A Guide to the diagnosis of occupational diseases : a reference manual for physicians / compiled jointly by the staffs of the Industrial Health Division Department of National Health and Welfare, and the Division of Industrial Hygiene, Department of Health for Ontario.

GUIDE TO DIAGNOSIS OF OCCUPATIONAL DISEASES

INDUSTRIAL HEALTH DIVISION
DEPARTMENT NATIONAL HEALTH AND WELFARE, OTTAWA
AND
DIVISION OF INDUSTRIAL HYGIENE
DEPARTMENT OF HEALTH, ONTARIO

H. E. Griffiths

with sincere appreciation
of happy memories & associations
at Albert Dock Hospital - 1923

11/5/50

T. B. Carney

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DIAGNOSIS *of* OCCUPATIONAL DISEASES

A REFERENCE MANUAL FOR PHYSICIANS

Compiled Jointly by the Staffs of

THE INDUSTRIAL HEALTH DIVISION
DEPARTMENT OF NATIONAL HEALTH AND WELFARE

and

THE DIVISION OF INDUSTRIAL HYGIENE
DEPARTMENT OF HEALTH FOR ONTARIO

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PREFACE

This reference manual is intended to be of assistance to physicians in the diagnosis and prevention of occupational diseases. It has been prepared in response to the growing interest in industrial health problems on the part of the medical profession, labour and management.

The growth of Canadian industry in recent years has been extremely rapid. During the course of World War II, Canada became one of the foremost industrial nations of the world. Between 1939 and 1946, the value of our manufactured goods and foreign trade increased threefold while the number of employees in manufacturing alone rose from 658,000 to 1,119,000. Today, in Canada, the number of persons gainfully employed in all occupations is about 5,000,000.

Not only has there been an increase in the number of employees in our industries, but industry itself has become more complex and diversified. Many new materials and processes, introduced during the war, have now become standard features in Canadian industry and some of these constitute further sources of potential or actual health hazards for the worker. The control of the working environment and the supervision of workers' health call for a wider knowledge of the potential health hazards in all industries and for enhanced skill in recognizing occupational diseases.

A comprehensive program of industrial hygiene requires the combined services of professional and technical people from various scientific fields. In addition to the plant physician and nurse, the modern industrial hygiene team includes chemists, engineers, physicists, as well as factory inspectors, safety supervisors, plant managers, personnel directors, and first-aid men. All these people must have some knowledge of the broad field of industrial health in order to understand their special function in a preventive program.

The general practitioner and the specialist in various branches of medicine are also important members of this team when their patients are industrial workers. They, as well as the plant physician, therefore, should have a practical working knowledge of occupational factors in the causation of disease and the information assembled in this book is intended to be helpful in determining that relationship. **For this purpose, in Chapter 2, a reference key is presented**

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giving an alphabetical list of "Occupations" with their associated potential health hazards; and in Chapter 3 there follows a description of occupational diseases which may result from those hazards. Harmful chemicals are described under the following headings: Properties, Uses and Occurrence, Mode of Entry into the Body, Physiological Action and Toxicity, Signs and Symptoms.

The remaining chapters are of more general interest, Chapter I providing a brief discussion of occupational diseases, Chapter IV a review of occupational dermatoses and Chapter V a summary of the various provincial workmen's compensation acts.

This publication is intended to present only a brief summary of current knowledge of the diagnostic features relevant to occupational diseases. The selection and arranging of the material, together with the writing of accompanying text, have been performed largely by Dr. R. B. Sutherland of the Ontario Division of Industrial Hygiene and Dr. E. A. Watkinson and H. N. Acker, B.A., of the Federal Division of Industrial Health. Much of the material has been selected from works listed in the bibliography. Acknowledgment is also made to the United States Department of Labor, whose bulletin No. 41, "Occupation Hazards and Diagnostic Signs", has served as the basis for Chapter II. The values given for maximum allowable concentrations are those generally accepted today, but of course, are subject to change when further scientific knowledge warrants revision.

It is hoped that this book will fill, at least in part, a long felt need for a ready reference manual on the diagnosis of occupational diseases.

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CHAPTER I

THE OCCUPATIONAL DISEASES

I. General Considerations

Since occupational diseases have their origin in the working environment, their prevention, diagnosis and treatment depend upon a knowledge of the health hazards associated with any particular environment. In respect to patients who are industrial workers, physicians will want to know the materials and the processes with which their patients have been dealing and whether these have involved an exposure to any potential or actual health hazard. While it is often impractical for the general practitioner to know the whole industrial field, he can, however, become familiar with the working environment associated with the industries in his own community.

Early diagnosis of an occupational disease is especially important, for until the cause is determined, other workers may continue to be exposed to the same hazard. A knowledge of the potential hazards and the early signs and symptoms of abnormal exposure will assist in establishing an early diagnosis. Through close observation too, the industrial physician may notice significant minor changes among a working group. For example, pallor, loss of appetite and general weakness occurring among several workers in a storage battery plant should suggest group exposure to dangerous concentrations of lead.

STEPS IN DIAGNOSIS:

In the diagnosis of occupational disease it is important to prove exposure to the toxic substance, absorption of the substance into the body and to demonstrate actual signs of ill health caused by absorption. Most industrial diseases are insidious in their onset. Although they usually develop while the workman is still exposed to the causative agent, some diseases such as silicosis, radium poisoning and benzol poisoning, may not become evident for months or even years after the workman has left the hazardous environment. In no other branch of medicine is the work history of the patient more important. Not only is a complete medical and personal history essential, but a detailed history of all occupations held by the workman since the time he left school is imperative.

Investigation of industrial materials must be thorough. It should be remembered that mixtures, rather than simple substances are often used, particularly in the case of solvents. Such materials may contain harmful impurities or, without the trade name being changed, their composition may be altered in such a way as to become hazardous. Consequently, it is often necessary to contact or visit the plant to identify materials used by the workman and to consult the manufacturer of trade products to ascertain their chemical composition. In certain provinces manufacturers may be required to inform departments of labour or health concerning the composition of their products.

The physician must then determine whether any of these materials are known to be harmful. If the patient's exposure to a specific toxic substance is responsible for his illness, the clinical picture should be consistent with those of other known cases of occupational disease produced by similar agents. However, it should be borne in mind that the workman is often exposed to a mixture of toxic substances, a fact which may produce a complex and confused clinical picture.

It must be established whether the exposure, in terms of concentration and duration, has been sufficient to produce toxic effects in the workman. Exposure may be continuous or intermittent and there may be great variations in concentration during the working day. These factors influence the degree of danger associated with industrial poisons. For example, daily exposures to low concentrations of benzol over a considerable period of time, are likely to result in severe changes in the haemopoietic system; on the other hand, brief exposures to high concentrations, short of narcosis, usually have no serious effect on the body provided they are not repeated. In contrast, a single exposure to a high concentration of nitrous fumes may produce pulmonary oedema within eight to twenty-four hours. Thus the conditions under which a toxic material is used may have a direct bearing on the degree of hazard involved.

Recurrence of an illness, coinciding with the workman's return to a similar previous exposure, is further evidence supporting a diagnosis of occupational disease. This applies particularly to occupational dermatoses which usually show improvement when the man is removed from the causative exposure, and which recur upon his return to former work.

The finding of similar cases of illness among several persons subject to the same working conditions may be an important point suggesting an occupational origin of the disease. However, not all similarly exposed personnel need be affected. As in non-industrial diseases, there is marked variation in individual susceptibility. In addition, there appears to be a relationship between the age, sex and race of the individual and his susceptibility to poisoning by certain chemicals. Alcoholism and previous liver damage from any cause in a worker are believed to increase his susceptibility to the effects of T.N.T. and the chlorinated hydrocarbons.

The diagnosis of an occupational disease is often difficult to establish. It may necessitate an environmental survey including the collection and examination of dusts, fumes, vapours and gases, for the purpose of determining whether the worker has been exposed to toxic substances or hazardous conditions. The medical examination may involve the determination of the poison or its excretion products in body fluids or tissues. For example, urinalysis may reveal the presence of arsenic, fluorides, lead, manganese, mercury or selenium, or the excretory products of aniline, benzol, nitriles, phenol, toluene, T.N.T. or xylidine. Blood examination may show abnormal amounts of arsenic, carbon monoxide, fluorides, lead or manganese. Analysis of hair and finger nail clippings may reveal the presence of arsenic and the analysis of exhaled air may indicate the absorption of radioactive substances.

Usually, the general practitioner does not maintain facilities for making all these investigations. *When he requires assistance either in investigating an exposure or in making diagnostic tests, he may obtain such essential help from his provincial department of health. Special divisions of industrial hygiene exist in Manitoba, Ontario, Quebec and Nova Scotia under the provincial departments of health. These agencies can also frequently supply library facilities and information concerning the materials contained in trade products. The assistance of these divisions is available to all members of the medical profession and to industrial plants.*

Classification of Occupational Health Hazards

The harmful conditions and substances encountered in industry may be divided into the following main groups:

A. Abnormalities of Air Pressure.
B. Abnormalities of Temperature and Humidity.
C. Dampness.
D. Defective Illumination.
E. Excessive Noise.
F. Radiant Energy.
G. Electrical Burns; Electrical Shock.
H. Repeated Motion and Pressure; Vibration.
I. Infections.
J. Dust sand fumes.
K. Harmful Chemicals.

Many of the above groups are further subdivided into specific individual hazards which are listed in the index.

In Chapter III the health hazards related to occupation are discussed in order of the above classification. An attempt has been made, in the description of each hazard, to stress those points which will assist the physician in arriving at a diagnosis. Thus, certain physical properties of each hazard have been given, *e.g.*, Boiling Point, Vapour Density and where possible, Volatility. It is hoped that the information given under these headings will assist the physician in assessing the workman's degree of exposure. Under "Physiological Action and Toxicity", the body systems usually affected are indicated and where known, the action of various concentrations of each harmful chemical is given. The paragraph on "Signs and Symptoms" gives a brief description of the common clinical findings. "Maximum Allowable Concentrations", as presently accepted by most authorities, are stated. Mention is made of the fire hazard associated with most of the chemicals, since it is felt that many physicians, especially those connected with industry, can be of assistance to management in pointing out instances where a fire hazard and its consequent health hazard may exist.

