaterally symmetrical.

Aseptic bone necrosis is usually asymptomatic unless joint surfaces are involved, in which case pain may be a symptom. Complete collapse of the affected joint has been known to occur. Healing takes place through an osteocondensing process. This increase in density may appear on roentgenographic examination as a *snowcap* on the top of the articular surface.

It is thought that prolonged daily exposure for at least 8 months is necessary to produce this type of bone necrosis since it is seen in tunnel workers but seldom in divers.

It is probable that the occurrence of aseptic bone necrosis can be avoided if strict observance of recommended decompression schedules is adhered to.

Decreased Air Pressure

When referring to the symptom complex developing as a result of exposure to high altitudes, *dysbarism* is the preferred term. That of decompression sickness is perfectly acceptable and includes the entire symptom complex which may develop. However, as determined by common usage, this term more often refers to the syndrome that develops in deep sea divers upon their return to the surface following exposure to several atmospheres of pressure.

The signs and symptoms of dysbarism result from the expansion of the gases within the body cavities and from the formation of nitrogen bubbles in body tissues and fluids from gas which is normally in solution at sea level pressure. Upon traveling to high altitudes, the resultant reduction in barometric pressure allows the gases within the body to expand to a greater volume than occupied at sea level. One volume of gas at sea level becomes two volumes at 18,000 feet, three at 28,000 feet, four at 33,000 feet, and five at 38,000 feet. On descent the volume changes in reverse order.

The greatest hazard at altitude is lack of oxygen; however, bends, chokes, neurologic disorders, aeroembolism, aerodontalgia, aerotitis, and aerosinusitis may all be experienced by those exposed to an environment of significantly decreased air pressure, that is, one above 30,000 feet.

Dysbarism may be complicated by a type of neurogenic peripheral circulatory failure or primary decompression shock consisting of any or all of the following manifestations: intense pallor, profuse sweating, faintness and dizziness, nausea, vomiting, loss of consciousness. These symptoms are usually rapidly relieved by descent from altitude.

The syndrome of primary decompression shock is much more commonly encountered following exposure to altitude than to depth. Spinal cord involvement is rarely a sequela of dysbarism but is not uncommon in divers. Aseptic bone necrosis is essentially unknown in pilots; is rare in divers; but is common in caisson and pressurized tunnel workers.

Potential Occupational Exposures

Occupations potentially associated with abnormal air pressures include the following.

Increased Air Pressure

- Caisson workers
- Canal tunnelers
- Divers
- Drain tunnelers
- Mine tunnelers
- Railroad tunnelers
- Road tunnelers
- Sewer tunnelers

Decreased Air Pressure

- Air crewmen
- Airplane pilots

References

ARMSTRONG, H. G. (EDITOR): *Aerospace Medicine.* Williams and Wilkins Co., Baltimore, 1961.

BEHNKE, A. R.: Decompression sickness following exposure to high pressures. *In* Subcommittee on Decompression Sickness, National Research Council: *Decompression Sickness; Caisson Sickness, Diver's and Flier's Bends and Related Syndromes.* W. B. Saunders Co., Philadelphia, 1951.

BUREAU OF MEDICINE AND SURGERY, DEPARTMENT OF THE NAVY: Aviation medicine practice. NAVPERS 10839-A. U.S. Government Printing Office, Washington, D.C., 1955.

BUREAU OF MEDICINE AND SURGERY, DEPARTMENT OF THE NAVY: Submarine medicine practice. NAVMED-P 5054. U.S. Government Printing Office, Washington, D.C., 1956.

DEPARTMENT OF THE AIR FORCE: Flight surgeon's manual. Air Force Manual No. 160-5. The Department, Washington, D.C., 1954.

DEWEY, A. W., JR.: Decompression sickness, an emerging recreational hazard; a discussion, with an illustrative case history of an increasingly common, but not yet widely understood, sports injury. *New Eng. J. Med.* 267: 759 and 812, 1962. 37 references.

DUFFNER, G. J.: Medical problems involved in underwater compression and decompression. *Ciba Clin. Symposia* 10: 99, 1958.

GRIBBLE, M. DE G.: A comparison of the *high-altitude* and *high-pressure* syndromes of decompression sickness. *Brit. J. Indust. Med.* 17: 181, 1960.

MILES, S.: *Underwater Medicine.* J. B. Lippincott Co. Philadelphia, 1962.

POPPEL, M. H. AND ROBINSON, W. T.: The roentgen manifestations of caisson disease. *Am. J. Roentgenol.* 76: 74, 1956.

RÓZSAMEGYI, I.: Die Rolle der Disposition bei der Entstehung der Dekompressionskrankheit. *Arch. Gewerbepath.* 17: 347, 1959. (Abst., *Bull. Hyg.* 35: 105, 1960)

VAN DER AUE, O. E.; DUFFNER, G. J.; AND BEHNKE, A. R.: The treatment of decompression sickness: an analysis of one hundred and thirteen cases. *Indust. Hyg. & Toxicol.* 29: 359, 1947.

(4) Abnormal Temperature

Heat

There are four distinct factors which influence the interchange of heat between man and his environment. These factors are the following.

(1) Air temperature
(2) Air velocity
(3) Moisture content of the air
(4) Radiant temperature

The industrial heat problem is one in which a combination of these factors interacts to produce a working environment which may be uncomfortable or even hazardous to the worker concerned.

The fundamental thermodynamic processes involved in heat exchange between the body and its environment may be described by the basic equation of heat balance. This equation reads as follows.

$$M = \pm S - E \pm R \pm C \qquad (1)$$

where M = rate of metabolism; S = rate of storage, or change in body heat content; E = rate of heat loss through evaporation; R = rate of heat loss or gain by radiation; and C = rate of heat loss or gain through convection.

Under conditions of comfort, this balance can be expressed as shown in *2*.

$$M-(\pm S-E\pm R\pm C)=0 \qquad (2)$$

For purposes of temperature determination, the body can be divided into two regions, the deep region or the *core*, and the superficial region, which is made up largely of the skin and subcutaneous tissues.

The heat regulatory mechanisms of the body are directed at keeping the core temperature at a uniform level, while the temperature of the superficial tissues may vary within a relatively wide range according to the amount of heat received from the environment.

When heat loss fails to keep pace with heat gain, the core temperature begins to rise and certain physiologic mechanisms come into play in an attempt to increase heat loss from the body. First, there is a dilation of the blood vessels of the skin and subcutaneous tissues with a diversion of a large pait of cardiac output to these superficial regions. There is a concomitant increase in circulating blood volume brought about by contraction of the spleen and by dilution of the circulating blood with fluids drawn from other tissues. Cardiac output is also increased. It is probable that neither thyroid nor adrenal hormones play a part in increasing body heat loss.

In equation *1*, $M\pm R\pm C$ may be taken to indicate the total heat load imposed by the combined effects of metabolic and environmental heat. The evaporative capacity required to maintain body heat balance, $E_{req,}$ is then equal to $M\pm R\pm C$. If E_{req} exceeds the maximum exaporative capacity of the body, E_{max}, a condition of stress will develop. Many heat stress indexes have been constructed, but each contains inherent limitations because of the conditions, usually experimental, under which they have been developed.

Acclimatization is essential if man is to withstand prolonged increased heat loads. This process of adaptation is characterized by the worker's ability to perform with less increase in core temperature and by the secretion of decreased amounts of perspiration. This perspiration is more dilute, that is, it contains a lower concentration of sodium chloride than the perspiration of a nonacclimatized individual. Thus essential salt is thereby conserved. Acclimatization to heat occurs in from 1 to 2 weeks.

In general, industrial heat exposures may be classified as either hot-dry or as warm-moist. Hot-dry environments are found in industrial situations in which ambient and radiant temperatures are elevated but in which moisture content of the air is not excessive. The difficulties in hot-dry situations arise when the body absorbs more heat by radiation or convection or both than it can lose through evaporation of perspiration, that is, $R\pm C$ exceeds E_{max}. Warm-moist working environments may be encountered in occupations where large amounts of moisture are released from the industrial processes involved, but where ambient and radiant temperatures are only moderate. Here, the heat load from radiation or convection, or both, is not great, but the high moisture content of the air inhibits heat loss from the body through the

perspiration mechanism. In such an environment, E_{max} is diminished because of the moisture content of the surrounding air.

Psychologic Reactions

Psychologic reactions to prolonged exposure to excessive heat include increased irritability, lassitude, decrease in morale, increased anxiety, and inability to concentrate. The results are mirrored by a general decrease in the efficiency of production and in the quality of the finished product.

Physical Reactions

Physical reactions to prolonged exposure to excessive heat include heat cramps, heat exhaustion, and heat stroke.

Heat cramps—These cramps may occur after prolonged exposure to heat attended by profuse perspiration with resultant loss of large amounts of salt. The signs and symptoms of heat cramps consist of spasm and pain in the muscles of the abdomen and extremities. Albuminuria may be a transient finding.

Heat exhaustion—This condition may result from physical exertion in a hot environment when vasomotor control and cardiac output are inadequate to meet the increased demand placed upon them by peripheral vasodilatation. Signs and symptoms of heat exhaustion may include lassitude, dizziness, syncope, profuse sweating, and cool moist skin. There is usually no attendant hyperthermia.

Heat stroke (*sunstroke*)—This is a much more serious condition than heat cramps or heat exhaustion. An important predisposing factor is excessive physical exertion. Heat pyrexia is closely related to humidity as well as to temperature and is less common in areas where relative humidity is very low. Signs and symptoms may include dizziness, nausea, severe headache, hot dry skin, very high body temperature (often greater than 108° F.), coma, and death.

Potential Occupational Exposures

An attempt to compile a list of occupations potentially associated with hot environments would probably result in a list of *hot jobs* having little usefulness. Climatic conditions and plant layout are probably factors of greater significance than heat per se; thus heat stress may be involved in many operations not categorized as hot jobs. Of importance, rather, is the recognition of the signs and symptoms previously referred to, which, in turn, will suggest appropriate treatment and environmental control. Finally, in the evaluation of an environmental heat problem, it is helpful to remember that some physiologic responses may be associated with ambient temperatures at levels much lower than generally believed, and that the age of the worker may be a factor of some importance in the physiologic reactions produced by the job.

Cold

As the environmental temperature is reduced below normal body temperature, equation *1* may be rearranged, as shown in *3*, to express the change in heat equilibrium that may occur.

$$S=M-E-R-C \qquad (3)$$

The change in body heat content is thus equal to the heat gain through metabolism minus the loss of heat through evaporation, radiation, and convection. It is evident that the body will tend to lose heat to the environment if heat loss by evaporation, radiation, and convection exceeds body heat gain from metabolic processes. In general, cooling stress is proportional to the total gradient between the skin and the environmental temperature since this gradient determines the rate of heat loss from the body by radiation and convection. Loss of heat through the mechanism of the evaporation of perspiration is not significant at temperatures below normal body temperatures.

For the body to maintain thermal homeostasis in a cold environment, certain physiologic mechanisms come into play which tend to limit heat loss and increase heat production. The first mechanism is one of peripheral vasoconstriction, especially in the extremities, resulting in a marked drop in skin temperature. Body heat loss to the environment is thereby diminished.

When vasoconstriction is no longer adequate to maintain body heat balance, muscular hypertonus and shivering become important mechanisms for increasing body temperature by causing metabolic heat production to increase to several times the normal rate. Final breakdown of thermal control depends upon the degree of physical activity of the worker; amount of clothing worn; and the nature, intensity, and duration of exposure to the environment. In the event of such a breakdown, the following may occur.

(1) If activity is restricted, the extremities, notably toes and fingers, approach freezing temperatures most rapidly. A depression of general body temperature follows. (2) If the individual is physically active, cooling develops with fatigue, and as exhaustion approaches, the vasoconstrictor mechanism is overpowered and sudden vasodilatation occurs with resultant rapid loss of heat. Critical cooling then ensues.

Physical Reactions

In addition to the above-mentioned generalized physiologic reactions to cold stress, certain localized phenomena may occur. These include trench foot and frostbite.

Trench foot—If there is long continuous exposure to cold without freezing combined with persistent dampness or actual immersion in water, a syndrome referred to as trench foot or immersion foot may be produced. This condition is due to persistent local tissue anoxia, combined with mild or severe cold, with resultant injury to the capillary wall. Edema, tingling, itching, and

218-695 O-66-19

severe pain occur and may be followed by blistering, superficial skin necrosis and ulceration.

Frostbite—Frostbite, in contradistinction to trench foot, occurs when there is actual freezing of the tissues with the attendant mechanical disruption of cell structure. Frostbite results from exposure to cold air, usually at temperatures below 10° F. Theoretically, the freezing point of the skin is about 30° F., but, when dry and oily, the skin's property of supercooling without freezing affords considerable protection. Once started, however, freezing progresses rapidly. The tissues of the cheeks, nose, ears, and digits are the first to be injured. The first warning of frostbite is often a sharp, pricking sensation. However, cold itself produces numbness and anesthesia which may permit serious freezing to develop without the warning of acute discomfort. Injury produced by frostbite may range from simple redness of the skin with transient anesthesia and superficial bullae to persisting ischemia, thrombosis, deep cyanosis and gangrene.