2. Definitions

Certain terms frequently used in this book may be defined as follows:

DUSTS: Solid particles generated by handling, crushing, grinding, rapid impact, detonation and decrepitation of organic or inorganic materials such as rock, ore, metal, coal, wood, grain, etc. Dusts do

not tend to flocculate except under electrostatic forces; they do not diffuse in air but settle under the influence of gravity. (American Standards Association)

FUMES: Solid particles generated by condensation from the gaseous state, generally after volatilization from molten metals, etc., and often accompanied by a chemical reaction such as oxidation. Fumes flocculate and sometimes coalesce. (ASA)

MISTS: Suspended liquid droplets generated by condensation from the gaseous to the liquid state or by breaking up a liquid into a dispersed state, such as by splashing, foaming, and atomizing. (ASA)

VAPOURS: The gaseous form of substances which are normally in the solid or liquid state and which can be changed to these states either by increasing the pressure or decreasing the temperature alone. Vapours diffuse. (ASA)

GASES: Normally formless fluids which occupy the space of enclosure and which can be changed to the liquid or solid state only by the combined effect of increased pressure and decreased temperature. Gases diffuse. (ASA)

SPECIFIC GRAVITY: This is the density or weight of a liquid or solid in relation to that of water. When the liquid or solid is heavier than water, the specific gravity is greater than 1; when the substance is lighter than water, the specific gravity is less than 1.

BOILING POINT: This is the temperature at which the vapour pressure of a liquid is equal to the atmospheric pressure. In general, the lower the boiling point of a liquid, the greater its vapour pressure at room temperature.

VOLATILITY: This term is used to express the rate of evaporation of a liquid. Usually, the lower the boiling point of a liquid the more rapidly will it evaporate at room temperature.

VAPOUR DENSITY: This is the density or weight of a gas or vapour in relation to that of air. When the gas or vapour is heavier than air, the vapour density is greater than 1; when the substance is lighter than air the vapour density is less than 1. In still air, heavier gases and vapours tend to settle to lower levels, as for example, in pits and wells but a portion is diffused in the air at all levels.

MAXIMUM ALLOWABLE CONCENTRATION (M.A.C.): This is the highest concentration of an atmospheric contaminant which is regarded as having no harmful effect upon the health of an individual exposed continuously during the working day (8 hour daily exposure) and for prolonged periods of employment. These concentrations are usually expressed in one of three ways:

(1) In the case of gases and vapours, the M.A.C. is usually given in parts per million (ppm), that is, as parts of the gas or vapour per million parts of air. It must be remembered that the "parts of gas or vapour" refer to the gas-volume of the substance (*i.e.*, the volume of the substance in its gaseous state) and must not be confused by estimating the liquid volume of the substance in a million parts of air. To illustrate, the volume occupied by a gram-molecular weight of a substance at 0° Centrigade and 760 mm. of mercury pressure (Normal Temperature and Pressure) is 22·4 litres when in the gaseous state. The molecular weight of carbon tetrachloride (CCl_4) is 154, (carbon 12, chlorine 35·5) and 154 grams of CCl_4, at N.T.P., occupies 22·4 litres when completely volatilized. The liquid volume, on the other hand, is only 96 cc., or 0·096 litres. In this example, the gas volume is $22 \cdot 4 \div 0 \cdot 096$, or 233 times the liquid volume, and if the concentration of carbon tetrachloride in air in a given exposure were wrongly calculated using the liquid volume, the result would be 233 times lower than the true concentration.

(2) In the case of fumes, dusts and mists, the M.A.C. is usually expressed as milligrammes of contaminant per cubic metre of air (or Mgm. cu. metre, or Mgm/M^3). For the most part, these concentrations are expressed as milligrammes per 10 cubic metres of air, since the volume of air breathed by the average man in 8 hours while doing moderately hard work is about 10 cubic metres. The M.A.C., given as milligrammes per 10 cubic metres of air, therefore, indicates that concentration of contaminant in air which is regarded as safe and relates it to the average amount of air breathed by the workman during a working day.

(3) In the case of mineral dusts, such as asbestos and dusts containing free silica, the M.A.C. is expressed in millions of particles per cubic foot of air (M.P.P.C.F.), the dust count being performed by the standard light field technique.

In the case of gamma rays from X-rays or radium, the recommended limits of exposure are expressed as roentgen units per 8 hour

day. Where the radioactive material is gaseous, such as radon, the M.A.C. is given as curies per cubic metre of air.

It should be noted that some recommended M.A.Cs. have been determined by actual study of exposures associated with the development of clinical cases in industry. In other instances, where hazardous materials have been recently introduced into industry, the M.A.Cs. have been determined largely through animal experiments and it should be realized that results thus obtained may not be directly referable to man. Therefore, as more industrial experience and clinical evidence become available, it may be considered advisable to lower some limits which are now regarded as safe, or to raise others which are found to be lower than necessary.

FLASH POINT: This is the temperature at which a liquid or solid gives off sufficient vapour to support combustion. The term applies chiefly to liquids, but there are certain solids, such as camphor and naphthalene, which evaporate at ordinary room temperatures and have flash points while in the solid state.

A substance having a flash point above room temperature does not, of course, constitute as great a fire hazard as a substance having a flash point at or below room temperature. The lower the flash point, the more readily will a material give off sufficient vapour to support combustion. For purposes of classification, flammable liquids may be divided into four groups:

Class 1: Liquids having a flash point below 25°F.; *e.g.* ethyl (or anaesthetic) ether, gasoline, benzol.

Class 2: Liquids having a flash point above 25°F. and below 70°F.; *e.g.*, ethyl alcohol, methyl alcohol, toluol.

Class 3: Liquids having a flash point above 70°F. and below 200° F.; *e.g.*, kerosene, turpentine, xylol.

Class 4: Liquids having a flash point above 200°F.; *e.g.*, dichlorobenzene, ethylene glycol, paraffin wax.

EXPLOSIVE LIMITS (OR FLAMMABLE LIMITS): The minimum explosive limit is that concentration of a vapour in air which is required to support combustion. Vapour concentrations lower than the minimum explosive concentration will not burn. The maximum explosive limit is that concentration of vapour which still supports combustion, but beyond which there is insufficient oxygen to support the burning

of the vapour. The difference between the minimum and the maximum limits, representing concentrations in which combustion is supported, is known as the explosive range. Explosive limits are usually expressed as percentages by volume, of the gas or vapour in air. The minimum explosive limit is nearly always a higher concentration than that regarded as the maximum which is non-injurious to health (M.A.C.).

Chapter II

LIST OF OCCUPATIONS AND THEIR POTENTIAL HAZARDS

Abrasives Workers
- Heat
- Inorganic dusts (free silica)
- Inorganic dusts (no free silica)
- Organic dusts

Acetaldehyde Workers
- Acetaldehyde
- Mercury and its compounds

Acetanilide Workers
- Amino compounds of benzol, toluol and xylol
- Aniline

Acetic Acid Makers
- Hydrochloric acid
- Mercury and its compounds

Acetone Workers
- Acetone
- Mercury and its compounds

Acetylene Workers (see also carbide makers and welders)
- Acetone
- Ammonia
- Arsine
- Carbon disulphide
- Carbon monoxide
- Chloride of lime
- Chromium and its compounds
- Inorganic dust
- Phosphine

Acid Dippers
- Arsine
- Cyanides
- Dampness
- Hydrochloric acid
- Nitric acid
- Nitrous fumes
- Sulphuric acid

Acid Finishers (glass)
- Hydrochloric acid
- Lead and its compounds
- Sulphuric acid

Actors
- Lead and its compounds

Agricultural Workers (see farmers)

Air Hammer Operators
- Excessive noise
- Repeated motion, pressure and vibration

Aeroplane-Dope Workers
- Acetone
- Amyl acetate
- Benzol and its homologues (toluol and xylol)
- Butyl acetate
- Butyl alcohol
- Ethyl acetate
- Ethylene glycol monomethyl ether
- Formic acid
- Ketones
- Petroleum hydrocarbons

Aeroplane-Hangar Employees
- Alkalis
- Benzol and its homologues (toluol and xylol)
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Petroleum hydrocarbons

Aeroplane Pilots—Crop Dusting
- Altitude (rarefied air, decreased atmospheric pressure)
- Arsenic and its compounds
- Carbon monoxide
- Lead and its compounds

Alcohol Distillery Workers
- Amyl acetate
- Amyl alcohol
- Benzol and its homologues (toluol and xylol)
- Ethyl alcohol
- Formic acid
- Mercury and its compounds
- Methyl alcohol
- Propyl alcohol

Aldehyde Pumpmen
- Acetaldehyde
- Methyl alcohol

Alkali Salt Makers
- Carbon dioxide
- Chlorine
- Dampness
- Hydrochloric acid
- Hydrogen sulphide
- Sulphur dioxide

Alloy Makers
- Antimony
- Arsenic
- Beryllium
- Cadmium
- Carbon monoxide
- Chromium
- Cobalt
- Copper
- Heat
- Inorganic dust (free silica)
- Lead
- Magnesium
- Manganese
- Metal fume fever
- Nickel
- Selenium
- Tellurium
- Tin
- Vanadium
- Zinc

Aluminum Extractors
- Alkalis
- Fluorine and its compounds

Alum Workers
- Sulphuric acid

Amalgam Workers
- Mercury and its compounds

Ammonia Workers
- Ammonia
- Calcium cyanamide
- Carbon monoxide

Ammonium Salt Makers
- Ammonia
- Carbon disulphide
- Cyanides
- Heat
- Hydrochloric acid
- Nitric acid
- Sulphuric acid

Amyl Acetate Workers
- Amyl acetate
- Amyl alcohol

Amyl Alcohol Workers
- Amyl alcohol

Aniline Dye Makers
- (see dye makers)

Aniline Workers
- Amino compounds of benzol, toluol and xylol
- Aniline
- Arsine
- Benzol and its homologues (toluol and xylol)
- Chromium and its compounds
- Hydrochloric acid
- Nitric acid
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol
- Nitrous fumes

Animal By-Products Workers
- Acrolein
- Anthrax
- Dampness
- Erysipeloid
- Fungus infections
- Septic infections
- Undulant fever
- Weil's disease

Animal Hair Dressers
(see hair workers)

Animal Handlers
Anthrax
Fungus infections
Glanders
Rabies
Septic infections
Undulant fever

Annealers
Ammonia
Heat

Antifreeze Makers
Glycols
Methyl alcohol

Antimony Extractors (refiners)
Antimony and its compounds
Heat

Antimony Fluoride Extractors
Hydrofluoric acid

Arsenic Roasters
Arsenic and its compounds
Heat

Art-Glass Workers
Amyl acetate
Arsenic and its compounds
Cobalt and its compounds
Hydrofluoric acid
Lead and compounds
Manganese and its compounds
Methyl alcohol
Petroleum hydrocarbons
Turpentine