Abnormal Responses to Cold

Certain vascular abnormalities may be either precipitated or aggravated by cold exposures. These include chilblain, pernio, Raynaud's disease, acrocyanosis, and thromboangiitis obliterans. Workers suffering from these ailments should take special precautions to protect against chilling.

Potential Occupational Exposures

Occupations potentially associated with cold environments include the following.

Arctic research technicians
Bridge builders
Bridge tenders
Construction workers
Divers
Dock workers
Dry ice workers
Farmers
Firemen
Fishermen
Harbor workers
Highway workers
Ice makers
Linemen
Liquefied gas workers
Loggers
Mail carriers
Packing-house workers
Pipeline workers
Policemen
Refrigeration workers
Road builders
Salvage workers
Shepherds
Surveyors
Tunnel traffic workers

References

General

WINSLOW, C.-E. A. AND HERRINGTON, L. P.: *Temperature and Human Life.* Princeton University Press, Princeton, N.J., 1949.

Heat

BELDING, H. S. AND HATCH, T. F.: Index for evaluating heat stress in terms of resulting physiological strains. *Heat. Pip. & Air Cond.* 27: 129, (Aug.) 1955.

BROUHA, L.: Heat and the older worker. *J. Am. Geriat. Soc.* 10: 35, 1962.

HAINES, G. F., JR. AND HATCH, T. F.: Industrial heat exposures; evaluation and control. *Air Cond. Heat & Vent.* 49: 94, (Nov.) 1952.

HATCH, T. F.: Heat control in the hot industries. *In* Patty, F. A. (editor): *Industrial Hygiene and Toxicology.* Vol. 1, 2nd ed. Interscience Publishers, New York, 1958.

MOSES, J. B.: The physiological effects of heat. *Indust. Med. & Surg.* 29: 20, 1960.

SCHERBERGER, R. F.: Evaluating hot environments. *Indust. Med. & Surg.* 29: 16, 1960.

Cold

DEPARTMENT OF THE AIR FORCE: Flight surgeon's manual. Air Force Manual No. 160–5. The Department, Washington, D.C., 1954.

SELLERS, E. A.: Cold and its influence on the worker. *J. Occup. Med.* 2: 115, 1960. The first of a series of 6 papers by different authors on work in cold environments.

WASHBURN, B.: Frostbite; what it is, how to prevent it, emergency treatment. *New Eng. J. Med.* 266: 974, 1962.

WILKINS, R. W.: Peripheral vascular disease due to exposure to cold. *In* Cecil, R. L. and Loeb, F. (editors): *A Textbook of Medicine.* 10th ed. W. B. Saunders Co., Philadelphia, 1959.

(5) Defective Illumination

The visibility of an object is determined by four factors. These are size, contrast, time of viewing, and brightness. As the size of the object being viewed increases, visibility increases and, up to a certain point, seeing becomes easier. Within limits, higher contrast between the object being viewed and its background means greater visibility of that object. Time of viewing is equally important in that lower levels of illumination require an increased amount of time of viewing to accomplish a visual task. Within certain limits, increased brightness will usually mean increased visibility.

In most tasks the factors of size and time of viewing are constant or hold little flexibility. Contrast, by the same token, may also be limited to a great extent by the nature of the task itself. Therefore, brightness is in general the most important controllable factor in the visibility of an object.

The effects of defective illumination are legion. A large share of all industrial accidents may be traced to this issue. Also, many specific as well as general effects may be seen in workers subjected to this hazard. Defective illumination may cause a feeling of general discomfort, muscular tension, eye fatigue, increased blinking of the eyes, a decrease in convergence reserve, a decrease in visual acuity, aggravation of eye defects, dizziness, headache, and even blindness. The worker may show the effects of this hazard in a more general, subtle way, such as a decrease in his efficiency and work output, increase in errors in his work, and a generally low morale.

We can all attest to the observation that our *spirits* or *emotions* tend to be low or high according to the brightness of our surroundings. Alertness, cheerfulness, and increased performance are all favored by the presence

of good illumination, while a general feeling of dullness and depression is frequently associated with the drab environment of a poorly lighted room.

Defective or unsuitable illumination will result when an abnormal quantity of illumination is used, either too low or too high. It may also be produced by glare, unsuitability of color of light source or surroundings, and shadows. Any one, or a combination of the factors producing defective illumination may be present in almost any working situation. The importance of each varies with the particular type of industry.

Low Illumination

Miners' nystagmus, evidenced by the rhythmic involuntary movement of the eyes, has been attributed to continued use of the eyes under conditions of very low illumination. Miners afflicted with this disease may become incapacitated for work. The disease is becoming less of a problem in Great Britain and continues to be rare among miners in the United States. The continued infrequent occurrence of the disease among American miners has been attributed to the level of illumination in the mines.

Data from Great Britain on the hazards of coal mining illustrate the part played by illumination. The gradual improvement of underground illumination was accompanied by a decrease in the number of coal miners certified as having the disease. Thus, during the years immediately prior to World War II, the annual certification rate was about 150 per 100,000 miners. In 1950 the corresponding rate was 34, and by 1960 it had decreased to 9. It would appear that, while discussions of miners' nystagmus frequently point to the psychologic factor as being responsible for the disease, illumination, regardless of its place in the chain of causation, cannot be ignored.

Miners' nystagmus may be seen in a latent, or subacute form, and in an acute form. Symptoms are first a marked loss of visual acuity, especially at night, oscillation of the eyes when fatigued, photophobia, and giddiness associated with bending. In the more acute forms, headache may be a complaint, the eye oscillations become more marked, and shock may develop.

Intense Illumination

A level of illumination in excess of the amount needed for good vision may produce a feeling of discomfort and eye fatigue. An intensely brilliant light source such as the sun, carbon arc, or welder's arc may effect temporary or permanent blind areas in the retina. This occurs when the retina is subjected to intense light without proper protection and is known as *eclipse blindness*. Possibly this is due to the retinal heating effect of intense visible light and of infrared rays. *Snow blindness*, which exhibits some of the symptoms of visual blindness, is principally characterized by a burning and scratching of the external surfaces of the eye resulting from exposure to the ultraviolet portions of the spectrum, which are absorbed in these tissues. In this regard, snow blindness is the equivalent of a sunburn of the eyeball.

Glare

Glare depends upon the general brightness of the field in which the glare source is seen. This is illustrated by the glare produced by a candle flame in a dark room, although the photometric brightness of the flame is not very great. The glare results because the eye is dark adapted to the decreased illumination of the whole field of view. There seems to be considerable individual variation with regard to susceptibility to glare. Prolonged exposure to an environment in which high contrasts of brightness produce glare may condition the observer so that he does not consider the glare to be as objectionable as before. Glare may produce a feeling of visual discomfort. If the glare is substantial or frequently induced it may effect tiredness, irritability, possibly headache, and a decrease in efficiency.

Glare may be produced directly by the light source itself, or indirectly by reflection of the light source from a shiny surface in the field of view such as shiny ceilings or walls, glass desk tops, polished metal surfaces, or glossy paper.

Color

The color of a light source may be an important cause of defective illumination; however, in most working conditions color control is not so important as the proper selection of the light intensity or the elimination of glare. The spectral quality of light, however, is of major importance in some jobs including those involving color discrimination or color matching.

The painting of machinery and surroundings with carefully chosen colors has been introduced into industry to improve seeing and provide cheerful, pleasant, and interesting work environments.

Shadows

Improper diffusion and distribution of light may cause shadows. This defect of illumination is an important cause of industrial accidents.

Artificial lighting in many modern factories and offices is accomplished by means of the fluorescent lamp. Special attention is necessary in planning a fluorescent lighting installation, especially on a moving production line or if revolving objects are to be in the field of view. This is important because the light from a fluorescent lamp varies rhythmically despite its apparent steadiness and may give rise to a stroboscopic effect, or an illusion of movement. Usually this effect can be eliminated by the proper grouping and installation of the fluorescent lamps.

Potential Occupational Exposures

Virtually all occupations offer potential exposure to the hazards of defective illumination. Some occupations, however, are especially subject to this hazard. Such occupations usually require close fine work and attention to detail for many hours a day; for example, engravers, draftsmen, jewelers, watchmakers, miners, mail sorters, and clerks.

References

AMERICAN STANDARDS ASSOCIATION: *American Standard Practice for Industrial Lighting.* The Association, New York, 1952. Final reprint by U.S. Department of Labor, Washington, D.C., 1955.

GUTH, S. K. AND LINDSAY, E. A.: Lighting for seeing. *In* Patty, F. A. (editor): *Industrial Hygiene and Toxicology.* Vol. 1, 2nd ed. Interscience Publishers, New York, 1958.

ILLUMINATING ENGINEERING SOCIETY: *IES Lighting Handbook. The Standard Lighting Guide.* 3rd ed. The Society, New York, 1959.

NATIONAL COAL BOARD: Medical service, annual report, 1960. The Board, London, no year. Presents annual data, 1955–1960, on nystagmus in Great Britain.

ROGAN, J. M.: *The hazards of coalmining. In* Davies, T. A. L.: *The Practice of Industrial Medicine.* 2nd ed. J. & A. Churchill, London, 1957. Presents data on nystagmus in Great Britain.

VARIOUS AUTHORS: Vision, lighting and colour. *Ann. Occup. Hyg.* 1: 208, 1959.

(6) Noise and Vibration

Advanced mechanization has created excessive noise and vibration conditions in many different occupational environments. While much is already known about the adverse effects of noise and vibration on man, more information will be required before specifications for tolerable noise or vibration conditions can be made on a valid basis. Presently, extensive efforts are being made to correlate various aspects of noise exposure with hearing loss in an attempt to verify proposed noise tolerance limits for protecting man's hearing. The potency of different vibration conditions for causing pain and discomfort to the individual has also been under study with regard to establishing safety limits for vibratory stimulation.

Basic Aspects of Sound and Noise

Airborne sound refers to the rapid pressure variations, that is, alternate increases and decreases in normal atmospheric pressure, caused by a vibrating object and also the resultant sensation when such pressure changes strike the ear. The rate of vibration of the object corresponds to the frequency of sound, which is expressed in cycles per second (cps).

The frequency range of audible sounds for healthy young ears is usually considered to extend from 20 to 20,000 cps although there is evidence to indicate that man's hearing extends beyond these limits. The simplest type of sound, called a pure tone, is described as having a single frequency. These sounds are produced by tuning forks or electric means. In contrast, music, speech, and noise, each containing a collection of different frequency sounds, are called complex sounds. The frequencies comprising speech are found principally between 250 and 3,000 cps. This is considered to be the most important range of frequencies since hearing loss for speech sounds would handicap the individual in most daily activities.

Sound pressure level (SPL) measurements are based upon the average (root mean square) amplitude of the pressure changes constituting the sound

stimulus and are directly related to the intensity or energy characteristics of the sound. Such measurements are specified on a decibel scale and defined by the formula:

$$\text{SPL in decibels (db)} = 20 \log_{10} P/P_o$$

where P is a measure of the pressure change corresponding to the sound under study, and P_o is a reference pressure. The reference value for SPL determinations is usually 0.0002 dyne/cm^2. This pressure corresponds to the weakest sound that the ear can hear under the most ideal listening conditions. SPL measurements must always state the reference value being used. Unless otherwise stipulated, all sound intensity values in this presentation refer to SPL values in db re 0.0002 dyne/cm^2. The accompanying table shows SPL values for some everyday sounds.

Noise is commonly defined as unwanted sound and can be classified into three basic types, namely, wide-band noise, narrow-band noise, and impulse noise. The spectrum of a wide-band noise shows that the acoustical energy is distributed over a large range of frequencies. In determining such spectra, the noise is usually divided into eight frequency bands, each one octave wide, and SPL measurements are made in each band. Examples of wide-band noise can be found in the weaving room of a textile mill and in jet aircraft operations.

SPL in decibels re 0.0002 dyne/cm^2 for everyday sounds

Threshold for pain	—140—	
	—130—	
Pneumatic chipper at 5 feet		Boiler shop (maximum level)
	—120—	
	—110—	
		Woodworking shop
	—100—	Weaving room
Subway train at 20 feet		
Train whistle at 500 feet	— 90—	Inside motor bus
	— 80—	
Heavy traffic at 25–50 feet		Office with tabulating machines
	— 70—	
		Average traffic
Conversational speech at 3 feet	— 60—	
	— 50—	Private business office
Light traffic at 100 feet		Average residence
	— 40—	
	— 30—	
	— 20—	
	— 10—	
Most sensitive hearing threshold	— 0—	

Narrow-band noises have most of their energy confined to a narrow range of frequencies and normally produce a definite pitch sensation. Accurate spectral determinations of narrow-band noises require SPL measurements in frequency bands which are smaller than an octave in width. The noise caused by a circular saw, planer, or other power cutting tools are of the narrow-band type.

The impulse type of noise consists of transient pulses, occurring in repetitive or nonrepetitive fashion. Repetitive impulse noise is associated with the operation of a rivet gun or a pneumatic hammer. The impact of a drop hammer and the firing of a gun are examples of nonrepetitive impulse noise. The instrumentation, together with the procedures used to describe impulse noise, differs from that used for narrow or wide-band types of noise. Repetitive impulse noise which occurs at a rate exceeding 200 pulses per minute, however, can be analyzed in a manner similar to that used for wide- or narrow-band noise.

Effects of Noise on Hearing

Exposure to intense noise causes hearing losses which may be temporary, permanent, or a combination of the two. These impairments are reflected by elevated thresholds of audibility for different frequency sounds, the increase in decibels required to hear such sounds being used as a measure of the loss. Temporary hearing losses, also called auditory fatigue, represent threshold losses which are recoverable after a period of time away from the noise. Such losses may occur after only a few minutes of exposure to intense noise. With prolonged exposures (months or years) to the same noise, there may be only partial recovery of the threshold losses, the residual loss being indicative of a permanent hearing impairment.

Temporary hearing impairment—Extensive studies have been made of temporary hearing losses following various conditions of noise exposure. Such investigations have yielded the following observations:

(1) Typical industrial noise exposures produce the largest temporary hearing losses at 4,000 and 6,000 cps. The greatest portion of the loss occurs within the first two hours of exposure. Recovery from such losses is greatest within one or two hours after exposure.

(2) The amount of temporary hearing loss from a given amount of noise varies considerably from individual to individual. Indeed, the losses at a given frequency from noise intensities of 100 db or more may range from 0 to more than 30 db for the exposed group. On the other hand, the amount of threshold loss for the group varies according to a normal statistical distribution; that is, few persons show very large or very small shifts, and most of the losses cluster around an average midway between the largest and smallest threshold shift.

(3) Low frequency octave bands of noise, below 300 cps, require considerably more intensity than middle or high frequency octave bands of

noise to produce significant threshold losses. The lowest intensity level capable of producing a temporary threshold loss is 80 db.

(4) Considerably fewer temporary hearing losses result from intermittent than from continuous noise exposures, even though the total amount of noise exposure is the same in both instances.

(5) The amount of temporary loss and its frequency location vary with the amount and frequency location of a permanent loss. Generally, the amount of temporary loss that occurs at a given frequency becomes less as the amount of permanent loss increases for that frequency.

It is presently believed that there is a direct relationship between temporary hearing loss and permanent hearing loss. A noise that does not cause temporary hearing loss following a short term exposure, for example, is assumed to be incapable of producing a permanent hearing impairment. Moreover, the pattern of temporary hearing losses shown for various frequency sounds following exposure to a given noise is assumed to resemble the pattern that will occur if and when a permanent loss is produced by a long-term exposure to the same noise. Recent findings concerning the nature of permanent noise-induced hearing loss provide some support for these assumptions and are contained in the discussion below.

Permanent hearing loss—Exposure to intense noise is only one cause of permanent hearing damage. Other causes may be disease, mechanical injury, and use of drugs. The time and nature of onset of the loss, the pattern of hearing loss for different frequencies, the findings of an otologic examination and medical history are means of determining whether a case of permanent hearing damage is due to these latter factors. Once these causes have been excluded from the etiology of hearing damage, the losses attributable to the aging process (presbycusis) must be considered. Curves showing the normal deterioration in hearing with increasing age have been reported. Such curves have been used to separate the amount of hearing loss due to noise exposure from that due to the aging process.

Evidence for permanent hearing losses from occupational noise has come from two basic types of study. In one, the hearing of personnel who work under characteristically noisy conditions, for example, factory workers, has been compared with that of a similar age group of persons who work under typically quiet conditions, such as office workers. In the other, pre-employment tests of hearing sensitivity have been compared with follow-up hearing tests for worker groups exposed to various amounts of noise. Suitable corrections are made in the latter type of study to account for hearing loss due to age only. The findings from both types of study show that permanent threshold losses caused by noise initially appear in the region 3,000 to 6,000 cps and are most prominent at 4,000 cps. With continued exposure, the losses become greater and spread to frequencies above and below the 3,000 to 6,000 cps range until the hearing at most frequencies is affected.

Since early noise-induced losses almost always occur at frequencies above the speech range, substantial impairments in hearing can occur without the individual's being aware of such damage. Actually, impairments in the perception of speech do not become noticeable until losses for the speech frequencies are 20 db or more.

The losses in hearing due to exposure to a given occupational noise tend to reach a maximum at certain frequencies (for example, 4,000 cps) after about 10 years of exposure; further losses in hearing at these frequencies appear to be accounted for by the aging process. The hearing loss for these frequencies which results from a 10-year exposure to noise for a worker appears to correlate closely with the temporary hearing loss at the same frequencies following a new worker's first day of exposure to such noise. The latter finding has particular significance for the assumed relationship between temporary and permanent hearing loss which was cited earlier. Indeed, it suggests the possibility of using temporary threshold losses as a susceptibility index for predicting permanent noise-induced hearing losses.

Damage Risk Criteria for Noise Exposure

Protecting the worker against noise-induced losses in hearing requires specification of the boundary conditions or limits for safe noise exposures. Initially, proposals for noise tolerance limits were expressed solely in terms of an over-all level of noise intensity. It became quickly apparent, however, that the spectrum of the noise, the time distribution of the individual exposure periods, the total duration of exposure, and the susceptibility of the person exposed also had to be included in a statement of such limits. At present considerable disagreement is reflected in estimates of safe noise exposure limits.

Speech Interference and Nonauditory Effects of Noise

Interference with communication—Noise which is not intense enough to cause hearing damage may still disrupt speech communication and the hearing of other desired sounds. Such disruptions will affect performance on those jobs which depend upon reliable speech communication. Even more important, however, is the fact that the inability to hear commands or danger signals due to excessive noise increases the probability of severe accidents.

The arithmetic average of the readings in decibels for the three octave bands (600–1,200 cps, 1,200–2,400 cps, and 2,400–4,800 cps) contained in wide-band noise can provide a simple indication of the ability of that noise to affect the intelligibility of person-to-person speech communication. This average is referred to as the *speech-interference-level* (SIL). In noises whose spectra yield an SIL of 75 db, personnel would have to speak in a very loud voice and use a selected and possibly prearranged vocabulary to be understood over a distance of 1 foot. Telephone use under these condi-

tions would probably be impossible. Noise having an SIL of 65 db would permit barely reliable communication with a raised voice over 2 feet. This range of communication would be extended to 4 feet by using a very loud voice, and to 8 feet by shouting. Telephone conversations under these SIL conditions would be difficult. In noise fields having an SIL of 55 db, a normal voice can communicate effectively over a distance of 3 feet, a raised voice over 6 feet, and a very loud voice over 12 feet. An SIL of 55 db would be permissible in work situations such as business or secretarial offices. When noise does not exceed an SIL of 45 db, a relaxed normal voice may be used for a distance of 10 feet. Such conditions would be ideal for private offices, or conference rooms.

Procedures for predicting speech communication under narrow-band noise conditions, under conditions where several conversations are going on simultaneously, and under peculiar reverberant room conditions have not as yet been completely worked out.

Impairments to performance—The effects of excessive noise on efficiency and work output seem to be somewhat slighter than is often thought. Performance on tasks involving simple repetitive operations, for example, does not appear to be affected by intense noise.

While efficiency in performing more complex operations may be adversely affected by noise, such effects in many instances tend to become dissipated as exposure time increases. One type of task, however, that shows pronounced and sustained performance decrements due to excessive noise is one requiring the worker to maintain a continuous watch over a number of dials to detect and report the presence of rarely occurring signals. This finding has implications for watch-keeping jobs where a worker may have to continuously scan or monitor a number of indicators to insure that no faults are developing in a machine or process. The finding also has practical importance for jobs requiring the inspection of items passing on a conveyor belt which cannot be viewed for an unlimited period of time.

The apparently limited effects of noise on performance make it difficult to determine which types of noise conditions will produce the greatest effects on work efficiency. The available evidence suggests that noises having over-all levels which exceed 90 db and containing predominantly high frequency components will be most effective in impairing performance.

Annoyance—Perhaps the most general reaction to noise is that of annoyance. Admittedly, there are wide individual differences as to what constitutes an annoying sound because of the many nonacoustical considerations that enter into such judgments. However, there are some basic characteristics of sound more annoying than others. These include:

(1) *Loudness*—the more intense and consequently louder noises are considered more annoying.

(2) *Pitch*—a high pitch noise, that is, one containing predominantly frequencies above 1,500 cps, is more annoying than a low pitch noise of equal loudness.

(3) *Intermittency and irregularity*—a sound that occurs randomly or varies in intensity or frequency is believed more annoying than one which is continuous and unchanging.

(4) *Localization*—a sound which repeatedly tends to change in localization is less preferred than one which remains stationary.

(5) *Inappropriateness to one's activity*—an example is the difference in attitude toward music when awake and when trying to sleep.

A measure of noise which is intended to describe its annoyance value has recently been developed and found to be successful in predicting the acceptability of fly-over noises produced by various types of aircraft. The measure is referred to as *perceived noise level in db* and is derived from calculations based upon the octave band intensity levels of the noise in question, together with data showing equal annoyance ratings for different octave bands of noise.

Physiologic effects—Physiologic reactions to a noise of sudden onset represent a typical startle pattern. There is a rise in blood pressure, an increase in sweating, an increase in heart rate, changes in breathing, and sharp contractions of the muscles over the whole body. These changes are often regarded as an emergency reaction of the body, increasing the effectiveness of any muscular exertion which may be required. However desirable in emergencies, these changes are not desirable for long periods since they would interfere with other necessary activities. Fortunately, these physiologic reactions subside with repeated presentations of the noise.

It has often been stated that for performance on a task to remain unimpaired by noise, man must exert greater effort than would be necessary under quiet conditions. Measures of energy expenditure—for example, oxygen consumption and pulse rate—show changes in the early stages of work under noisy conditions which are indicative of increased effort. With continued exposure, however, these responses return to their normal level.

Effects of Ultrasonic Stimulation

Sounds whose frequencies are above the upper frequency limit of audibility are called ultrasonic. Ordinarily, ultrasonic sounds are defined as being in excess of 20,000 cps.

Interest in the possible harmful effects of ultrasonic sounds on man became highlighted when jet propulsive devices came into use. The noise spectra of these devices contained a broad range of ultrasonic frequencies which were initially believed to be the basis for the headaches, nausea, undue fatigue, dizziness, and other complaints reported by personnel who worked in the jet sound field. Subsequent research, however, indicated no support for this belief. It was suggested that the ill effects were more probably due to the

tremendous intensities of sound, over 140 db, created in the audible range of frequencies by the jet engines.

One of the best known effects of airborne ultrasonic radiation is the production of heat on the body's surface. On surfaces having a high coefficient of heat absorption such as furry animals, ultrasonic stimulation above 150 db in intensity can cause death through overheating. Much higher intensities of ultrasonic stimulation are needed to create a similar effect on man because of the comparatively low coefficient of heat absorption of the human skin and the ability of the body to throw off heat through its thermal regulatory mechanism. These factors, together with the losses in ultrasonic intensity due to attenuation in air, make it highly unlikely that an individual will be exposed to lethal doses of airborne ultrasonic radiation. High intensity ultrasonic stimulation focussed at specific areas of the human body, however, will cause localized tissue and cellular damage which is attributed to the heating effect. This type of stimulation has been useful in experiments aimed at identifying the functions of various tissues and cells through selective destruction techniques. It also has implications for the removal of tumors that might not be otherwise reached with usual surgical procedures.

When transmitted through a liquid, intense ultrasounds form cavities which, upon collapsing, produce shock waves strong enough to tear holes in metals and other solids. This property, called *cavitation*, has led to the use of ultrasonics in the cleaning of metals. It has also provided the means for mixing liquids which would not otherwise mix, and breaking up complex chemical compounds. Subjecting blood to the cavitation effect caused by ultrasonic stimulation can rupture the membranes of the red blood cells. The conditions required for cellular disintegration, however, are not found with the typical applications of ultrasound so that this hazard is considered as unlikely.

With the exception of experimental applications, present uses for ultrasound are believed to pose only a slight risk to the exposed individual. Future applications, however, may require higher intensity sources which can pose a more serious hazard to the operator, particularly if he has contact with the source or if the medium of ultrasonic transmission to the operator is other than air—for example, liquid or solid.

Characteristics of Vibratory Motion

Vibration refers to any back-and-forth motion of matter. For present purposes, however, vibrations will refer only to low frequency back-and-forth motions of objects which are in contact with human beings. The vibratory range of particular interest to man is 1 to 400 cps, although it must be mentioned that the skin can detect vibrations in excess of 1,500 cps in frequency.

Besides frequency, other features of vibratory motion are displacement, velocity, and acceleration, each of which can serve to describe the magnitude

of vibratory motion. *Displacement* refers to the distance between the normal resting position of an object and its position at a given time in its vibratory cycle. The maximum displacement of the object from its normal resting position is called the amplitude of vibration. *Velocity* refers to the time rate of change of displacement. When a vibrating object reaches its peak upward or peak downward displacement, its velocity is zero. As the object passes in either an upward or downward direction through its normal resting position its velocity becomes maximal. The rate at which the velocity of the vibratory motion changes in direction (upward or downward) and magnitude defines the *acceleration* of the motion. Acceleration has been the measure most frequently used to describe the magnitude of vibratory motion since it is proportional to the forces contained in the motion and because it yields a smaller and consequently less cumbersome range of numerical values.