Artificial Flower Makers
Arsenic and compounds
Chromium and compounds
Lead and compounds
Mercury and compounds
Methyl alcohol
Repeated motion, pressure and vibration

Artificial Gem Makers
Thallium and its compounds

Artificial Ice Makers
Ammonia
Carbon dioxide
Dampness
Sudden variations of temperature
Sulphur dioxide

Artificial Leather Makers
Acetone
Amino compounds of benzol, toluol and xylol
Amyl acetate
Aniline
Arsenic and compounds
Benzol and its homologues (toluol and xylol)
Butanone
Butyl alcohol
Heat
Methyl alcohol
Nitric acid
Nitrous fumes
Sulphuric acid

Artificial Pearl Makers
Acetone
Amyl acetate
Lead
Nitric acid
Nitrous fumes
Tetrachloroethane

Artificial Rubber Makers
(see rubber makers, synthetic)

Artificial Silk Makers
(see rayon)

Artificial Stone Makers
Inorganic dust (free silica)
Inorganic dust (no free silica)
Tar and pitch

Asbestos Miners
(see also miners)
Asbestos dust

Asbestos Products Workers
- Asbestos dust
- Benzol and its homologues (toluol and xylol)
- Formaldehyde
- Heat
- Tar and pitch

Ashmen
- Inorganic dust (no free silica)
- Organic dust

Asphalt Workers
- Heat
- Tar and pitch

Automobile Mechanics (see garage workers)

Automobile Painters (see also painters)
- Amyl acetate
- Benzol and its homologues (toluol and xylol)
- Chlorinated hydrocarbons
- Dampness
- Ethyl acetate
- Ketones
- Methyl alcohol
- Petroleum hydrocarbons

Babbitt Metal Workers
- Antimony and its compounds
- Lead and its compounds

Babbitters
- Antimony and its compounds
- Lead and its compounds

Bakers
- Carbon dioxide
- Carbon monoxide
- Organic dust
- Sudden variations of temperature
- Ultraviolet and infrared rays

Baking Powder Makers
- Carbon dioxide
- Inorganic dust (no free silica)
- Organic dust

Barbers
- Fungus infections
- Repeated motion, pressure and vibration

Barium Carbonate Makers
- Barium and its compounds
- Hydrogen sulphide

Bar-Mill Workers (iron and steel)
- Heat

Barometer Makers
- Mercury and its compounds

Basic Slag (artificial manure) **Handlers**
- Inorganic dust (no free silica)

Batch Makers (glass works) (see glass mixers)

Batch Makers (rubber works) (see compounders, rubber)

Baters (tannery)
- Anthrax

Battery (dry) **Makers**
- Amyl acetate
- Benzol and its homologues (toluol and xylol)
- Chromium and its compounds
- Hydrochloric acid
- Inorganic dust (no free silica)
- Lead and its compounds
- Manganese and its compounds
- Mercury and its compounds
- Organic dust
- Tar and pitch
- Zinc and its compounds

Battery (storage) **Makers** (see storage battery makers)

Beamers (textiles)
- Organic dust

Beam-House Workers (tannery)
- Anthrax
- Dampness

Beatermen (paper and pulp)
Chlorine
Dampness

Beauty Parlour Operators
Ammonium thioglycollate
Benzol and its homologues (toluol and xylol)
Fungus infections
Hydrogen sulphide

Bed Rubbers (stone)
Inorganic dust (free silica)
Inorganic dust (no free silica)

Benzene Workers (see benzol workers)

Benzine (petroleum) **Workers**
Aliphatic hydrocarbons
Petroleum hydrocarbons

Benzol Purifiers
Benzol and its homologues (toluol and xylol)
Sulphuric acid

Benzol-Stillmen
Benzol
Heat

Benzol Workers
Benzol

Bessemer-Converter Workers (iron and steel)
Carbon monoxide
Heat
Ultraviolet and infrared rays

Bevelers
Inorganic dust (no free silica)

Billet Mill Workers (iron and steel)
Heat

Bisque-Kiln Workers
Carbon monoxide
Heat
Inorganic dust (free silica)
Inorganic dust (no free silica)

Blacksmiths
Carbon dioxide
Carbon monoxide
Cyanides
Heat
Lead and its compounds
Repeated motion, pressure and vibration
Ultraviolet and infrared rays

Blanket Makers
Anthrax

Blasters
Carbon monoxide
Hydrogen sulphide
Inorganic dust (free silica)
Inorganic dust (no free silica)
Nitrous fumes

Blast Furnace Workers
Carbon dioxide
Carbon monoxide
Heat
Hydrocyanic acid
Hydrogen sulphide
Phosphine
Sulphur dioxide
Ultraviolet and infrared rays

Bleachers
Alkalis
Chloride of lime
Chlorine
Chromium compounds
Heat
Hydrochloric acid
Hydrofluoric acid
Nitric acid
Nitrous fumes
Oxalic acid
Ozone
Phosgene
Sulphur dioxide
Sudden variations of temperature

Bleaching Powder Makers
Arsine
Chloride of lime
Chlorine
Manganese compounds

Blenders (motor fuel)
(see gasoline blenders)

Blockers (felt hats)
Carbon monoxide
Heat

Blooders (tannery)
Lead and its compounds

Blooming-Mill Workers (iron and steel)
Heat

Blowers (glass manufacturing) (see glass blowers)

Blowers-Out (zinc smelting)
Heat
Metal fume fever
Zinc and its compounds

Blue Print Makers
Chromium compounds

Blue Print Paper Makers
Amino compounds of benzol, toluol and xylol
Aniline
Oxalic acid

Bluers (revolvers)
Heat

Boiler Cleaners and Washers
Carbon monoxide
Dampness

Boiler-Room Workers
Carbon dioxide
Carbon monoxide
Heat

Boneblack Makers
Ammonia
Phosphorus and its compounds

Bone Renderers, Extractors, etc.
Acrolein
Anthrax
Heat
Hydrocyanic acid
Organic dust
Sulphur dioxide

Bookbinders
Acrolein
Amyl acetate
Arsenic and its compounds
Lead and its compounds
Methyl alcohol
Oxalic acid

Bottle-Cap Makers
Lead and its compounds

Bottlers (mineral water)
Carbon dioxide
Hydrogen sulphide

Brake-Lining Makers
Asbestos dust
Benzol and its homologues (toluol and xylol)

Brass Founders
Antimony and its compounds
Arsenic and its compounds
Carbon dioxide
Carbon monoxide
Copper and its compounds
Heat
Inorganic dust (free silica)
Lead and its compounds
Metal fume fever
Phosphorus and its compounds
Sulphur dioxide
Ultraviolet and infrared rays
Zinc and its compounds

Brass Polishers (see also polishers and cleaners, metal)
Lead and its compounds

Braziers
Heat
Lead and its compounds
Ultraviolet and infrared rays
Zinc and its compounds

Brewers
Amyl alcohol
Carbon dioxide
Carbon monoxide

Dampness
Fungus infections
Formaldehyde
Heat
Hydrofluoric acid
Phenol
Sudden variations of temperature
Sulphuric acid

Brick Burners
Carbon dioxide
Carbon monoxide
Heat
Lead and its compounds

Brick Layers
Inorganic dust (no free silica)

Brick Makers
Dampness
Heat
Hydrofluoric acid
Inorganic dust (free silica)
Inorganic dust (no free silica)
Lead and its compounds
Magnesium and its compounds
Manganese and its compounds
Sulphur dioxide

Briquette Makers
Arsenic and its compounds
Tar and pitch

Bronze Powder Makers
Acetone
Lead and its compounds
Zinc and its compounds

Bronzers
Ammonia
Amyl acetate
Arsenic and its compounds
Arsine
Benzol
Cyanides
Hydrochloric acid
Hydrocyanic acid
Hydrogen sulphide
Inorganic dust (no free silica)
Lead and its compounds
Manganese and its compounds
Mercury and its compounds
Methyl alcohol
Petroleum hydrocarbons
Zinc and its compounds

Broom Makers
Anthrax
Chlorine
Formaldehyde
Organic dust
Sulphur dioxide

Browners (gun barrels)
Phosphoric acid

Brush Makers
Anthrax
Formaldehyde
Lead and its compounds
Methyl alcohol
Organic dust
Tar and pitch

Buffers
Defective illumination
Inorganic dust (free silica)
Inorganic dust (no free silica)
Organic dust

Buffers (rubber)
Amyl acetate
Ethyl acetate
Lead and its compounds
Petroleum hydrocarbons

Bulb (mercury) **Makers**
Mercury and its compounds

Buoy Makers
Phosphine

Burners (enameling)
Heat
Lead and its compounds

Burnishers (metals)
- Antimony and its compounds
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Defective illumination
- Petroleum hydrocarbons
- Sulphuric acid
- Trichloroethylene

Burrers (needles)
- Heat
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Burr Filers
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Butchers
- Anthrax
- Dampness
- Erysipeloid
- Glanders
- Septic infections
- Sudden variations of temperature
- Tularaemia
- Undulant fever
- Weil's disease

Button Makers
- Acetaldehyde
- Acetone
- Acids
- Chloride of lime
- Formaldehyde
- Fungus infections
- Inorganic dust (no free silica)
- Lead and its compounds
- Organic dust

Butyl Acetate Makers
- Butyl alcohol

Butyl Alcohol Makers
- Butyl alcohol

Cable Makers
- Lead and its compounds

Cable Splicers
- Acrolein
- Aliphatic hydrocarbons (methane, etc.)
- Carbon monoxide
- Cresol
- Dampness
- Electrical burns; electrical shock
- Hydrogen sulphide
- Lead and its compounds
- Petroleum hydrocarbons
- Phenol
- Poison ivy
- Turpentine

Cadmium Alloy Makers
- Cadmium and its compounds

Cadmium and Cadmium-Compound Makers
- Arsine
- Cadmium and its compounds

Cadmium Platers (see also electroplaters)
- Cadmium and its compounds

Caisson Workers
- Carbon dioxide
- Carbon monoxide
- Compressed air
- Dampness
- Defective illumination
- Hydrogen sulphide
- Sudden variations of temperature

Calcium Carbide Makers (see carbide makers)

Calcium Cyanamide Makers
- Calcium cyanamide
- Heat
- Inorganic dust (no free silica)

Calenderers (rubber)
- Inorganic dust (no free silica)
- Sudden variations of temperature

Calico Printers (see textile printers)

Candle Makers
- Acrolein
- Amino compounds of benzol, toluol and xylol
- Aniline
- Arsenic and its compounds
- Chromium and its compounds
- Sulphuric acid