Effects of Whole Body Vibration on Man

The effect of vibration upon the body is motion and relative displacement. If the vibration frequency is below 3 cps, the body moves as a unit and the adverse effects experienced are of the type associated with motion sickness. As the frequency of vibration is increased, various parts of the body tend to respond differentially to the fluctuating forces. Specific frequencies within the range 4 to 12 cps, for example, will cause the hips, shoulders, and abdominal parts to resonate, resulting in an amplification of their response to the imposed vibration. The direction of the vibration (whether applied vertically or longitudinally) and the position of the person (sitting or standing) will have some influence upon the amount as well as the specific frequencies of resonance for these body components. Between 20 and 30 cps the skull will begin to resonate, which leads to a deterioration in visual acuity. A similar disturbance will occur between 60 and 90 cps, when the eyeballs show a tendency to resonate with the vibrating forces.

Animal studies have shown that high amplitudes of whole body vibration (acceleration of 10 to 20g; g equals 32 ft. per sec. per sec.) for short durations may cause mechanical damage to the heart, lungs, brain, intestines and other parts of the abdominal region. These types of vibration-induced injury appear possible for man. It is too early, however, to speculate from the animal data as to what frequency, amplitude, and duration conditions will cause such effects in humans. It is also of importance to note that a person may suffer chronic injuries from vibration exposures of long duration which cause no apparent acute effects. These effects are found after exposure to repeated blows or to random jolts such as those experienced in riding vehicles. Buffeting in aircraft or high speed small craft on the water, or shaking in tractors or in other heavy vehicles on rough surfaces, gives rise to jolting motions. Acute injuries from these conditions are rare, but complaints of discomfort are common. Truck and tractor operators, for example, often

have sacroiliac strain. Minor kidney injuries are sometimes suspected, and traces of blood may, in rare cases, appear in the urine.

Subjective responses to whole body vibration include perception of motion, feeling of discomfort, apprehension, and pain. Such responses depend upon a number of factors including vibration frequency, acceleration and duration of exposure.

Physiologic reactions to vibration have not as yet been extensively studied. Changes in respiration, heart activity, and peripheral circulation have been found in response to vibration but appear to be of a transient nature. Certain postural reflexes, such as the knee jerk, appear to be inhibited by vibratory motion.

Localized Vibration Effects

The best known of the injurious effects of vibration, and the one of immediate interest to industry, is associated with the use of hand-held power tools. Extensive use of pneumatic picks, hammers, and drills have been found to lead to a condition called *dead hand* or *white fingers*. This condition is characterized by numbness and blanching of the fingers with some loss of muscular control and reduction of sensitivity to heat, cold, and pain. Clinical findings show that the localized vibratory effect on the hands leads to pathologic changes in the vascular and nervous systems, and in the joints, tissues, and bones.

Heavy hand tools, such as pneumatic hammers and drills, produce vibrations which typically are below 60 cps but have peak accelerations which may be 100g or more. In contrast, light hand tools, such as those used in polishing and buffing, produce vibratory frequencies which range from 170 to 800 cps and have peak accelerations below 15g. The apparent differences in the vibration conditions for the heavy and light hand tools has led to some differences in the associated hand injuries. A comparison of the pathologic hand conditions caused by heavy low-speed hand tools and light high-speed hand tools is shown in the accompanying table.

Nature of hand impairment	Heavy, low-speed tools	Light, high-speed tools
Blanching of fingers	Characteristic	Absent.
Pain	Usually not a major complaint.	Major complaint.
Change in vascular tone	Not reported	Tone increased.
Swelling	Occasional	Frequent.
Degenerative changes in bone	Frequent	Absent.
Distribution of symptoms of neurovascular disturbance.	Usually in same hand as symptoms.	Usually in both hands.

Note: Duration of work prior to onset of impairment was several years in the case of heavy, low-speed tools; days to months in the case of light, high-speed tools.
Based on *Dart*, 1946.

References

Noise

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION: *Industrial Noise Manual.* The Association, Detroit, 1958. Presents data on proposed noise tolerance criterion, figure 7.5.

BROADBENT, D. E.: Effects of noise on behavior. *In* Harris, C. M. (editor): *Handbook of Noise Control.* McGraw-Hill Book Co., New York, 1957.

BROADBENT, D. E.: *Perception and Communication.* Pergamon Press, New York, 1958.

CARPENTER, A.: The effects of noise on work. *Ann. Occup. Hyg.* 1: 42, 1958.

FABRICANT, N. D.: Medicolegal otolaryngology. *Am. J. Med. Sc.* 237: 499, 1959. Occupational deafness, pp. 502–507; reproduces a table relating hearing loss to percent of compensable hearing loss.

GLORIG, A.: *Noise and Your Ear.* Grune & Stratton, New York, 1958.

GLORIG, A. AND DAVIS, H.: Age, noise and hearing loss. *Ann. Otol.* 70: 556, 1961.

GLORIG, A.; GRINGS, W., AND SUMMERFIELD, A.: Hearing loss in industry. *Laryngoscope* 68: 447, 1958.

GLORIG, A.; WARD, W. D., AND NIXON, J.: Damage risk criteria and noise-induced hearing loss. *Arch. Otolaryng.* 74: 413, 1961.

GRIMM, R. C. AND KUSNETZ, H. L.: The plasma torch; industrial hygiene aspects. *Arch. Environ. Health* 4: 295, 1962.

HINCHCLIFFE, R.: Threshold changes at 4 Kc/s produced by bands of noise. *Acta Otolaryng.* 47: 497, 1957.

HINCHCLIFFE, R.: Has your worker a noise problem? *Ann. Occup. Hyg.* 1: 55, 1958.

KARPLUS, H. B. AND BONVALLET, G. L.: A noise survey of manufacturing industries. *Am. Indust. Hyg. Assoc. Quart.* 14: 235, 1953.

KESSLER, H. E.: Noise problems in dentistry. *Oral Hyg.* 51: 27, (July) 1961. Refers to audio-distraction analgesia and ultraspeed equipment.

KYLIN, B.: Temporary threshold shift and auditory trauma following exposure to steady-state noise; an experimental and field study. *Acta Otolaryng.* Supp. 152, 1960.

LITTLER, T. S.: Noise measurement; analysis and evaluation of harmful effects. *Ann. Occup. Hyg.* 1: 11, 1958.

PARRACK, H. O.: Evaluating effects of industrial noise on man. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 5: 415, 1952.

PARRACK, H. O.: Ultrasound and industrial medicine. *In* Lectures Presented at University of Michigan Inservice Training Course on Acoustical Spectrum; Sound, Wanted and Unwanted. University of Michigan Press, Ann Arbor, Mich., 1952.

PETERSON, A. P. G. AND GROSS, E. E., JR.: *Handbook of Noise Measurement.* 4th ed. General Radio Co., West Concord, Mass., 1960.

RILEY, E. C.; STERNER, J. H.; FASSETT, D. W., AND SUTTON, W. L.: Ten years' experience with industrial audiometry. *Am. Indust. Hyg. Assoc. J.* 22: 151, 1961.

ROSENBLITH, W. A.; STEVENS, K. N.; AND BOLT, BERANEK & NEWMAN, INC.: *Handbook of Acoustic Noise Control.* Vol. 2, Noise and Man. WADC Technical Rept. 52–204. Wright-Patterson Air Force Base, Dayton, Ohio, 1953.

SCHNEIDER, E. J.; PETERSON, J. E.; HOYLE, H. R.; ODE, E. H., AND HOLDER, B. B.: Correlation of industrial noise exposures with audiometric findings. *Am. Indust. Hyg. Assoc. J.* 22: 245, 1961.

TEICHNER, W. H.; AREES, E., AND REILLY, R.: Noise and human performance, a psychophysiological approach. *Ergonomics* 6: 83, 1963.

WARD, W. D.; GLORIG, A., AND SKLAR, D. L.: Temporary threshold shift from octave-band noise; applications to damage risk criteria. *J. Acoust. Soc. Am.* 31: 522, 1959.

YAFFE, C. D. AND JONES, H. H.: Noise and hearing; relationship of industrial noise to hearing acuity in a controlled population. Pub. Health Service Pub. No. 850. U.S. Government Printing Office, Washington, D.C., 1961.

YOUNG, R. W.: Physical properties of noise and their specification. *In* Harris, C. M. (editor): *Handbook of Noise Control.* McGraw-Hill Book Co., New York, 1957.

Vibration

AGATE, J. N.: An outbreak of cases of Raynaud's phenomenon of occupational origin. *Brit. J. Indust. Med.* 6: 144, 1949.

AGATE, J. N. AND DRUETT, H. A.: A study of portable vibrating tools in relation to the clinical effects which they produce. *Brit. J. Indust. Med.* 4: 141, 1947.

DART, E. E.: Effects of high speed vibrating tools on operators engaged in the airplane industry. *Occup. Med.* 1: 515, 1946.

GOLDMAN, D. E.: Effects of vibration on man. *In* Harris, C. M. (editor): *Handbook of Noise Control.* McGraw-Hill Book Co., New York, 1957. Presents data on subjective responses to vibration, figure 11.4.

GOLDMAN, D. E. AND VON GIERKE, H. E.: The effects of shock and vibration on man. Lecture and review series 60–3. 111 references. Naval Medical Research Institute, Bethesda, Md., 1960.

HOERNER, E. F.: Traumatic vasospastic disease of the hand (Raynaud's phenomenon). *Indust. Med. & Surg.* 21: 297, 1952.

HUNTER, D.:M'LAUGHLIN, A. I. G., AND PERRY, K. M. A.: Clinical effects of the use of pneumatic tools. *Brit. J. Indust. Med.* 2: 10, 1945.

LINDER, G. S.: Mechanical vibration effects on human beings. *Aerospace Med.* 33: 939, 1962.

PARFITT, G. G.: The analysis and control of vibration. *Ann. Occup. Hyg.* 1: 68, 1958.

218-695 O-66-20

• section X

BIOLOGIC HAZARDS

MARCUS M. KEY, M.D.

Biologic agents as a cause of occupational disease have declined in importance since the advent of environmental sanitation and the anti-infection drugs. However, the danger is still present for several diseases, as is evidenced by the 15 to 20 cases of anthrax reported yearly in the United States, and the frequently observed elevated Brucella agglutination titers and Q fever complement-fixing antibody titers among certain occupational groups. So long as these diseases persist in the animal reservoir, there will be human cases.

Biologic agents may be conveniently classified as viral and rickettsial, bacterial, fungal, and parasitic. Potential occupational exposures are listed for the diseases in each of the four groups. For clinical manifestations and diagnostic tests the reader may consult items in the appended list of references.

Viruses and Rickettsia

Two important diseases in this group are ornithosis and Q fever. Though ornithosis was originally associated with the parrot family, it also affects ducks, chickens, and turkeys and has caused several outbreaks in poultry processing plants. The virus is present in nasal discharges and droppings of infected birds, and in aerosols from poultry processing. The virus enters man by the upper respiratory tract. Q fever is probably acquired by inhalation of the dried rickettsia in dairy or wool dust. Both of these infections have been acquired in the laboratory.

Potential Occupational Exposures

The following viral or rickettsial infections are potentially associated with the given occupations.

CATSCRATCH DISEASE
- cat handlers
- dog handlers
- veterinarians

ECTHYMA CONTAGIOSUM (ORF)
- ranchers
- sheep handlers
- shepherds

MILKER'S NODULES
- dairy workers
- farmers
- veterinarians

NEWCASTLE DISEASE
- chicken handlers
- laboratory workers
- poultry house workers
- poultry processors
- turkey handlers

ORNITHOSIS (PSITTACOSIS)
- canary handlers
- chicken handlers
- dock workers
- duck handlers
- laboratory workers
- lovebird handlers
- parakeet handlers
- parrot handlers
- pigeon handlers
- poultry processors
- turkey handlers

Q FEVER
- animal breeders
- cattle handlers
- dairy workers
- farmers
- goatherds
- laboratory workers
- shepherds
- slaughterhouse workers
- wool handlers

RABIES
- dog pound workers
- mail carriers
- meter readers
- veterinarians

ROCKY MOUNTAIN SPOTTED FEVER
- farmers
- foresters
- hunters
- laboratory workers
- ranchers
- shepherds
- trappers

Bacteria

Secondarily infected occupational trauma or dermatitis may be found in almost any occupation. The causative organisms are usually staphylococci or streptococci. Insoluble cutting oils commonly cause folliculitis and furuncles, but the infection occurs from skin bacteria being trapped within the follicle rather than from pathogenic bacteria in the insoluble cutting oil.

Industrial anthrax is an infection acquired from infected wool, hair, or hides imported from countries where anthrax is present. It is usually cutaneous, but may be pulmonary when the spores are inhaled. Agricultural anthrax is acquired from infected livestock in certain enzootic areas of the United States.

Potential Occupational Exposures

The following bacterial infections are potentially associated with the given occupations.

ANTHRAX (WOOLSORTER'S DISEASE)
- animal handlers
- bacteriologists
- blanket makers
- bonemeal workers
- broom makers

ANTHRAX (WOOLSORTER'S DISEASE)—con.
brush makers
butchers
carpet cleaners
carpet makers
cattle handlers
cobblers
cordage factory workers
curriers
dairy workers
dock workers
farmers
fat renderers
felt makers
fertilizer makers
freight handlers
fur carders
fur clippers
fur cutters
fur handlers
fur preparers
fur pullers
gelatin makers
glue makers
goat hair handlers
goat hide handlers
hair workers
leather workers
mattress makers
meat inspectors
minkery workers
plasterers
ragmen
shavers, felt hat
shavers, fur
shaving brush makers
shepherds
slaughterhouse workers
stablemen
tanners
tannery workers
taxidermists
upholsterers
veterinarians
warehouse workers
wooden heel workers
wool carders
wool scourers
wool spinners
wool workers

BRUCELLOSIS (UNDULANT FEVER)
butchers
carcass handlers
cattle handlers
cooks
dairy workers
farmers
gardeners
goatherds
hide handlers
manure handlers
meat inspectors
milk inspectors
packing-house workers
sausage stuffers
sewer workers
shearers
shepherds
slaughterhouse workers
stablemen
stockmen
swine handlers
veterinarians
zoologic technicians

ERYSIPELOID
butchers
button makers, bone
cooks
farmers
fishermen
fish handlers
game handlers
kitchen workers
meat inspectors
swine handlers
veterinarians

FOLLICULITIS, FURUNCULOSIS
- animal handlers
- battery makers, storage
- chocolate workers
- glue workers
- hospital attendants
- ice cream workers
- machinists
- mechanics
- oilers
- pitch workers
- sugar workers
- tallow refiners
- tar workers
- veterinarians

LEPTOSPIROSIS (WEIL'S DISEASE)
- animal handlers
- canal workers
- cane field workers
- cattle handlers
- dairy workers
- ditch diggers
- dock workers
- dog pound workers
- farmers
- fishermen
- fish market workers
- gardeners
- miners
- pig farm workers
- poultry dressers
- rice field workers
- sewer workers
- slaughterhouse workers
- street cleaners
- swine handlers
- trench diggers
- tunnel diggers
- veterinarians

LISTERIOSIS
- animal handlers
- cattle handlers
- dairy workers
- sheep handlers

PLAGUE
- geologists
- hunters
- linemen
- shepherds

TETANUS
- butchers
- cattle handlers
- farmers
- horse handlers
- packing-house workers
- slaughterhouse workers

TULAREMIA
- bacteriologists
- butchers
- cooks
- farmers
- forestry workers
- hunters
- rabbit handlers
- shearers

VERRUCA NECROGENICA
- anatomists
- autopsy room attendants
- butchers
- dissecting room attendants
- embalmers
- nurses
- physicians
- slaughterhouse workers

Fungi

A wide variety of fungi is responsible for mycotic infections. Many of these infections are superficial and localized, and of minor clinical importance; others are systemic and involve deeper tissues, with occasional fatal outcome.

Dermatophytosis or athlete's foot is not usually an occupational disease. It is generally accepted that the causative dermatophytes are not acquired from the floors of showers and locker rooms, but are carried on the skin of most persons without causing disease. The cause is a lowering of local resistance by occlusive footwear, increased perspiration, and poor foot hygiene in certain susceptible individuals.

Occupationally-caused fungous diseases of the lungs include histoplasmosis, coccidioidomycosis, and aspergillosis. The symptoms and chest findings are similar to those associated with tuberculosis, but sputum and immunologic studies help to differentiate these diseases. Blastomycosis is sometimes classified as an occupational disease of farmers. This disease, as well as histoplasmosis and coccidioidomycosis, is limited to certain endemic areas.

Potential Occupational Exposures

The following fungal infections are potentially associated with the given occupations

ASPERGILLOSIS
- bird handlers
- grain mill workers

CHROMOBLASTOMYCOSIS
- farmers
- laborers

COCCIDIOIDOMYCOSIS
- farmers
- fruit pickers
- shepherds

HISTOPLASMOSIS
- farmers
- guano workers
- poultrymen

MONILIASIS
- bakers
- bartenders
- cannery workers
- dishwashers
- kitchen workers

MYCETOMA PEDIS
- farmers
- laborers

SPOROTRICHOSIS
- berry pickers
- farmers
- florists
- foresters
- gardeners
- nurserymen
- South African miners

TINEA CIRCINATA (RINGWORM)
- animal handlers
- barbers
- cat handlers
- cattle handlers
- dog handlers
- farmers
- fur handlers
- hide bundlers
- horse handlers
- monkey handlers
- wool sorters

Parasites

Many vegetables, fruits, and food products have associated mites which may fortuitously attack man. These mites may also parasitize rats and several species of birds.

Creeping eruption is limited to the southeastern United States, where the warm, sandy soil favors the hatching out of infective larvae from dog and cat hookworm eggs.

Potential Occupational Exposures

The following parasitic infections or infestations are potentially associated with the given occupations.

CHIGGER BITES
- construction workers
- farmers
- linemen
- pipeline workers
- surveyors

CREEPING ERUPTION
- brick masons
- ditch diggers
- laborers
- lifeguards
- plumbers

FOWL MITE DERMATITIS
- office workers
- poultrymen

GRAIN ITCH
- barley handlers
- cotton seed handlers
- farmers
- grain elevator workers
- strawboard makers

GROCER'S ITCH
- cheese handlers
- copra handlers
- date handlers
- dock workers
- fig handlers
- flour handlers
- grocers
- meal handlers
- prune handlers
- vanilla handlers

References

ANON.: Catscratch disease. 22 references. *Brit. Med. J.* 1: 189, 1961.

ANON.: Epidemiological reports, anthrax in man, 1960. *Morbidity and Mortality Weekly Report.* Communicable Disease Center, Public Health Service 10: 1, (June 2) 1961.

AMERICAN PUBLIC HEALTH ASSOCIATION: *Control of Communicable Diseases in Man.* 9th ed. The Association, New York, 1960.

BAKER, C. C.; FELTON, F. G., AND MUCHMORE, H. G.: Listeriosis; report of 5 cases. *Am. J. Med. Sc.* 241: 739, 1961.

BEAVER, P.: Wandering nematodes as a cause of disability and diseases. *Am. J. Trop. Med. & Hyg.* 6: 433, 1957.

BOOTH, B. H. AND JONES, R. W.: Mites in industry. *A.M.A. Arch. Dermat. & Syph.* 69: 531, 1954.

BRACHMAN, P. S.; PAGANO, J. S., AND ALBRINK, W. S.: Two cases of fatal inhalation anthrax, one associated with sarcoidosis. *New Eng. J. Med.* 265: 203, 1961.

COGHLAN, J. D. AND NORVAL, J.: Canicola fever in man from contact with infected pigs. *Brit. Med. J.* 2: 1711, 1960.

DALRYMPLE-CHAMPNEYS, W.: *Brucella Infection and Undulant Fever in Man.* Oxford University Press, New York, 1960.

DUBOS, R. J. (EDITOR): *Bacterial and Mycotic Infections of Man.* 3rd ed. J. B. Lippincott Co., Philadelphia, 1958.

FARMER, J. L. AND PERRY, H. O.: Orf, an occupational disease in Minnesota. *Minn. Med.* 43: 818, 1960.

FRANCIS, T., JR. AND SMADEL, J. E. (EDITORS): *Diagnostic Procedures for Virus and Rickettsial Diseases.* American Public Health Association, New York, 1956.

HANSON, R. P. AND BRANDLY, C. A.: Newcastle disease. *In* Animal Disease and Human Health. *Ann. N.Y. Acad. Sc.* 70 (art. 3): 585, 1958.

JILLSON, O. F. AND BUCKLEY, W. R.: Fungous disease in man acquired from cattle and horses (due to Trichophyton faviforme). *New Eng. J. Med.* 246: 996, 1952.

KENDALL, C. E.: Occupational anthrax in the United States. *J. Occup. Med.* 1: 174, 1959.

LITWIN, S. B.: "Pigeon mites" causing a pruritic dermatitis; report of a case. *J. Am. Med. Assoc.* 177: 714, 1961.

MACPHERSON, L. W.: Bacterial infections of animals transmissible to man. 121 references. *Am. J. Med. Sc.* 239: 347, 1960.

MEEK, S. F. AND MCCORD, C. P.: Medical entomology in industrial medicine in the United States. *Indust. Med.* 13: 1003, 1944.

MENGIS, C. L.: Plague. *New Eng. J. Med.* 267: 543, 1962. Three case reports; the first patient had hunted in wooded areas and herded sheep, the second was a geologist, and the third, a telephone lineman.

MILLER, J. K.: Human anthrax in New York State. *N.Y. State J. Med.* 61: 2046, 1961.

MITCHELL, C. A.: Animal viruses transmissible to man. 25 references. *Am. J. Med. Sc.* 237: 359, 1959.

PIKE, R. M. AND SULKIN, S. E.: Occupational hazards in microbiology. *Sc. Monthly* 75: 222, 1952.

RAPHAEL, S. S. AND SCHWARZ, J.: Occupational hazards from fungi causing deep mycoses. *A.M.A. Arch. Indust. Hyg & Occup. Med.* 8: 154, 153.

RINDGE, M. E.; JUNGHERR, E. L., AND SCRUGGS, J. H.: Serologic evidence of occupational psittacosis in poultry-plant workers. *New Eng. J. Med.* 260: 1214, 1959.

RIVERS, T. M. AND HORSFALL, F. L., JR. (EDITORS): *Viral and Rickettsial Infections of Man.* 3rd ed. J. B. Lippincott Co., Philadelphia, 1959.

STITT, E. R.; CLOUGH, P. W., AND BRANHAM, S. E.: *Practical Bacteriology, Hematology, and Parasitology.* 10th ed. McGraw-Hill Book Co., New York, 1948.

SWIFT, S. AND COHEN, H.: Granulomas of the skin due to Mycobacterium balnei after abrasions from a fish tank. *New Eng. J. Med.* 267: 1244, 1962.

TREVER, R. W.; CLUFF, L. E.; PEELER, R. N., AND BENNETT, I. L.: Brucellosis. 1, Laboratory-acquired acute infection. *A.M.A. Arch. Int. Med.* 103: 381, 1959.

U.S. DEPARTMENT OF AGRICULTURE: *Animal Diseases.* The Yearbook of Agriculture, 1956. U.S. Government Printing Office, Washington, D.C., 1956.

WINN, J. F.; CHERRY, W. B., AND KING, E. O.: Listeriosis; a potential public health problem. 81 references. *In* Animal Disease and Human Health. *Ann. N.Y. Acad. Sc.* 70 (art. 3): 624, 1958.

• section XI

PLANT AND WOOD HAZARDS

MARCUS M. KEY, M.D.

Many plants and plant products, as well as woods, are known to cause occupational diseases with local and systemic manifestations. Plants and plant products are common offending agents producing these manifestations, dermatitis being the most frequently observed effect.

Plants and Plant Products

The most common occupational disease from plants is allergic contact dermatitis from poison ivy and oak which usually has such characteristic linear and bullous lesions that it is easily recognized as of plant origin. Poison ivy thrives throughout the United States, except in the southwest, and causes contact dermatitis in a high percentage of outdoor workers. Poison oak is equally troublesome, but is restricted to the west coast. A variant of poison ivy, called oakleaf poison ivy, is found in the southeastern United States.

Poison ivy and oak belong to the Anacardiaceae, or cashew, family of plants which includes other sources of sensitizing or cross-reacting agents, such as poison sumac, mango, Japanese lac tree, Indian marking nut, and cashew nut tree. From cashew nut shells is extracted a phenolic liquid which forms a condensation resin when combined with formalin. It is known as cashew nut shell liquid-formaldehyde resin and is used in the manufacture of varnishes and brake shoe linings. The liquid is a potent sensitizer as well as a strong primary irritant.

In the southern United States, the bastard feverfew, a common weed, produces an allergic contact dermatitis similar to that caused by poison ivy.

The harvesting and processing of fruits and vegetables are attended by allergic contact dermatitis. This has been reported from handling asparagus, carrots, oranges, and lemons, among others. In addition, fruit and vegetable handlers may also develop contact dermatitis from insecticides and fungicides; severe chapping and moniliasis from excessive exposure to

moisture; photosensitization dermatitis from concurrent or subsequent exposure to sunlight; and parasitism from exposure to fruit, vegetable, and grain mites.

Frequent plant photosensitizers are fig, rue, lime, bergamot, and members of the Umbelliferae including parsnip, parsley, carrot, fennel, dill, and celery (pink rot). By photosensitization is meant the delayed development of erythema, edema, vesicles, and bullae following contact with the plant juices and exposure to sunlight. This is an accentuated localized sunburn, and eventuates in either hyperpigmentation or depigmentation, depending on the severity of the reaction. Reported case of phytophotosensitization have been instances of phototoxicity rather than photoallergy. Phytophototoxins are psoralen compounds, and their activity is believed to be associated with the furocoumarin ring.