Candy Makers
- Dermatitis
- Heat
- Sudden variations of temperature

Canners
- Arsenic and its compounds
- Carbon dioxide
- Dampness
- Heat
- Lead and its compounds
- Sudden variations of temperature
- Septic infections

Can Sealers
- Benzol and its homologues (toluol and xylol)

Cap Loaders
- Mercury and its compounds

Cappers (window glass)
- Heat

Carbide Makers
- Acetylene
- Ammonia
- Carbon monoxide
- Heat
- Inorganic dust (no free silica)
- Organic dust
- Ultraviolet and infrared rays

Carbolic Acid Makers
- Benzol and its homologues (toluol and xylol)
- Phenol
- Sulphur dioxide
- Sulphuric acid

Carbonated-Water Makers
- Carbon dioxide

Carbon Black Workers
- Heat
- Organic dust

Carbon Brush Makers
- Inorganic dust (no free silica)
- Organic dust

Carbon Dioxide Makers
- Carbon dioxide

Carbon Disulphide Makers
- Carbon disulphide
- Hydrogen sulphide

Carbonizers (shoddy)
- Arsine
- Hydrochloric acid
- Organic dust
- Sulphuric acid

Carbon Paper Makers
- Organic dust

Carbon Printers (photography)
- Chromium compounds

Carbon Tetrachloride Workers
- Carbon disulphide
- Carbon tetrachloride
- Phosgene
- Sulphur chloride

Carders (asbestos)
- Asbestos dust

Carders (textiles)
- Organic dust

Card Grinders (textile)
- Inorganic dust (no free silica)
- Organic dust

Carpenters
- Organic dust
- Repeated motion, pressure and vibration

Carpet Cleaners
- Anthrax
- Chlorinated hydrocarbons
- Organic dust
- Petroleum hydrocarbons

Carpet Makers
- Anthrax
- Arsenic and its compounds
- Organic dust

Cartridge Cup Washers
- Dampness

Cartridge Dippers
- Hydrochloric acid
- Nitric acid
- Nitrous fumes
- Sulphuric acid

Cartridge Felt and Wad Makers
- Dampness

Cartridge Cap Operators
- Lead and its compounds
- Mercury and its compounds

Cartridge Shot Shell Paraffin Dippers
- Dampness
- Sudden variations of temperature

Case Hardeners
- Cyanides
- Heat
- Hydrocyanic acid
- Ultra high frequency radiations

Casters (metal) (see foundry and also particular metal)

Casting Cleaners (foundry) (see also acid dippers)
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Cast Scrubbers (electroplaters)
- Benzol and its homologues (toluol and xylol)
- Petroleum hydrocarbons

Cattlemen
- Anthrax
- Undulant fever

Cellulose Makers
- Alkalis
- Dampness
- Hydrogen sulphide
- Sulphur dioxide
- Sulphuric acid

Cellulose Products Makers (see rayon, pyroxylin plastics, lacquers)

Cementers (rubber)
- Benzol and its homologues (toluol and xylol)
- Butyl alcohol
- Carbon disulphide
- Carbon tetrachloride
- Ethyl acetate
- Chlorinated hydrocarbons
- Methyl alcohol
- Petroleum hydrocarbons
- Tetrachloroethane
- Trichloroethylene
- Turpentine

Cement (Portland) **Workers**
- Carbon monoxide
- Heat
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Cement (rubber, plastic, etc.) **Mixers**
- Acetone
- Ammonia
- Amyl acetate
- Benzol and its homologues (toluol and xylol)
- Carbon disulphide
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Diethylene dioxide
- Ethyl acetate
- Lead and its compounds
- Petroleum hydrocarbons

Pyridine
Sulphur chloride
Tar and pitch
Tetrachloroethane

Ceramic Workers (see pottery workers)

Chambermen (sulphuric acid)
Sulphur dioxide
Sulphuric acid

Charcoal Burners
Carbon dioxide
Carbon monoxide
Organic dust

Charcoal Workers
Carbon monoxide
Organic dust

Charcoal Workers (sugar refining)
Heat
Sudden variations of temperature

Chargers (furnace) (see also particular metal)
Carbon monoxide
Heat
Inorganic dust (no free silica)

Chargers (smelting and refining) (see also particular metal)
Carbon monoxide
Heat
Inorganic dust (no free silica)

Chasers (steel)
Inorganic dust (free silica)
Inorganic dust (no free silica)

Chauffeurs
Carbon monoxide
Dampness
Petroleum hydrocarbons
Repeated pressure, motion and vibration
Sudden variations of temperature

Chemists (analytical)
Acetaldehyde
Acetone
Alkalis
Antimony and compounds
Arsenic and compounds
Benzol and its homologues (toluol and xylol)
Cadmium and its compounds
Carbon disulphide
Carbon monoxide
Carbon tetrachloride
Chlorinated hydrocarbons
Chlorine
Chromium and its compounds
Cyanides
Cyanogen and compounds
Formaldehyde
Hydrochloric acid
Hydrogen sulphide
Lead and its compounds
Mercury and its compounds
Methyl alcohol
Nitric acid
Nitrous fumes
Petroleum hydrocarbons
Phosphorus and its compounds
Sulphuric acid
Zinc and its compounds

Chemists (radium research)
X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.).

Chimney Masons
Carbon monoxide

Chimney Sweepers
Arsenic and its compounds
Carbon monoxide
Inorganic dust (no free silica)
Tar and pitch

Chippers
Inorganic dust (free silica)
Inorganic dust (no free silica)
Lead and its compounds

Chloride of Lime Makers
Chloride of lime
Chlorine

Chlorinated Diphenyl Workers
Chlorinated diphenyls

Chlorinated Naphthalene Workers
Chlorinated naphthalenes

Chlorine Makers
Chlorine
Hydrochloric acid
Manganese and its compounds
Mercury and its compounds

Chlorodiphenyl Users
Chlorinated diphenyls

Chloroform Makers
Acetone
Carbon tetrachloride
Chloride of lime
Methyl chloride

Chrome Workers
Chromium and its compounds

Chromium Platers (see also electroplaters)
Chromium and its compounds

Cigar Makers
Fungus infections
Lead and its compounds
Nicotine and tobacco
Organic dust

Clay and Bisque Makers (pottery)
Dampness
Inorganic dust (free silica)
Inorganic dust (no free silica)
Sudden variations of temperature

Clay-Plug Makers (pottery)
Dampness
Inorganic dust (no free silica)

Clay-Products Workers (see pottery workers)

Cleaners (metal) (see polishers and cleaners, metal)

Clerks
Defective illumination
Repeated motion, pressure and vibration

Clothes Pressers
Carbon monoxide
Heat

Cloth Singers
Carbon monoxide

Coal Carbonizers
Carbon monoxide
Hydrogen sulphide
Sulphur dioxide

Coal Miners
(see miners)

Coal Passers
Inorganic dust (no free silica)
Organic dust

Coal Tar Workers (see also coke oven workers)
Amino compounds of benzol, toluol and xylol
Aniline
Benzol and its homologues (toluol and xylol)
Carbon monoxide
Cresol
Cyanides
Cyanogen compounds
Heat
Phenol
Tar and pitch

Cobbers (asbestos)
Asbestos

Cobblers
Anthrax
Benzol and its homologues (toluol and xylol)
Carbon tetrachloride
Organic dust
Repeated motion, pressure and vibration

Coin Makers
- Nickel
- Silver

Coke-Oven Workers (see also coal tar workers)
- Ammonia
- Benzol and its homologues (toluol and xylol)
- Carbon monoxide
- Heat
- Hydrogen sulphide
- Sulphur dioxide
- Tar and pitch

Cold Storage Plant Workers (see refrigerating plant workers)

Collar (fused) **Makers**
- Acetone
- Ethylene glycol monomethyl ether
- Methyl alcohol

Collodion Makers
- Nitric acid
- Nitrous fumes

Coloured Paper Workers
- Arsenic and its compounds

Colourers (marble)
- Chromium compounds

Colourers (white) **of Shoes**
- Lead and its compounds

Colour Makers
- Amino compounds of benzol, toluol and xylol
- Ammonia
- Aniline
- Antimony and its compounds
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Brominated hydrocarbons (methyl bromide)
- Bromine
- Cadmium and its compounds
- Chlorinated hydrocarbons
- Chlorine
- Chromium and its compounds
- Cobalt and its compounds
- Dimethyl sulphate
- Ethylene chlorohydrin
- Heat
- Iodine
- Inorganic dust (no free silica)
- Lead and its compounds
- Manganese and its compounds
- Mercury and its compounds
- Methyl chloride
- Naphthols
- Petroleum hydrocarbons
- Selenium compounds
- Sulphuric acid
- Tetrachloroethane
- Thallium and its compounds

Comb Makers
- Acetone
- Ketones
- Organic dust

Compositors
- Alkalis
- Amino compounds of benzol, toluol and xylol
- Aniline
- Antimony and compounds
- Benzol and its homologues (toluol and xylol)
- Defective illumination
- Inorganic dust (no free silica)
- Lead and its compounds
- Petroleum hydrocarbons
- Repeated motion, pressure and vibration
- Turpentine

Compounders (rubber)
- Amino compounds of benzol, toluol and xylol
- Aniline
- Antimony and its compounds
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Chromium compounds

Hexamethylene tetramine
Inorganic dust (no free silica)
Lead and its compounds
Organic dust
Petroleum hydrocarbons

Compressed Air (caisson) **Workers**
(see caisson workers)

Compressed Air (pneumatic tool) **Workers**
(see pneumatic tool workers)

Concentrating Mill Workers (see also oil flotation plant workers)
Dampness
Inorganic dust (free silica)
Inorganic dust (no free silica)
Lead and its compounds
Manganese and its compounds
Selenium and its compounds

Confectioners (see candy makers)

Construction Workers
Dampness
Inorganic dust (free silica)
Inorganic dust (no free silica)

Cooks
Carbon monoxide
Erysipeloid
Septic infections
Sudden variations of temperature
Ultraviolet and infrared rays

Copper Founders
Arsenic and compounds
Copper and its compounds

Copper Miners (see miners)

Copper Refiners and Smelter Workers
Antimony and its compounds
Arsenic and its compounds
Carbon monoxide
Copper and its compounds
Heat
Hydrofluoric acid
Lead and its compounds
Manganese and its compounds
Selenium compounds
Sulphur dioxide
Tellurium and compounds

Coppersmiths
Arsenic and its compounds
Copper and its compounds

Copper (strip) **Roller-Mill Workers**
Acrolein

Cordage Factory Workers
Anthrax
Tar and pitch

Core Makers
Acrolein
Carbon monoxide
Carbon tetrachloride
Heat
Inorganic dust (free silica)
Inorganic dust (no free silica)
Zinc and its compounds