Contact with certain flowers frequently produces dermatitis on an allergic basis. Examples are chrysanthemum, pyrethrum, primrose, and the bulbs of narcissus, tulips, and hyacinth. Ragweed pollen may cause several types of occupational allergy among farmers and highway workers. The water-soluble protein fraction of the pollen may cause hay fever and asthma. The lipid fraction may cause an eczematous dermatitis of exposed areas.

Reports of asthma, hay fever, and urticaria from castor bean processing are common. The dried pomace resulting from castor oil extraction contains a potent allergen which is responsible for symptoms reported among castor bean workers and others in the vicinity of extraction plants, among farmers using the pomace as fertilizer, and among dock workers engaged in unloading bags of castor bean pomace.

Potential Occupational Exposures

Botanists
Bulb handlers, plant
Camp workers
Canners
Construction workers
Dock workers
Farmers
Field laborers
Flower cutters
Flower packers
Foresters
Fruit pickers
Fruit processors
Gardeners
Highway workers
Hop pickers
Horticulturists
Pipeline workers
Road builders
Surveyors
Telephone linemen
Utility workers
Vegetable harvesters
Vegetable processors

Woods

Occupational diseases from woods may be classified as toxic, irritant, or allergenic in nature.

A toxic wood is one that contains a substance, usually an alkaloid, which may be inhaled, ingested, or absorbed through the skin with the resultant occurrence of systemic signs and symptoms. These may include headache, anorexia, nausea, vomiting, bradycardia, dyspnea, and somnolence. Examples of toxic woods are East Indian satinwood, South African boxwood, and ipe.

An irritant wood may cause injury to mucous membranes when contact occurs and symptoms of sneezing, coughing, rhinorrhea, or tearing may result. Some irritant woods may damage the intact skin resulting in contact dermatitis of the primary irritation type. Mansonia, dahoma, and cocobolo are examples of irritant woods.

An allergenic wood may precipitate many different allergic manifestations in a sensitized individual. Most common of these are asthma and contact dermatitis. Certain members of the birch, pine, dogwood, beech, mahogany, mulberry, and myrtle families present examples of such woods.

Patch testing with sawdust, shavings, and sap may aid in differentiating between primary skin irritation and skin sensitization.

Potential Occupational Exposures

Cabinet makers
Carpenters
Lumbermen
Musicians
Sawmill operators
Violin makers

References

BIRMINGHAM, D. J.; KEY, M. M.; TUBICH, G. E., AND PERONE, V. B.: Phototoxic bullae among celery harvesters. *Arch. Dermat.* 83: 73, 1961.

BRUGSCH, H. G.: Toxic hazards; the castor bean. *New Eng. J. Med.* 262: 1039, 1960.

FROMER, J. L. AND BURRAGE, W. S.: Ragweed oil dermatitis. *J. Allergy* 24: 425, 1953.

HUNTER, D.: *The Diseases of Occupations.* Little, Brown and Co., Boston, 1955.

KLAUDER, J. V. AND KIMMICH, J. M.: Sensitization dermatitis to carrots. *A.M.A. Arch. Dermat.* 74: 149, 1956.

KROGH, H. K.: Contact eczema caused by true teak (Tectona grandis). An epidemiological investigation in a furniture factory. *Brit. J. Indust. Med.* 19: 42, 1962.

MCCORD, C. P.: The toxic properties of some timber woods. *Indust. Med. & Surg.* 27: 202, 1958. The occupational toxicity of cultivated flowers. *Ibid.* 31: 365, 1962.

MUENSCHER, W. C.: *Poisonous Plants of the United States.* Revised ed. Macmillan Co., New York, 1951.

ROOK, A.: Plant dermatitis. *Brit. Med. J.* 2: 1771, 1960.

SCHWARTZ, L.; TULIPAN, L., AND BIRMINGHAM, D. J.: *Occupational Diseases of the Skin.* 3rd ed. Lea & Febiger, Philadelphia, 1957.

WEBER, L. F.: Dermatitis venenata due to native woods. *A.M.A. Arch. Dermat.* 67: 388, 1953.

• section XII

SOURCES OF CONSULTATION ON OCCUPATIONAL HEALTH

The problem of a possible relationship between the patient's illness and his occupational environment is not always easily solved. At times the investigator may feel the need of consultation after he has reviewed the pertinent literature and given consideration to all of the factors believed relevant. Sources of consultation reside in the official and nonofficial agencies. The official agencies include those established in the Federal Government and those existing in States, counties and cities. Among the nonofficial agencies are the insurance companies, particularly those issuing workmen's compensation insurance policies, a number of universities, and professional groups engaged in private consultation. This section presents a list of the State and local agencies with resources available to the investigator, the diversity and extent of such resources being dependent upon administrative and budgetary factors and the supply of trained personnel.

State and Local Agencies

ALASKA Environmental Health, Division of Public Health, Alaska Department of Health and Welfare, Alaska Office Building, Juneau 99801

CALIFORNIA (a) Bureau of Occupational Health, California Department of Public Health, 2151 Berkeley Way, Berkeley 94704. (b) Division of Industrial Safety, Department of Industrial Relations, 455 Golden Gate Avenue, San Francisco 94102. (c) State Compensation Insurance Fund. Department of Industrial Relations, 525 Golden Gate Avenue, San Francisco 94102

Alameda County Bureau of Environmental Health, Alameda County Health Department, 499 Fifth Street, Oakland 94607

Albany City Albany City Health Department, 1045 Solano Avenue, Albany 94706

Berkeley City Berkeley City Health Department, 2121 McKinley Street, Berkeley 94704

Contra Costa County Occupational Health Division, Contra Costa County Health Department, P.O. Box 871, Martinez

Long Beach City Long Beach City Health Department, 2655 Pine Avenue, Long Beach

Los Angeles City Division of Occupational and Radiological Health, Los Angeles City Health Department, 111 East First Street, Los Angeles 90012

Los Angeles County Division of Industrial Health and Hygiene, Los Angeles County Health Department, 241 North Figueroa Street, Los Angeles 90012

Orange County Division of Sanitation, Orange County Health Department, P.O. Box 355, Santa Ana 92702

Pasadena City Occupational and Radiological Health Services Section, Environmental Health Division, City of Pasadena, Department of Public Health, 100 North Garfield Avenue, Pasadena 91109

San Bernardino County Division of Environmental Sanitation, San Bernardino County Health Department, 316 Mountain View Avenue, San Bernardino

San Diego County Bureau of Industrial Hygiene and Air Pollution Control, County of San Diego Department of Public Health, Civic Center, San Diego 92101

San Jose City San Jose City Health Department, 151 West Mission, San Jose

San Mateo County Sanitation Section, San Mateo County Department of Public Health and Welfare, 225 37th Avenue, San Mateo

Santa Clara County Bureau of Occupational Health, County of Santa Clara Health Department, 2220 Moorpark Avenue, San Jose

Santa Cruz County Environmental Health Section, Santa Cruz County Health Department, P.O. Box 962, Santa Cruz

Stanislaus County Stanislaus County Health Department, 810 Scenic Drive, Modesto

Vernon City Vernon City Health Department, 4305 Santa Fe Avenue, Vernon

COLORADO Division of Occupational and Radiological Health, Colorado State Department of Public Health, 4210 East 11th Avenue, Denver 80220

Denver City Occupational Health Section, Environmental Health Service, Department of Health and Hospitals, 659 Cherokee Street, Denver 80204

CONNECTICUT Occupational Health Section, Division of Medical Services, Connecticut State Department of Health, State Office Building, Hartford 06115

FLORIDA Division of Radiological and Occupational Health, Bureau of Preventable Diseases, Florida State Board of Health, P.O. Box 210, Jacksonville 32201

Hillsborough County Division of Occupational and Radiological Health, Hillsborough County Health Department, P.O. Box 1731, Tampa 33601

GEORGIA Occupational Health Service, Georgia Department of Public Health, Atlanta 30303

Fulton County Industrial Sanitation Section, Fulton County Health Department, 99 Butler Street, S.W., Atlanta

HAWAII Occupational and Radiological Health Section, Health Engineering Branch, Division of Environmental Health, Department of Health, P.O. Box 3378, Honolulu

IDAHO Engineering and Sanitation Division, Idaho Department of Health, Statehouse, Boise 83701

ILLINOIS Industrial Hygiene Unit, Safety Inspection and Education Division, Illinois Department of Labor, 160 North LaSalle Street, Chicago

INDIANA Division of Industrial Hygiene, Indiana State Board of Health, 1330 West Michigan Street, Indianapolis

KANSAS Industrial, Radiation and Air Hygiene Program, Kansas State Department of Health, State Office Building, Topeka

Wichita-Sedgwick County Division of Environmental Health, Wichita-Sedgwick County Department of Public Health, 1900 East Ninth, Wichita 67214

KENTUCKY Occupational Health Program, Division of Environmental Health, Kentucky Department of Health, 275 East Main Street, Frankfort 40601

LOUISIANA Occupational Health and Safety Section, Division of Preventive Medicine, Louisiana State Board of Health, P.O. Box 60630, New Orleans

MAINE Occupational and Radiological Health Section, Division of Sanitary Engineering, State Department of Health and Welfare, Augusta 04330

MARYLAND Division of Occupational Health, State Department of Health, State Office Building, 301 West Preston Street, Baltimore

Baltimore Bureau of Industrial Hygiene, Baltimore City Health Department, 602 American Building, Baltimore 21202

MASSACHUSETTS Division of Occupational Hygiene, Massachusetts Department of Labor and Industries, 286 Congress Street, Boston 02210

MICHIGIAN Division of Occupational Health, Michigan Department of Health, 3500 North Logan Street, Lansing 48914

Detroit Bureau of Industrial Hygiene, Detroit Department of Health, 3801 John C. Lodge Expressway, Detroit 48202

MINNESOTA Section of Occupational Health, Division of Environmental Health, Minnesota Department of Health, University Campus, Minneapolis 55440

Minneapolis Occupational Health Service, Division of Public Health, 250 Fourth Street South, Minneapolis 55415

218-695 O-66-21

MISSISSIPPI Division of Occupational Health, Mississippi State Board of Health, P.O. Box 1700, Jackson

MISSOURI Radiological and Occupational Health, Division of Health of Missouri, Jefferson City

St. Louis City Industrial Hygiene Section, St. Louis Division of Health, 62 Municipal Courts Building, St. Louis

MONTANA Division of Disease Control, State Board of Health, Helena

NEW HAMPSHIRE Occupational Health Service, Division of Public Health, Department of Health and Welfare, 61 South Spring Street, Concord

NEW JERSEY Occupational Health Program, Division of Environmental Health, New Jersey State Department of Health, 17 West State Street, Trenton 08625

NEW MEXICO Occupational Health Section, Division of Environmental Sanitation Services, New Mexico Department of Public Health, Santa Fe

Albuquerque Albuquerque City Health Department, P.O. Box 1293, Albuquerque 87103

NEW YORK Division of Industrial Hygiene, New York State Department of Labor, 80 Centre Street, New York 10013

NORTH CAROLINA Occupational Health Section, North Carolina State Board of Health, Raleigh

Charlotte Division of Environmental Health, City of Charlotte Health Department, 1200 Blythe Boulevard, Charlotte

OHIO (a) Division of Industrial Hygiene, Ohio Department of Health, 1147 Chesapeake Avenue, Columbus 43212. (b) Division of Safety and Hygiene, Industrial Commission of Ohio, 700 W. 3rd Avenue, Columbus

Cincinnati Occupational Health Services, Cincinnati Health Department, City Hall, Cincinnati

Cleveland Bureau of Industrial Hygiene, Division of Air Pollution Control, Cleveland Department of Urban Renewal and Housing, 14101 Lakeshore Boulevard, Cleveland 44110

OKLAHOMA Occupational and Radiological Health Section, Environmental Health Service, Oklahoma State Department of Health, 3400 North Eastern, Oklahoma City 73105

OREGON Occupational and Radiological Health Section, Division of Preventive Medical Services, Oregon State Board of Health, 1400 S.W. Fifth Avenue, Portland 97207

PENNSYLVANIA Division of Occupational Health, Pennsylvania Department of Health, Health and Welfare Building, Harrisburg 17120

Allegheny County Division of Occupational Health, Allegheny County Health Department, 620 City-County Building, Pittsburgh 15219

Philadelphia Occupational and Radiological Health Section, Division of Environmental Health, Philadelphia Department of Public Health, 500 South Broad Street, Philadelphia 19146

PUERTO RICO Section of Occupational Health, Division of Sanitation, Puerto Rico Department of Health, San Juan

RHODE ISLAND Division of Industrial Hygiene, Rhode Island Department of Health, 365 State Office Building, Providence

SOUTH DAKOTA Occupational and Radiological Health Section, Division of Sanitary Engineering, State Department of Health, Pierre

TENNESSEE Industrial Hygiene Service, Division of Preventable Diseases, Tennessee Department of Public Health, Cordell Hull State Office Building, Nashville 37219

TEXAS Division of Occupational Health and Radiation Control, Texas State Department of Health, 1100 West 49th Street, Austin

Dallas City of Dallas, Public Health Department, Dallas

Harris County Harris County Health Department, P.O. Box 4116, Houston 77014

Houston Industrial Hygiene Program, Houston City Health Department, 612 Bagby, Houston 77002

UTAH Section of Industrial Hygiene, Division of Sanitation, Utah State Department of Health, 45 S. Fort Douglas Boulevard, Salt Lake City 84113

VERMONT Industrial Hygiene Division, Vermont Department of Health, P.O. Box 333, Barre 05641

VIRGINIA Bureau of Industrial Hygiene, Virginia State Department of Health, State Office Building, Richmond 23219

WASHINGTON Division of Safety, Department of Labor and Industries, General Administration Building, Olympia 98502

WEST VIRGINIA Bureau of Industrial Hygiene, West Virginia Department of Health, State Office Building, Charleston

WISCONSIN Occupational Health Division, Wisconsin State Board of Health, State Office Building, 1 West Wilson Street, Madison

Milwaukee Industrial Hygiene Section, Environmental Technical Services Division, City of Milwaukee Health Department, 841 North Broadway, Milwaukee 53202

WYOMING Division of Industrial Hygiene, Wyoming Department of Public Health, State Office Building, Cheyenne

• section XIII

OCCUPATIONAL HEALTH REFERENCE AIDS

The occupational health reference aids listed in this section supplement the references appended to other sections. The material includes indexes, abstract journals and abstracting services, occupational health journals, bibliographies, and texts.