Cork Workers
Organic dust

Corn-Products Workers
Dampness
Heat
Sudden variations of temperature

Cosmetic Workers
Arsenic and its compounds
Ethylene glycol monomethyl ether
Mercury and its compounds
Nitrobenzol
Nitro compounds of benzol, toluol and xylol

Cotton Mill Workers
Byssinosis
Dampness
Heat
Inorganic dust (no free silica)
Organic dust

Cottonseed-Oil Workers
Heat

Cotton Twisters
Organic dust
Repeated motion, pressure and vibration

Cranemen (glass industry)
Heat

Cranemen (iron and steel)
Acrolein
Carbon monoxide
Heat
Inorganic dust (free silica)

Cranemen (non-ferrous)
Arsenic and compounds
Heat
Inorganic dust (free silica)
Lead and compounds
Metal fume fever
Selenium and compounds
Sulphur dioxide
Zinc and compounds

Crayon (coloured) **Makers**
Chromium and its compounds
Lead and its compounds

Creosoting-Plant Workers
Cresol
Dampness
Phenol
Tar and pitch

Cresol-Soap Makers
Cresol

Cresylic Acid Makers
Cresol

Crucible Mixers
Inorganic dust (no free silica)
Organic dust

Crucible-Steel-Department Employees
Heat

Crushermen (clay and stone)
Inorganic dust (free silica)
Inorganic dust (no free silica)

Crushers (asbestos)
Asbestos dust

Cupola Men (foundries)
Carbon dioxide
Carbon monoxide
Heat

Curers, Vapour (rubber) (see vulcanizers)

Curriers (tannery)
Anthrax
Arsenic and compounds
Organic dust
Petroleum hydrocarbons

Cut-Glass Workers
Arsenic and its compounds
Inorganic dust (no free silica)
Lead and its compounds

Cutlery Makers
Amyl acetate
Inorganic dust (free silica)
Inorganic dust (no free silica)
Lead and its compounds

Cutters (oxyacetylene and other gases) (see welders)

Cyanamide Makers
Calcium cyanamide
Heat
Inorganic dust (no free silica)

Cyanide Workers
Ammonia
Cyanides
Hydrocyanic acid

Cyanogen Makers
Cyanogen
Hydrogen sulphide
Mercury and its compounds

Dairy Workers
Anthrax
Undulant fever

Damascening Workers
- Nitric acid
- Nitrous fumes

Dancers
- Repeated motion, pressure and vibration

De-Brassers
- Nitric acid
- Nitrous fumes

Decorators (pottery)
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Lead and its compounds
- Mercury and its compounds
- Petroleum hydrocarbons
- Turpentine

Degreasers
- Benzol and its holomogues (toluol and xylol)
- Carbon disulphide
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Chlorinated naphthalenes
- Cycloparaffins
- Dichloroethyl ether
- Diethylene dioxide
- Ethylene dichloride
- Methylene chloride
- Petroleum hydrocarbons
- Tetrachloroethane
- Tetrachloroethylene
- Trichloroethylene

Denatured-Alcohol Workers (see particular denaturant)

Dental Workers
- Lead and its compounds
- Mercury and its compounds

Dentists
- Mercury and its compounds
- X-rays, radium and other radio-active substances

Depilatory Makers
- Barium and its compounds
- Thallium and its compounds

Detinning Workers
- Chlorine

Detonator Cleaners
- Mercury and its compounds

Detonator Fillers
- Mercury and its compounds

Detonator Packers
- Mercury and its compounds

Diamond Cutters
- Inorganic dust (no free silica)
- Organic dust
- Repeated motion, pressure and vibration

Diamond Polishers
- Lead and its compounds

Diatomaceous Earth Workers
- Inorganic dust (free silica)

Digester-House Workers (paper and pulp)
- Heat
- Hydrogen sulphide
- Sudden variations of temperature
- Sulphur dioxide

Dimethyl Sulphate Makers
- Arsine
- Dimethyl sulphate
- Methyl alcohol
- Nitric acid
- Nitrous fumes
- Sulphuric acid

Dinitrobenzol Workers
- Benzol
- Nitric acid
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol
- Nitrous fumes

Dinitrophenol Workers
- Dinitrophenol
- Nitric acid
- Nitrous fumes
- Phenol

Dioxan Users
- Diethylene dioxide

Dippers (gun cotton)
- Nitric acid
- Nitrous fumes

Dippers (rubber)
- Benzol and its homologues (toluol and xylol)
- Carbon tetrachloride
- Petroleum hydrocarbons

Dippers (see also acid dippers)

Dish-Washers
- Fungus infections

Disinfectant Makers (see also insecticide makers)
- Acetaldehyde
- Amino compounds of benzol, toluol and xylol
- Aniline
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Brominated hydrocarbons
- Bromine
- Carbon dioxide
- Chloride of lime
- Chlorine
- Chlorobenzols
- Cresol
- Cyanides
- Cyanogen
- Formaldehyde
- Glycols
- Hydrocyanic acid
- Mercury and its compounds
- Ozone
- Phenol
- Sulphur dioxide
- Thallium and its compounds
- Trichloroethylene
- Trinitrophenol
- Ultraviolet rays

Divers
- Carbon dioxide
- Carbon monoxide
- Compressed air (increased atmospheric pressure)

Doffers (textile)
- Dampness
- Heat
- Organic dust

Dope Workers (see aeroplane dope workers)

Dressers (glass)
- Heat

Dresser Tenders (textile)
- Dampness
- Heat
- Sudden variations of temperature

Driers (lacquer)
- Amyl acetate
- Ethyl acetate
- Ketones
- Methyl alcohol
- Petroleum hydrocarbons
- Infrared rays

Driers (rubber)
- Benzol and its homologues (toluol and xylol)
- Carbon disulphide
- Petroleum hydrocarbons

Drier Workers (foundry)
- Carbon monoxide

Drillers (rock)
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Drivers (see chauffeurs)

Drop Forgers
- Heat

Dry Cleaners
- Amyl acetate
- Benzol and its homologues (toluol and xylol)
- Carbon disulphide
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Ethylene dichloride
- Methyl alcohol
- Oxalic acid
- Petroleum hydrocarbons
- Sudden variations of temperature
- Tetrachloroethane
- Tetrachloroethylene
- Trichloroethylene
- Turpentine

Drying Room Workers (miscellaneous)
- Carbon dioxide
- Carbon monoxide
- Sudden variations of temperature

Dye Makers
- Acetaldehyde
- Acetone
- Acridine
- Alkalis
- Amino compounds of benzol, toluol and xylol
- Ammonia
- Aniline
- Antimony and its compounds
- Aromatic hydrocarbons
- Arsenic and its compounds
- Arsine
- Barium and its compounds
- Benzol
- Brominated hydrocarbons (ethyl bromide, methyl bromide)
- Bromine
- Butyl alcohol
- Carbon dioxide
- Carbon tetrachloride
- Chloride of lime
- Chlorinated hydrocarbons
- Chlorine
- Chlorobenzols
- Chromium compounds
- Cresol
- Cyanides
- Diethylene dioxide
- Dimethyl sulphate
- Dinitrophenol
- Ethylene chlorohydrin
- Ethylene glycol monomethyl ether
- Formaldehyde
- Formic acid
- Furfural
- Heat
- Hydrochloric acid
- Hydrogen sulphide
- Lead and its compounds
- Manganese and its compounds
- Mercury and its compounds
- Methyl alcohol
- Methyl chloride
- Methylene chloride
- Naphthols
- Nitric acid
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol
- Nitrous fumes
- Oxalic acid
- Phenol
- Phenyl hydrazine
- Phosgene
- Pyridine
- Sudden variations of temperature
- Sulphur dioxide
- Sulphuric acid
- Thallium and its compounds
- Toluol
- Trichloroethylene
- Trinitrophenol
- Turpentine
- Uranium and its compounds
- Vanadium and its compounds
- Xylol

Dyers (see also mordanters and other preparatory process workers)
- Acetone
- Amino compounds of benzol, toluol and xylol
- Ammonia
- Amyl acetate
- Aniline
- Antimony and its compounds
- Aromatic hydrocarbons
- Arsenic and its compounds
- Chlorinated hydrocarbons
- Chromium and its compounds
- Ethylene dichloride
- Formaldehyde
- Hydrochloric acid
- Hydrofluoric acid
- Lead and its compounds
- Manganese and its compounds
- Methyl alcohol
- Nitric acid
- Nitrous fumes
- Oxalic acid
- Petroleum hydrocarbons
- Phenol
- Pyridine
- Sudden variations of temperature
- Sulphur chloride
- Titanium oxide
- Trinitrophenol
- Uranium and its compounds
- Vanadium and its compounds

Electrical Condenser Makers
- Chlorinated diphenyls
- Chlorinated naphthalenes

Electrical Transformer Makers
- Chlorinated diphenyls
- Chlorinated naphthalenes

Electricians
- Electrical burns; electrical shock
- Ozone
- Ultraviolet and infrared rays

Electric Induction Furnace Workers
- Mercury and its compounds

Electric Linemen (see also cable splicers)
- Dampness
- Electrical burns; electrical shock
- Ultraviolet and infrared rays

Electrode Makers
- Organic dust
- Tar and pitch

Electrolytic Process (copper) **Workers**
- Arsine

Electroplaters
- Alkalis
- Antimony and its compounds
- Arsenic and its compounds
- Arsine
- Benzol and its homologues (toluol and xylol)
- Cadmium and its compounds
- Carbon disulphide
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Chlorinated naphthalenes
- Chromium compounds
- Cyanides
- Dampness
- Formic acid
- Hydrochloric acid
- Hydrocyanic acid
- Hydrofluoric acid
- Lead and its compounds
- Mercury and its compounds
- Nickel and its compounds
- Nitric acid
- Nitrous fumes
- Petroleum hydrocarbons
- Sulphuric acid
- Tetrachloroethane
- Trichloroethylene

Electrotypers (see also electroplaters)
- Ammonia
- Antimony and its compounds
- Inorganic dust (no free silica)
- Lead and its compounds
- Organic dust
- Sudden variations of temperature