Separately presented is a selection of texts published in 1943 or earlier. An examination of these texts will reveal that some so-called modern practices date back many years. In short, the inclusion of the older works will have served its purpose if the reader sampling them comes to recognize that they do not deserve the oblivion to which many of them have been carelessly consigned. It should be pointed out, moreover, that the older literature is by no means confined to the recent past. The literature of previous centuries is relatively vast. The historically-minded reader may gain supplementary knowledge as well as stimulation by using as a guide the *Index-Catalogue of the Library of the Surgeon General's Office, U.S. Army*, particularly volume 10 published in 1889. The caption, "Occupations and trades (diseases and hygiene of)," requires seven quarto pages (67–73) to index the books and the articles carried by journals on the shelves of the library (now the National Library of Medicine, U.S. Public Health Service).

Indexes

CHEMICAL TITLES, Current Author and Keyword Indexes from Selected Chemical Journals, a Product of the Chemical Abstracts Service. American Chemical Society, Easton, Pa. Titles chosen from some 600 journals of pure and applied chemistry and chemical engineering are covered in semimonthly issues.

INDEX-CATALOGUE of the Library of the Surgeon General's Office, U.S. Army. Vol. 10. U.S. Government Printing Office, Washington, D.C., 1889.

INDEX MEDICUS, formerly *Current List of Medical Literature*. National Library of Medicine, U.S. Public Health Service, Washington, D.C. Indexes by subject and by author articles appearing in world medical literature. Published monthly. U.S. Government Printing Office, Washington, D.C. American Medical Association, Chicago, publishes annual cumulations under the title, *Cumulated Index Medicus*. Volume 1 covering 1960 was published in 1961 in three separately bound parts. Part 1 carries a list of the journals indexed as well as an author index. Parts 2 and 3 constitute a subject index.

Abstract Journals and Abstracting Services

APCA ABSTRACTS. Air Pollution Control Association in cooperation with U.S. Public Health Service and Library of Congress. The Association, Mellon Institute, Pittsburgh, Pa. Abstracts prepared from some 500 journals appear monthly.

BIOLOGICAL ABSTRACTS. Biological Abstracts, Inc., University of Pennsylvania, Philadelphia. Abstracts covering the areas of biology, zoology and botany are published in semimonthly issues.

BULLETIN OF HYGIENE. Bureau of Hygiene & Tropical Diseases, Keppel Street, London. Carries a section on occupational hygiene. Presents abstracts of articles from the world literature. Published monthly in 2 editions, one printed on both sides of the page and the other on one side only.

CHEMICAL ABSTRACTS, Key to the World's Chemical Literature, a Product of the Chemical Abstracts Service. American Chemical Society, Easton, Pa. Published biweekly.

CIS, International Abstracting and Information Service on Occupational Safety and Health. International Labor Office, Geneva, Switzerland. Abstracts of articles on occupational safety and health appearing in the world literature are distributed monthly to subscribers on 3- by 5-inch cards.

DIGEST OF NEUROLOGY AND PSYCHIATRY; Abstracts and Reviews of Selected Literature in Psychiatry, Neurology and their Allied Fields. The Institute of Living, Hartford, Conn. Published monthly.

EXCERPTA MEDICA, International Medical Abstracting Service. Excerpta Medica Foundation, 2 East 103rd Street, New York, N.Y. Section 17 entitled Public Health, Social Medicine and Hygiene (including Industrial Medicine and Hygiene) is published monthly.

INDUSTRIAL HYGIENE DIGEST. Industrial Hygiene Foundation, Mellon Institute, Pittsburgh, Pa. Abstracts prepared from some 400 journals appear monthly.

NUCLEAR SCIENCE ABSTRACTS. U.S. Atomic Energy Commission, Oak Ridge, Tenn. Abstracts and indexes the literature of nuclear science and technology. Published twice monthly. U.S. Government Printing Office, Washington, D.C.

PUBLIC HEALTH ENGINEERING ABSTRACTS. Robert A. Taft Sanitary Engineering Center, U.S. Public Health Service, Cincinnati, Ohio. Includes abstracts on occupational health, radiologic health, and insect-borne diseases. Published monthly. U.S. Government Printing Office, Washington, D.C.

U.S. GOVERNMENT RESEARCH REPORTS. Office of Technical Services, U.S. Department of Commerce, Washington, D.C. Abstracts of research and development reports of the Army, Navy, Air Force, Atomic Energy Commission and other agencies of the U.S. Government. Published twice monthly. U.S. Government Printing Office, Washington, D.C.

Occupational Health Journals

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION JOURNAL. American Industrial Hygiene Association, 14125 Prevost, Detroit, Mich.

ANNALS OF OCCUPATIONAL HYGIENE (London). Pergamon Press, 122 East 55th Street, New York, N.Y.

ARCHIVES BELGES DE MÉDECINE SOCIALE, HYGIÈNE, MÉDECINE DU TRAVAIL ET MÉDECINE LÉGALE. Ministère de la Santé Publique et de la Famille, 43 Avenue des Arts, Brussels, Belgium

ARCHIVES OF ENVIRONMENTAL HEALTH. American Medical Association, 535 North Dearborn Street, Chicago, Ill.

ARCHIVES DES MALADIES PROFESSIONELLES, DE MÉDECINE DU TRAVAIL ET DE SÉCURITÉ SOCIALE. Masson et Cie, Libraires de L'Académie de Médecine, 120 Boulevard Saint-Germain, Paris, France

ARCHIV FÜR GEWERBEPATHOLOGIE UND GEWERBEHYGIENE. Springer-Verlag, Heidelberger Platz 3, Berlin-Wilmersdorf, Germany

BERUFSDERMATOSEN. Editio Cantor, Verlag für Medizin und Naturwissenschaften, Aulendorf, Württemberg, Germany

BRITISH JOURNAL OF INDUSTRIAL MEDICINE. British Medical Association, Tavistock Square, London, England.

INDUSTRIAL MEDICINE AND SURGERY. Industrial Medicine Publishing Co., P.O. Box 44–306, Miami, Fla.

JOURNAL OF OCCUPATIONAL MEDICINE. Hoeber Medical Division of Harper & Row, 2 Park Avenue, New York, N.Y.

LA MEDICINA DEL LAVORO. Via S. Barnaba 8, Milan, Italy

TRANSACTIONS OF THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS. Association of Industrial Medical Officers, 47 Lincoln's Inn Fields, London, England

ZEITSCHRIFT FÜR UNFALLMEDIZIN UND BERUFSKRANKHEITEN. Buchdruckerei Berichthaus, Zwingliplatz 3, Zürich, Switzerland

ZENTRALBLATT FÜR ARBEITSMEDIZIN UND ARBEITSSCHUTZ. Verlag von Dr. Dietrich Steinkopff Holzhofallee 35, Darmstadt, Germany

Bibliographies

COUNCIL ON OCCUPATIONAL HEALTH, AMERICAN MEDICAL ASSOCIATION: (1) Selected publications on occupational health. (2) Publications of the Council on Occupational Health. The Association, Chicago, 1962.

HYSLOP, F. L. AND GAFAFER, W. M.: Bibliography of occupational health; occupational health and related publications from the Public Health Service, 1909–1953. Pub. Health Bibliography Series No. 9. Pub. Health Service Pub. No. 300. U.S. Government Printing Office, Washington, D.C., 1954. Processed supplements are periodically issued.

MOCK, H. E.: *Industrial Medicine and Surgery.* W. B. Saunders Co., Philadelphia, 1920. A bibliography covering 1916–1919 appears on pages 801–824; lists sources from which "a very complete bibliography" can be obtained on industrial medicine and surgery prior to 1916.

OCCUPATIONAL HEALTH BOOKSHELF. Selected references as recommended by the Industrial Medical Association and the U.S. Public Health Service. *J. Occup. Med.* 5: 150, 1963.

STRATTON, H. J.: List of publications Issued by the Bureau of Mines from July 1, 1910 to January 1, 1960; with Subject and Author Index. Bureau of Mines Special Publications. U.S. Government Printing Office, Washington, D.C., 1960.

Texts, Since 1943 (See also, General References, Chemical Hazards Section)

ANON.: Guiding notes for the notification of occupational diseases. *Occup. Safety & Health* (International Labor Office) 5: 171, 1955; 6: 5, 1956.

BROWN, M. L. AND MEIGS, J. W.: *Occupational Health Nursing.* Springer Publishing Co., New York, 1956.

BUREAU OF MINES, DEPARTMENT OF THE INTERIOR: *Minerals Yearbook.* Vol. 1, Metals and Minerals (Except Fuels); vol. 2, Fuels. U.S. Government Printing Office, Washington, D.C., annually. Includes material on production, consumption and uses.

CHIEF INSPECTOR OF FACTORIES: Report on industrial health. Her Majesty's Stationery Office, London, annually.

DAVIES, T. A. L.: *The Practice of Industrial Medicine.* 2nd ed. J. &. A. Churchill, London, 1957.

DEICHMANN, W. B. AND GERARDE, H. W.: *Signs, Symptoms and Treatment of Certain Acute Intoxications.* 2nd ed. Charles C. Thomas, Springfield, Ill., 1958.

DREISBACH, R. H.: *Handbook of Poisoning; Diagnosis and Treatment.* 3rd ed. Lange Medical Publications, Los Altos, Calif., 1961.

ECKARDT, R. E.: *Industrial Carcinogens.* Grune & Stratton, New York, 1959.

GONZALES, T. A.; VANCE, M.; HELPERN, M., AND UMBERGER, C. J.: *Legal Medicine; Pathology and Toxicology.* 2nd ed. Appleton-Century-Crofts, New York, 1954.

INDUSTRIAL HEALTH DIVISION, DEPARTMENT OF NATIONAL HEALTH AND WELFARE, AND DIVISION OF INDUSTRAL HYGIENE, DEPARTMENT OF HEALTH FOR ONTARIO: *A Guide to the Diagnosis of Occupational Diseases.* King's Printer and Controller of Stationery, Ottawa, Canada, 1949.

JACOBS, M. B.: *The Analytical Chemistry of Industrial Poisons, Hazards, and Solvents.* Interscience Publishers, New York, 1949.

LANZA, A. J. (EDITOR): *The Pneumoconioses.* Grune & Stratton, New York, 1963.

LEVINE, B. S. (TRANSLATOR AND EDITOR): *U.S.S.R. Literature on Air Pollution and Related Occupational Diseases;* a Survey. 3 vols., Nos. 60–21049, 60–21188 and 60–21475. Office of Technical Services, U.S. Department of Commerce, Washington, D.C., 1960.

MAISEL, A. Q. (EDITOR): *The Health of People Who Work.* The National Health Council, New York, 1960. Based upon papers presented at the 1959 National Health Forum.

MANUFACTURING CHEMISTS' ASSOCIATION: *Chemical Statistics Handbook* (formerly *Chemical Facts & Figures*) ; *Useful Information and Statistics Relating to the Chemicals and Allied Products Industries.* 5th ed. The Association, Washington, D.C., 1960. Supp. 1961.

MCCORD, C. P.: *A Blind Hog's Acorns; Vignettes of the Maladies of Workers.* Cloud, Chicago, 1945.

MCGEE, L. C.: *Manual of Industrial Medicine.* 3rd ed. University of Pennsylvania Press, Philadelphia, 1956.

MEREWETHER, E. R. A. (EDITOR): *Industrial Medicine and Hygiene.* 3 vols. Butterworth & Co., London, 1954–1956.

NATIONAL ASSOCIATION OF MUTUAL CASUALTY COMPANIES: *Handbook of Organic Industrial Solvents.* 2nd ed. The Association, Chicago, 1961.

SCHEFLAN, L. AND JACOBS, M. B.: *The Handbook of Solvents.* D. Van Nostrand Co., New York, 1953.

SHEPARD, W. P.: *The Physician in Industry.* McGraw-Hill Book Co., New York, 1961.