Elevator Operators
- Repeated motion, pressure and vibration

Embalmers
- Formaldehyde
- Mercury and its compounds

Embalming Fluid Makers
- Mercury and its compounds

Embossers
- Mercury and its compounds

Embroidery Workers
- Defective illumination
- Lead and its compounds

Emery Wheel Makers
- Inorganic dust (no free silica)
- Lead and its compounds

Enamelers
- Amyl acetate
- Antimony and its compounds
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Carbon disulphide
- Carbon monoxide
- Chlorinated hydrocarbons
- Chromium and its compounds
- Dampness
- Ethyl acetate
- Heat
- Inorganic dust (free silica)
- Lead and its compounds
- Manganese and its compounds
- Nickel and its compounds
- Petroleum hydrocarbons
- Repeated motion, pressure and vibration
- Tetrachloroethane
- Turpentine

Enamel Makers
- Amyl acetate
- Antimony and its compounds
- Arsenic and its compounds
- Barium and its compounds
- Benzol and its homologues (toluol and xylol)
- Carbon disulphide
- Carbon monoxide
- Chlorinated hydrocarbons
- Chromium compounds
- Ethyl acetate
- Ethylene glycol monomethyl ether
- Hydrochloric acid
- Hydrofluoric acid
- Lead and its compounds
- Manganese and its compounds
- Nitric acid
- Nitrous fumes
- Petroleum hydrocarbons
- Tetrachloroethane
- Turpentine

Engineers (stationary)
- Carbon monoxide
- Heat
- Inorganic dust (no free silica)
- Sudden variations of temperature

Engravers
- Alkalis
- Benzol and its homologues (toluol and xylol)
- Copper and its compounds
- Defective illumination
- Hydrochloric acid
- Inorganic dust (no free silica)
- Lead and its compounds
- Mercury and its compounds
- Nitric acid
- Nitrous fumes
- Oxalic acid
- Repeated motion, pressure and vibration
- Sulphuric acid

Etchers
- Arsine
- Hydrochloric acid
- Hydrofluoric acid
- Nitric acid
- Nitrous fumes
- Phenol
- Sulphuric acid

Ethyl Benzene Makers
Ethyl benzene

Ethyl Bromide Users
Brominated hydrocarbons (ethyl bromide)

Ethyl Chloride Users
Chlorinated hydrocarbons (ethyl chloride)

Ethylene Dibromide Users
Brominated hydrocarbons (ethylene dibromide)

Ethylene Dichloride Users
Ethylene dichloride

Ethylene Oxide Users
Ethylene oxide

Examiners Using Fluoroscope or X-Ray
X-rays

Excavation Workers
Inorganic dust (free silica)

Explosives Workers (see also particular occupation)
Acetaldehyde
Acetone
Amino compounds of benzol, toluol and xylol
Ammonia
Amyl acetate
Amyl alcohol
Aniline
Antimony and its compounds
Benzol and its homologues (toluol and xylol)
Bromine
Carbon dioxide
Carbon disulphide
Chromium and its compounds
Cresol
Dampness
Dinitrophenol
Formaldehyde
Lead and its compounds
Mercury and its compounds
Methyl alcohol
Nitric acid
Nitrobenzene
Nitro compounds of benzol, toluol and xylol
Nitroglycerine
Nitrous fumes
Phenol
Phosphorus and its compounds
Pyridine
Sulphuric acid
Tetryl
Trinitrophenol

Exterminators and Fumigators
(see insecticide makers)

Extractor Operators (soap)
Dampness
Sudden variations of temperature

Extractors (gold and silver) (see gold and silver refiners and extractors)

Extractors (oils and fats)
Acetone
Alcohols
Benzol and its homologues (toluol and xylol)
Carbon disulphide
Chlorinated hydrocarbons
Cycloparaffins
Ethylene dichloride
Petroleum hydrocarbons
Tetrachloroethane
Trichloroethylene

Farmers
Anthrax
Arsenic and its compounds
Calcium cyanamide
Cyanides
D.D.T.
2, 4-D
Fungus infections
Glanders
Lead and its compounds

Mercury compounds
Nicotine
Phosphorus and its compounds
Septic infections
Tetanus
Tularaemia
Undulant fever

Fat Renderers
Acrolein
Anthrax
Heat
Hydrogen sulphide
Magnesium and its compounds
Ozone
Sudden variations of temperature
Sulphuric acid

Feather Curers
Arsenic and its compounds
Organic dust

Feather Workers
Amino compounds of benzol, toluol and xylol
Aniline
Arsenic and its compounds
Benzol and its homologues (toluol and xylol)
Methyl alcohol
Organic dust
Petroleum hydrocarbons
Septic infections
Sulphur dioxide
Turpentine

Felt Hat Makers
Carbon monoxide
Heat
Mercury and its compounds
Methyl alcohol
Nitric acid
Nitrous fumes
Organic dust
Sudden variations of temperature
Sulphuric acid

Felt Makers
Anthrax
Heat
Hydrogen sulphide

Ferrosilicon Workers
Arsenic and its compounds
Arsine
Inorganic dust (free silica)
Phosphine

Fertilizer Makers (see also phosphate mill workers)
Acrolein
Ammonia
Anthrax
Arsenic and its compounds
Arsine
Benzol and its homologues (toluol and xylol)
Calcium cyanamide
Carbon dioxide
Cyanides
Dampness
Hydrochloric acid
Hydrofluoric acid
Hydrogen sulphide
Inorganic dust (free silica)
Inorganic dust (no free silica)
Magnesium and its compounds
Manganese and its compounds
Nicotine
Nitric acid
Nitrous fumes
Organic dust
Phosphorus and its compounds
Septic infections
Sulphur dioxide
Sulphuric acid

Fiberizers (asbestos)
Asbestos dust

Fiber Workers
Organic dust

Filament Makers and Finishers (incandescent lamps)
- Amyl acetate
- Carbon monoxide
- Methyl alcohol
- Thallium and its compounds

File Makers
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Filers
- Antimony and its compounds
- Inorganic dust (no free silica)
- Lead and its compounds

Filling Station Workers
- Carbon monoxide
- Lead and its compounds
- Petroleum hydrocarbons
- Tetraethyl lead

Film Makers (see photographic film makers)

Filter Press Workers
- Dampness

Finishers (leather)
- Organic dust

Fire Extinguisher Makers
- Brominated hydrocarbons (ethyl bromide, ethylene dibromide, methyl bromide)
- Carbon dioxide
- Carbon tetrachloride
- Chlorinated hydrocarbons (ethyl chloride)

Firemen (city)
- Acrolein
- Carbon monoxide
- Dampness
- Heat
- Phosgene
- Sudden variations of temperature
- Sulphur dioxide

Firemen (stationary)
- Carbon monoxide
- Heat
- Inorganic dust (no free silica)
- Sudden vatiations of temperature
- Ultraviolet and infrared rays

Fireworks Makers (see also explosive workers)
- Antimony and its compounds
- Arsenic and its compounds
- Barium and its compounds
- Manganese and its compounds
- Mercury and its compounds
- Phosphorus and its compounds
- Thallium and its compounds
- Trinitrophenol

Fishermen
- Dampness
- Septic infections
- Sudden variations of temperature
- Tar and pitch

Flatteners (glass)
- Heat

Flavouring Extract Makers
- Amyl acetate
- Amyl alcohol
- Benzol and its homologues (toluol and xylol)
- Butyl alcohol
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol

Flax-Rettery Workers
- Hydrogen sulphide

Flax Spinners
- Heat
- Organic dust

Flint Workers
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Floor Polish Makers (see polish makers)

Flour Mill Workers
Fungus infections
Organic dust

Flue Cleaners
Carbon monoxide
Inorganic dust (no free silica)
Sulphur dioxide
Tar and pitch

Flue Dust Recoverers (sulphuric acid manufacturing)
Thallium and its compounds

Fluorescent Lamp Makers
Beryllium and its compounds

Flush Tenders (aluminum)
Dampness

Fly Paper Makers
Arsenic and its compounds

Food Irradiators
Ultraviolet and infrared rays

Forgemen
Heat

Formaldehyde Workers
Formaldehyde

Formic Acid Workers
Formic acid
Oxalic acid

Foundry Workers (see also particular metal)
Carbon dioxide
Carbon monoxide
Heat
Inorganic dust (free silica)
Inorganic dust (no free silica)
Ultraviolet and infrared rays

Freight Handlers
Anthrax

Frosters (glass and pottery)
Chromium compounds

Fruit Essence Makers (see flavouring extract makers)

Fruit Preservers
Sulphur dioxide

Fullers (textiles)
Benzol and its homologues (toluol and xylol)
Carbon tetrachloride
Chlorinated hydrocarbons
Dichloroethyl ether
Tetrachloroethane

Fulminate Mixers
Hydrocyanic acid
Mercury and its compounds
Nitric acid
Nitrous fumes

Fumigant Makers (see insecticide makers)

Fumigators and Exterminators (see insecticide makers)

Fur Carders
Anthrax
Organic dust

Fur Clippers
Anthrax
Organic dust

Fur Cutters
Anthrax
Organic dust

Fur Handlers
Amino compounds of benzol, toluol and xylol
Anthrax
Hydrogen sulphide
Mercury and its compounds
Organic dust

Furnace Workers (see also particular metal)
Carbon dioxide

Carbon monoxide
Heat
Inorganic dust (no free silica)
Ultraviolet and infrared rays

Furnace Workers (bauxite)
Inorganic dust and fume (Group IV)
Ultraviolet and infrared rays

Furniture Polishers
Amyl acetate
Chromium compounds
Methyl alcohol
Organic dust
Petroleum hydrocarbons
Repeated motion, pressure and vibration
Turpentine

Fur Preparers
Anthrax
Formaldehyde
Mercury and its compounds
Nitric acid
Nitrous fumes
Organic dust

Fur Pullers
Anthrax
Organic dust

Fusel Oil Workers
Amyl alcohol

Fused Quartz Workers
Inorganic dust (free silica)

Galvanizers
Acrolein
Ammonia
Arsenic and its compounds
Arsine
Dampness
Heat
Hydrochloric acid
Lead and its compounds
Metal fume fever
Nitric acid
Nitrous fumes
Petroleum hydrocarbons
Sulphur dioxide
Sulphuric acid
Trichloroethylene
Zinc and its compounds

Garage Workers
Acrolein
Carbon monoxide
Lead and its compounds
Oxalic acid
Petroleum hydrocarbons
Tetraethyl lead

Gardeners
Arsenic and its compounds
Calcium cyanamide
Cyanides
D.D.T.
2, 4-D.
Lead and its compounds
Nicotine
Phosphorus compounds
Tetanus
Undulant fever

Gas (illuminating) **Workers**
Ammonia
Arsine
Benzol and its homologues (toluol and xylol)
Carbon monoxide
Heat
Hydrocyanic acid
Hydrofluoric acid
Hydrogen sulphide
Phenol
Sudden variations of temperature
Tar and pitch
Trichloroethylene