SCHILLING, R. S. F. (EDITOR): *Modern Trends in Occupational Health.* Butterworth & Co., London, 1960.

TELEKY, L.: *History of Factory and Mine Hygiene.* Columbia University Press, New York, 1948.

U.S. NATIONAL HEALTH SURVEY: Preliminary report on disability, United States, July-September 1957, Series B–4; Disability days, United States, July 1957–June 1958, Series B–10; Disability days, United States, July 1959–June 1960, Series B–29; Currently employed persons, illness and work-loss days, United States, July 1959–June 1960, Series C–7. Public Health Service; U.S. Department of Health, Education, and Welfare; Washington, D.C.; 1958, 1959, 1961, 1962. The U.S. National Health Survey is a continuing program under which the Public Health Service makes studies to determine the extent of illness and disability in the population of the United States and to gather

related information. The publications issued under the general designation, Public Health Service Publication No. 584, appeared in four lettered series (A, B, C and D), each separately numbered. The areas covered by the series were program descriptions, survey designs, concepts and definitions (A); health interview survey results by topics (B); health interview survey results for population groups (C), and developmental and evaluation reports (D).

In the spring of 1963, Publication No. 584 (Health Statistics from the U.S. National Health Survey) was discontinued. Its four series (A, B, C and D) were incorporated into Public Health Service Publication No. 1000 (Vital and Health Statistics) with the use of numbers rather than letters. Under this publication plan, Series A is included in Series 1 (statistical programs and collection methods), Series B and C are included in Series 10 (interview survey results), and Series D is included under Series 2 (research in statistical method). An updated version of the former Series A–1, Origin and Program of the U.S. National Health Survey, will be published in the new Series 1. An article describing the National Health Survey programs was published in the February 1963 issue of the journal, *New Medical Materia* (pp. 60–61). Of occupational health interest is the first publication to appear under the new plan. The title is Acute conditions, incidence and associated disability, United States, July 1961–June 1962; identified as Series 10, Number 1, and dated May 1963.

U.S. TARIFF COMMISSION: Synthetic Organic Chemicals; United States Production and Sales. U.S. Government Printing Office, Washington, D.C., annually. Includes a directory of manufacturers.

VON OETTINGEN, W. F.: The aliphatic acids and their esters; toxicity and potential dangers. *A.M.A. Arch. Indust. Health* 20: 517, 1959; 21: 28, 100 and 404, 1960.

ZIMMERMAN, O. T. AND LAVINE, I.: *Handbook of Material Trade Names.* Industrial Research Service, Dover, N.H., 1953. Supp. 1, 1956. Supp. 2, 1957. Supp. 3, 1960.

Texts, 1943 or Earlier

ARLIDGE, J. T.: *The Hygiene, Diseases and Mortality of Occupations.* Percival & Co., London, 1892. Outgrowth of Milroy Lectures of 1889.

BRIGHAM, REV. C. H.: The influence of occupations upon health; an essay read at the quarterly meeting of the State Board of Health in Michigan at Lansing, October 12th, 1875. In *Third Annual Report of the Secretary of State Board of Health of the State of Michigan for the Fiscal Year Ending September 30th, 1875.* W. S. George & Co., Lansing, 1876. Pp. 39–54. A layman's account of the physical, chemical, biologic, mental and emotional hazards of the work environment with some suggestions on prevention and control. In his discussion of whether a disease is occupationally derived the author raises the question, "Who can say what proportion of the malady comes from the kind of work, and what proportion from other sources, bad surroundings, bad air, bad constitution, bad personal habits?" It is emphasized that, "one is never in more danger physically or spiritually than when his permanent occupation is no occupation, only in killing time."

BUREAU OF LABOR STATISTICS, U. S. DEPARTMENT OF LABOR: Hours, fatigue, and health in British munition factories; Bull. No. 221. Welfare work in British munition factories; Bull. No. 222. Employment of women and juveniles in Great Britain during the war; Bull. No. 223. Industrial efficiency and fatigue in British munition factories; Bull. No. 230. U.S. Government Printing Office, Washington, D.C., 1917. The final report, Industrial health and efficiency, Bull. No. 249, appeared in 1919. The five bulletins are reprints of memoranda of the British Health of Munition Workers Committee published at the request of the U.S. Council of National Defense. The bulletins contain material of importance in a number of areas including hours of work, industrial fatigue, and occupational diseases.

COLLIER, H. E.: *Outlines of Industrial Medical Practice.* Williams & Wilkins Co., Baltimore, 1941.

COLLIS, E. L. AND GREENWOOD, M.: *The Health of the Industrial Worker.* J. & A. Churchill, London, 1921.

FAY, A. H.: A Glossary of the Mining and Mineral Industry. Bureau of Mines, Department of the Interior, Bull. No. 95. U.S. Government Printing Office, Washington, D.C., 1920. A 754-page glossary, defining 20,000 words and terms used in geology, mining, chemistry, and metallurgy, including localisms, provincialisms, and obsolete words. Reprinted, 1947.

GAFAFER, W. M. (EDITOR): *Manual of Industrial Hygiene and Medical Service in War Industries.* Issued under the auspices of the Committee on Industrial Medicine of the Division of Medical Sciences of the National Research Council; prepared by the Division of Industrial Hygiene, National Institute of Health, U.S. Public Health Service. W. B. Saunders Co., Philadelphia, 1943. Spanish translation by M. Charnes. Pub. No. 213. Pan American Sanitary Bureau, Washington, D.C., 1946.

GOLDBERG, R. W.: *Occupational Diseases in Relation to Compensation and Health Insurance.* Columbia University Press, New York, 1931.

HAMILTON, A.: *Industrial Poisons in the United States.* Macmillan Co., New York, 1925.

HAMILTON, A.: *Exploring the Dangerous Trades.* Little, Brown and Co., Boston, 1943. An autobiography.

HAYHURST, E. R.: *A Survey of Industrial Health Hazards and Occupational Diseases in Ohio, February 1915.* F. J. Heer Printing Co., Columbus, 1915. It is stated in the Survey (page 1) that ". . . in the First Annual Report of the Ohio State Board of Health, 1886, page 10, we read that the president (of the board) appointed a standing committee upon Hygiene of Occupations and Railway Sanitation."

HAYHURST, E. R.: The industrial hygiene section (of the American Public Health Association), 1914–1934. *Am. J. Pub. Health* 24: 1039, 1934. Early history, books, papers, problems and personalities.

HENDERSON, Y. AND HAGGARD, H. W.: *Noxious Gases and the Principles of Respiration Influencing their Action.* 2nd ed. Reinhold Publishing Corp., New York, 1943.

HOPE, E. W.; HANNA, W., AND STALLYBRASS, C. O.: *Industrial Hygiene and Medicine.* Baillière, Tindall and Cox, London, 1923.

HUEPER, W. C.: *Occupational Tumors and Allied Diseases.* Charles C. Thomas, Springfield, Ill., 1942.

KOBER, G. M. AND HANSON, W. C. (EDITORS): *Diseases of Occupation and Vocational Hygiene.* P. Blakiston's Son and Co., Philadelphia, 1916.

KOBER, G. M. AND HAYHURST, E. R. (EDITORS): *Industrial Health.* P. Blakiston's Son & Co., Philadelphia, 1924.

LANZA, A. AND GOLDBERG, J. A.: *Industrial Hygiene.* Oxford University Press, New York, 1939.

LEGGE, R. T.: Progress of American industrial medicine in the first half of the twentieth century. *Am. J. Pub. Health* 42: 905, 1952. Historical review of industrial medicine as witnessed by the author.

LEGGE, T. M.: *Industrial Maladies.* Oxford University Press, London, 1934. Posthumously published, edited by S. A. Henry.

LEHMANN, K. B. AND FLURY, F. (EDITORS); KING, E. AND SMYTH, H. F., JR. (TRANSLATORS): *Toxicology and Hygiene of Industrial Solvents.* Williams & Wilkins Co., Baltimore, 1943.

LÉVY, M.: *Traité d'hygiène publique et privée.* Baillière, Paris, 1844. 5th ed. 1869.

LLOYD, J. H.: The diseases of occupations. *In* Stedman, T. L. (editor): *Twentieth Century Practice, an International Encyclopedia of Modern Medical Science by Leading*

Authorities of Europe and America. Vol. 3. William Wood and Company, New York, 1895. Of interest is the author's concept of a "disease of occupation." This is disclosed in his decision to limit his paper "to a description of those injurious effects or diseases that are direct, characteristic, and indisputable." With the adoption of this view he declares that "the bronchial catarrh of a street car driver, the anemia of many ill-paid artisans, which is caused rather by poor food, and bad lodgings than by the pursuit of their trade, or the tuberculosis of silk weavers, will not be described. To describe all such affections, which may be due to other causes, such as poor hygiene, vicious habits, infectious processes, and even heredity, and to strain a point to ascribe them to the various trades, would stretch this paper beyond all reasonable limits. Yet there is a temptation to pursue this plan, and it has been pursued by not a few writers" (page 318). On another occasion (page 314) in referring to Lévy's *Treatise on Hygiene,* 1844, Lloyd states that "this author was apparently the first to draw a distinction between the evil effects of a mechanic's vices and those of his trade." Lévy is quoted as follows, "It is unjust to stigmatize the trades . . . by an enumeration of the vices and the bad traits which are observed in those who practice them; it is not proved that their corruption surpasses that of other classes of society." "This important distinction," wrote Lloyd, "is observed by Lévy with more care and consistency than by some more recent writers who might have profited by his example." Lloyd refers to some texts not found in the *Index-Catalogue.*

M'READY, B. W.: On the influence of trades, professions, and occupations in the United States in the production of disease; being the prize dissertation for 1837. *Trans. Med. Soc. State N.Y.* 3: 91, 1837. Reprinted, The Johns Hopkins Press, Baltimore, 1943.

OLIVER, T. (EDITOR): *Dangerous Trades; the Historical, Social, and Legal Aspects of Industrial Occupations as Affecting Health.* John Murray, London, 1902.

RAMBOUSEK, J. (LEGGE, T. M., TRANSLATOR): *Industrial Poisoning from Fumes, Gases and Poisons of Manufacturing Processes.* Edward Arnold, London, 1913.

SAPPINGTON, C. O. (EDITOR): Symposium on Industrial Health. *Clinics,* vol. 2, October 1943.

SAPPINGTON, C. O.: *Essentials of Industrial Health.* J. B. Lippincott Co., Philadelphia, 1943.

SELBY, C. D.: Studies of the medical and surgical care of industrial workers. Pub. Health Bull. No. 99. U.S. Government Printing Office, Washington, D.C., 1919.

SELBY, *C. D.* (EDITOR): Symposium on Industrial Medicine. *Med. Clinics N. Am.,* vol. 26, July 1942.

SYDENSTRICKER, E.: *Health and Environment.* McGraw-Hill Book Co., New York, 1933. Ch. 7, Occupational environment.

THACKRAH, C. T.: *The Effects of Arts, Trades, and Professions, and of Civic States and Habits of Living, on Health and Longevity; with Suggestions for the Removal of Many of the Agents which Produce Disease, and Shorten the Duration of Life.* 2nd ed., greatly enlarged. Longman, Rees, Orme, Brown, Green, & Longman; London, 1832. A biographic essay together with a photo-lithographic copy of this edition was prepared by A. Meiklejohn: *The Life, Work and Times of Charles Turner Thackrah, Surgeon and Apothecary of Leeds (1795–1833).* E. & S. Livingstone, Edinburgh, 1957.

THOMPSON, W. G.: *The Occupational Diseases; their Causation, Symptoms, Treatment and Prevention.* D. Appleton and Co., New York, 1914.

VARIOUS AUTHORS: *Occupation and Health; Encyclopaedia of Hygiene, Pathology and Social Welfare.* Vol. 1, A–H; vol. 2, I–Z. International Labour Office, Geneva, 1930 and 1934.

VARIOUS AUTHORS: *Report of Commission on Occupational Diseases to his Excellency, Governor Charles S. Deneen (of Illinois), January 1911.* Warner Printing Co., Chicago, no year. Among the investigators were Alice Hamilton and E. R. Hayhurst.

VERNON, H. M.: *Health in Relation to Occupation.* Oxford University Press, London, 1939.

WAMPLER, F. J. (EDITOR): *The Principles and Practice of Industrial Medicine.* Williams & Wilkins Co., Baltimore, 1943.

WARREN, B. S. AND SYDENSTRICKER, E.: Health insurance; its relation to public health. Pub. Health Bull. No. 76. U.S. Government Printing Office, Washington, D.C., 1916. Reference is made to occupational environmental conditions causing sickness among workers.

WOODBURY, R. M.: *Workers' Health and Safety; a Statistical Program.* Macmillan Co., New York, 1927.

• index

Index entries are primarily occupations and occupational disease agents. Included also are diseases, signs, and toxicologic terms. Numbers refer to pages.

218-695 O-66-22

218-695 O-66-23

218-695 O-66-24

U.S. GOVERNMENT PRINTING OFFICE : 1966 O—218-695

Edinburgh Health Dept
Aug 15 1967

Public Health Service Publication No. 1097