Gasoline Blenders
Aliphatic hydrocarbons
Amino compounds of benzol, toluol and xylol
Aniline
Benzol and its homologues (toluol and xylol)

Brominated hydrocarbons (ethylene dibromide)
Ethyl benzene
Lead and its compounds
Nitrobenzol
Nitro compounds of benzol, toluol and xylol
Petroleum hydrocarbons
Tetraethyl lead

Gasoline Engine Workers
Acrolein
Carbon monoxide
Petroleum hydrocarbons

Gas Purifiers
Ammonia
Hydrocyanic acid
Hydrogen sulphide
Phenol

Gassers (textile)
Carbon monoxide

Gatherers (glass)
Heat

Gelatine Makers
Acrolein
Anthrax
Sulphur dioxide

Germicide Makers (see disinfectant makers)

Gilders
Amyl acetate
Benzol and its homologues (toluol and xylol)
Cyanides
Ethyl acetate
Mercury and its compounds
Methyl alcohol
Nitric acid
Nitrous fumes
Petroleum hydrocarbons
Pyridine

Glass Blowers
Heat
Inorganic dust (no free silica)
Ultraviolet and infrared rays

Glass Colourers
Cadmium compounds
Chromium compounds
Cobalt compounds
Selenium compounds
Tellurium compounds
Thallium compounds

Glass Cutters
Dampness
Inorganic dust (no free silica)

Glass Etchers
Formaldehyde
Hydrofluoric acid

Glass Finishers
Dampness
Hydrochloric acid
Hydrofluoric acid
Inorganic dust (no free silica)
Lead and its compounds
Sulphuric acid

Glass Furnace Workers (see also glass mixers)
Carbon monoxide
Heat
Inorganic dust (free silica)
Inorganic dust (no free silica)
Ultraviolet and infrared rays

Glass Mixers
Alkalis
Antimony and its compounds
Arsenic and its compounds
Barium and its compounds
Hydrochloric acid
Inorganic dust (free silica)
Inorganic dust (no free silica)
Lead and its compounds
Magnesium and it compounds
Manganese and its compounds
Selenium and its compounds
Thallium and its compounds
Uranium and its compounds
Vanadium and its compounds

Glass Polishers
Lead and its compounds

Glass (safety) **Makers**
- Butyl alcohol
- Chlorinated hydrocarbons
- Methyl alcohol
- Tetrachloroethane

Glaze Dippers (pottery)
- Antimony and its compounds
- Arsenic and its compounds
- Chromium compounds
- Dampness
- Hydrochloric acid
- Lead and its compounds
- Manganese and its compounds

Glaze Mixers (pottery)
- Antimony and its compounds
- Arsenic and its compounds
- Chromium and its compounds
- Hydrochloric acid
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Lead and its compounds
- Manganese and its compounds

Glost-Kiln Workers
- Carbon monoxide
- Lead and its compounds
- Sudden variations of temperature

Glove Makers (leather preparers) (see also tannery workers)
- Dampness
- Organic dust

Glue Workers
- Acrolein
- Ammonia
- Anthrax
- Benzol and its homologues (toluol and xylol)
- Carbon dioxide
- Carbon disulphide
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Cresol
- Dampness
- Hydrochloric acid
- Hydrogen sulphide
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol
- Organic dust
- Petroleum hydrocarbons
- Septic infections
- Sudden variations of temperature
- Sulphur dioxide
- Sulphuric acid
- Trichloroethylene

Glycerine Refiners
- Oxalic acid

Gold Beaters
- Inorganic dust (no free silica)
- Repeated motion, pressure and vibration

Gold and Silver Refiners and Extractors
- Arsenic and its compounds
- Arsine
- Bromine
- Chlorine
- Cyanides
- Formaldeyhde
- Hydrocyanic acid
- Hydrofluoric acid
- Inorganic dust (no free silica)
- Lead and its compounds
- Mercury and its compounds
- Sulphur chloride

Grain Elevator Workers
- Carbon dioxide
- Fungus infections
- Organic dust

Granite Workers (see stone-cutters)

Graphite Workers
- Heat
- Inorganic dust (no free silica)
- Organic dust

Grinders (colours) (see colour makers)

Grinders (metals)
- Antimony and its compounds
- Dampness
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Lead and its compounds
- Repeated motion, pressure and vibration

Grinders (rubber)
- Antimony and its compounds
- Lead and its compounds
- Organic dust

Grinding Wheel Makers
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Grooms
- Fungus infections
- Glanders
- Septic infections
- Tetanus

Guncotton Dippers
- Nitric acid
- Nitrous fumes
- Sulphuric acid

Guncotton Pickers
- Organic dust

Guncotton Washers
- Dampness

Gypsum Workers
- Hydrogen sulphide
- Inorganic dust (no free silica)
- Sudden variations of temperature

Hair Workers
- Anthrax
- Dampness
- Mercury and its compounds
- Organic dust
- Septic infections

Hammermen
- Repeated motion, pressure and vibration

Hardeners (see temperers)

Harness Makers
- Organic dust

Hat Makers (felt) (see felt hat makers)

Heel Makers (shoe)
- Organic dust

Hemp Workers
- Organic dust

Hide Workers
- Alkalis
- Anthrax
- Chromium compounds
- Fungus infections
- Septic infections

Horn Workers
- Organic dust

Hothouse Workers (see also gardeners)
- Arsenic compounds
- Cyanides
- D.D.T.
- 2, 4-D.
- Lead compounds
- Nicotine
- Phosphorus compounds
- Sudden variations of temperature

Hot Rod Rollers (iron and steel)
- Heat

Housemaids
- Alkalis
- Chlorinated hydrocarbons
- Repeated motion, pressure and vibration

House Wreckers
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Hydraulic Construction Workers
- Dampness

Hydraulic Miners
Dampness

Hydrochloric Acid Makers
Arsine
Hydrochloric acid
Hydrogen sulphide
Sulphuric acid

Hydrocyanic Acid Makers
Cyanides
Hydrocyanic acid
Sulphuric acid

Hydrofluoric Acid Makers
Hydrofluoric acid

Ice (artificial) Makers (see artificial ice makers)

Ice Cream Makers
Ammonia
Carbon dioxide
Dampness
Sudden variations of temperature

Incandescent Lamp Makers (see also particular occupation)
Amyl acetate
Carbon monoxide
Lead and its compounds
Mercury and its compounds
Methyl alcohol
Thallium and its compounds

Incandescent Mantle Hardeners
Hydrofluoric acid
Pyridine
Thorium and its compounds

Ink Makers
Alkalis
Ammonia
Arsenic and its compounds
Barium compounds
Benzol and its homologues (toluol and xylol)
Brominated hydrocarbons (methyl bromide)
Bromine
Carbon monoxide
Carbon tetrachloride
Chlorinated hydrocarbons
Chlorine
Chromium compounds
Cresol
Ethylene glycol monomethyl ether
Formaldehyde
Glycols
Hydrochloric acid
Lead and its compounds
Mercury and its compounds
Methyl alcohol
Nitrobenzol
Nitro compounds of benzol, toluol and xylol
Oxalic acid
Petroleum hydrocarbons
Silver compounds
Turpentine
Vanadium compounds

Insecticide Makers (see also disinfectant makers)
Arsenic compounds
Barium compounds
Bromine
Carbon dioxide
Carbon disulphide
Carbon monoxide
Carbon tetrachloride
Chlorinated hydrocarbons
Chlorobenzols
Cresol
Cyanides
D.D.T.
Diethylene dioxide
Ethylene dichloride
Ethylene oxide
Formaldehyde
Hydrocyanic acid
Lead and its compounds
Mercury and its compounds
Methyl formate
Nicotine
Nitric acid
Nitrous fumes

Petroleum hydrocarbons
Phosphorus compounds
Sulphur chloride
Sulphur dioxide
Tetrachloroethane
Thallium compounds
Trichloroethylene
Turpentine

Inspectors Using Fluoroscope or X-Ray
X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Instrument Dial (luminous) **Painters**
X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Insulation (sound, heat) **Workers**
Asbestos dust
Inorganic dust (free silica)
Inorganic dust (no free silica)

Insulators (wire)
Antimony and its compounds
Arsenic and its compounds
Benzol and its homologues (toluol and xylol)
Carbon tetrachloride
Chlorinated diphenyls
Chlorinated hydrocarbons
Chlorinated naphthalenes
Ethylene dichloride
Tar and pitch

Iodine Makers
Chlorine

Iron and Steel Workers (all departments) (see also particular occupation and alloy makers)
Arsenic and its compounds
Carbon monoxide
Heat
Inorganic dust (no free silica)
Titanium dioxide
Ultraviolet and infrared rays

Ironers
Carbon monoxide
Repeated motion, pressure and vibration
Sudden variations of temperature

Irradiators (food)
Ultraviolet rays

Japan Makers
Arsenic and its compounds
Lead and its compounds
Methyl alcohol
Petroleum hydrocarbons
Sudden variations of temperature
Turpentine

Japanners
Arsenic and its compounds
Lead and its compounds
Methyl alcohol
Petroleum hydrocarbons
Turpentine

Jewelers
Amyl acetate
Arsine
Cyanides
Defective illumination
Hydrochloric acid
Inorganic aust (no free silica)
Lead and its compounds
Mercury and its compounds
Nitric acid
Nitrous fumes
Repeated motion, pressure and vibration
Sulphuric acid

Junk (metal) **Refiners**
Heat
Inorganic dust (no free silica)
Lead and its compounds
Metal fume fever
Zinc and its compounds

Jute Workers
Inorganic dust (free silica)
Inorganic dust (no free silica)
Organic dust

Kiln Tenders
Carbon monoxide
Heat

Knitters
Repeated motion, pressure and vibration

Knitting Mill Workers
Organic dust

Labelers (paint cans)
Lead and its compounds

Laboratory Workers (see also chemists)
Carbon monoxide
Mercury and its compounds

Laboratory Workers (radium research)
X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Lace Makers
Organic dust

Lacquerers
Acetone
Amyl acetate
Amyl alcohol
Arsenic and its compounds
Benzol and its homologues (toluol and xylol)
Chlorobenzols
Ethyl acetate
Ethyl benzene
Formic acid
Ketones
Lead and its compounds
Methyl alcohol
Petroleum hydrocarbons
Pyridine
Triorthocresyl phosphate
Turpentine

Lacquer Makers
Acetaldehyde
Acetone
Ammonia
Amyl acetate
Amyl alcohol
Arsenic and its compounds
Barium compounds
Benzol and its homologues (toluol and xylol)
Butanone
Butyl alcohol
Chlorinated diphenyls
Chlorinated naphthalenes
Chlorobenzols
Diethylene dioxide
Ethylene chlorohydrin
Ethylene glycol monoethyl ether
Ethylene glycol monomethyl ether
Formaldehyde
Formic acid
Ketones (hexone, hexanone)
Lead and its compounds
Methyl alcohol
Nitric acid
Nitrous fumes
Pentanone
Petroleum hydrocarbons
Propyl alcohol
Pyridine
Triorthocresyl phosphate
Turpentine

Lampblack Makers
Organic dust
Petroleum oils
Phenol

Lamps (electric) (see incandescent lampmakers)

Lapidaries
Inorganic dust (no free silica)

Lard Makers
Acrolein

Lasters (shoes)
Dampness
Methyl alcohol
Organic dust
Sudden variations of temperature

Lathe Turners
Repeated motion, pressure and vibration

Laundry Workers
Carbon monoxide
Chloride of lime
Chlorine
Dampness
Formaldehyde
Heat
Ozone
Sudden variations of temperature

Lead Arsenate Makers
Arsenic and its compounds
Lead and its compounds

Lead Burners
Arsine
Lead and its compounds

Leadfoil Makers
Heat
Lead and its compounds

Lead Miners (see also miners)
Lead and its compounds

Lead Pipe Makers
Lead and its compounds

Lead Platers (on iron)
Mercury and its compounds

Lead Salts Makers
Lead and its compounds

Lead Smelter Workers
Antimony and its compounds
Arsenic and its compounds
Cadmium and its compounds
Carbon monoxide
Heat
Lead and its compounds
Selenium and its compounds
Sulphur dioxide
Tellurium and its compounds

Leather Workers (see also tannery workers)
Amino compounds of benzol, toluol and xylol
Amyl acetate
Anthrax
Barium compounds
Carbon tetrachloride
Chlorinated hydrocarbons
Chromium and its compounds
Hydrochloric acid
Methyl alcohol
Organic dust
Trichloroethylene

Lehr Tenders (glass)
Heat

Letter Sorters
Defective illumination
Repeated motion, pressure and vibration

Levermen (iron and steel)
Heat

Lime Burners
Arsine
Carbon dioxide
Carbon monoxide
Heat
Inorganic dust (no free silica)
Selenium compounds

Lime Kiln Chargers
Carbon dioxide
Carbon monoxide
Inorganic dust (no free silica)

Lime Pullers (tannery)
Anthrax
Dampness

Lime Workers
Inorganic dust (no free silica)

Linen Workers
Organic dust

Linoleum Makers
- Acrolein
- Amyl acetate
- Arsenic and its compounds
- Barium compounds
- Benzol and its homologues (toluol and xylol)
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Chromium compounds
- Dampness
- Ethylene chlorohydrin
- Heat
- Inorganic dust (no free silica)
- Lead and its compounds
- Manganese compounds
- Methyl alcohol
- Organic dust
- Petroleum hydrocarbons
- Sudden variations of temperature
- Sulphuric acid
- Turpentine

Linotypers
- Antimony and its compounds
- Carbon monoxide
- Lead and its compounds

Linseed Oil Boilers
- Acrolein
- Carbon dioxide
- Lead and its compounds

Litharge Workers
- Lead and its compounds

Lithographers
- Amino compounds of benzol, toluol and xylol
- Aniline
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Chromium compounds
- Glycols
- Hydrochloric acid
- Inorganic dust (no free silica)
- Lead and its compounds
- Mercury and its compounds
- Methyl alcohol
- Nitric acid
- Nitrous fumes
- Oxalic acid
- Petroleum hydrocarbons
- Repeated motion, pressure and vibration
- Sulphuric acid
- Tetrachloroethane
- Turpentine

Lithopone Makers
- Barium and its compounds
- Cadmium and its compounds

Lithotransfer Workers
- Lead and its compounds

Locksmiths
- Repeated motion, pressure and vibration

Longshoremen
- Anthrax
- Manganese compounds
- Weil's disease

Luminous Dial Factory Workers
- X-rays, radium, and other radioactive substances (radiothorium, mesothorium, etc.)

Luters (zinc smelting)
- Heat
- Metal fume fever
- Zinc and its compounds

Lye Makers
- Alkalis

Machinists
- Petroleum oils
- Repeated motion, pressure and vibration

Magnesium Alloy Makers
- Magnesium and its compounds

Mail Sorters
- Defective illumination
- Repeated motion, pressure and vibration

Manganese Dioxide Workers
Manganese and its compounds

Manganese Grinders
Manganese and its compounds

Manganese Ore Separators
Manganese and its compounds

Manganese Steel Makers
Manganese and its compounds

Manometer Makers
Mercury and its compounds

Manure Handlers
Septic infections
Tetanus
Undulant fever

Marble Cutters
Inorganic dust (no free silica)

Masons
Dampness
Inorganic dust (free silica)
Inorganic dust (no free silica)
Repeated motion, pressure and vibration

Masseurs
Fungus infections
Septic infections

Match Factory Workers
Alkalis
Antimony and its compounds
Carbon disulphide
Chromium compounds
Dampness
Hydrogen sulphide
Inorganic dust (no free silica)
Lead and its compounds
Manganese compounds
Organic dust
Phosphorus and its compounds

Mattress Makers
Anthrax
Organic dust

Meat Inspectors
Anthrax
Erysipeloid
Fungus infections
Septic infections
Undulant fever

Meat Packing Employees (see packing house employees and slaughterhouse workers)

Mechanics (gas engines)
Carbon monoxide
Petroleum hydrocarbons
Petroleum oils

Melters (foundry, glass)
Heat

Mercerizers
Alkalis
Hydrochloric acid
Sulphuric acid

Mercury Alloy Makers
Mercury and its compounds

Mercury Boiler Workers
Mercury and its compounds

Mercury Bronzers
Mercury and its compounds

Mercury Pump Workers
Mercury and its compounds

Mercury Salt Workers
Mercury and its compounds

Mercury Solder Workers
Mercury and its compounds

Mercury Still Cleaners
Mercury and its compounds

Mercury Switch Makers
Mercury and its compounds

Mercury Vapour Lamp Makers
Mercury and its compounds

Metallizers
- Cadmium and its compounds
- Copper and its compounds
- Lead and its compounds
- Metal fume fever
- Selenium and its compounds
- Zinc and its compounds

Metal Polishers and Cleaners (see polishers and cleaners, metal)

Metal Polish Makers (see polish makers)

Metal Turners
- Inorganic dust (no free silica)

Metal Workers (see particular occupation)

Metal Washers
- Chlorinated hydrocarbons
- Petroleum hydrocarbons

Methyl Alcohol Workers
- Acetone
- Carbon monoxide
- Methyl alcohol

Mica Strippers or Splitters
- Inorganic dust (no free silica)

Mica Workers
- Inorganic dust (no free silica)

Microscopists
- Repeated motion, pressure and vibration

Milkers
- Fungus infections
- Repeated motion, pressure and vibration
- Undulant fever

Milk Inspectors
- Undulant fever

Millers
- Fungus infections
- Mercury and its compounds

Millinery Workers
- Acetone
- Amino compounds of benzol, toluol and xylol
- Amyl acetate
- Aniline
- Benzol and its homologues (toluol and xylol)
- Defective illumination
- Methyl alcohol
- Petroleum hydrocarbons
- Turpentine

Mineral Earth Workers
- Inorganic dust (no free silica)

Miners
- Asbestos dust
- Carbon dioxide
- Carbon monoxide
- Dampness
- Defective illumination
- Heat
- Hydrogen sulphide
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Manganese and its compounds
- Nitrous fumes
- Repeated motion, pressure and vibration
- Silver and its compounds
- Sudden variations of temperature

Minkery Workers
- Anthrax
- Glanders
- Septic infections

Mirror Silverers
- Acetaldehyde
- Ammonia
- Benzol and its homologues (toluol and xylol)
- Cyanides
- Dampness
- Formaldehyde
- Formic acid
- Lead and its compounds
- Mercury and its compounds
- Silver and its compounds
- Sudden variations of temperature

Mixers (rubber)
- Amino compounds of benzol, toluol and xylol
- Aniline
- Antimony and its compounds
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Carbon tetrachloride
- Chromium compounds
- Hexamethylene tetramine
- Inorganic dust (no free silica)
- Lead and its compounds
- Paraldehyde
- Petroleum hydrocarbons
- Sudden variations of temperature
- Synthethic rubber

Mixing Room Workers (miscellaneous)
- Inorganic dust (no free silica)
- Organic dust

Mould Breakers (foundry)
- Heat
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Mould Breakers (pottery)
- Carbon monoxide

Moulders (asbestos)
- Asbestos dust

Moulders (foundry)
- Heat
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Lead and its compounds
- Metal fume fever
- Zinc and its compounds

Monotypers
- Antimony and its compounds
- Carbon monoxide
- Lead and its compounds

Mordanters
- Amyl alcohol
- Antimony and its compounds
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Chloride of lime
- Chromium compounds
- Cyanides
- Formic acid
- Nitric acid
- Nitrous fumes
- Petroleum hydrocarbons
- Vanadium compounds

Motion Picture Film Workers (see also pyroxylin plastics workers)
- Amyl acetate
- Butyl alcohol
- Carbon monoxide
- Chlorinated hydrocarbons
- Tetrachloroethane

Motion Picture Machine Operators
- Mercury and its compounds
- Nitrous fumes
- Ultraviolet and infrared rays

Motion Picture Studio Workers and Actors
- Ultraviolet and infrared rays

Mottlers (leather)
- Amyl acetate
- Methyl alcohol

Muffle Tenders
- Heat

Mule Handlers
- Anthrax
- Glanders

Muriatic Acid Makers (see hydrochloric acid makers)

Musical Instrument Makers
- Lead and its compounds

Musicians
- Repeated motion, pressure and vibration

Naphthylamine Workers
- Amino compounds of benzol, toluol and xylol
- Aniline
- Naphthylamines

Neon Lights Lettermakers
- Carbon monoxide

Nickel Extractors
- Nickel and its compounds

Nickel Platers (see also electroplaters)
- Cyanides
- Dampness

Nickel Purification Workers (mond process)
- Carbon monoxide
- Nickel and its compounds
- Nickel carbonyl

Nitraniline Workers
- Amino compounds of benzol, toluol and xylol
- Aniline

Nitrators
- Nitric acid
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol
- Nitroglycerine
- Nitrous fumes
- Sulphuric acid

Nitric Acid Workers
- Ammonia
- Lead and its compounds
- Nitric acid
- Nitrous fumes
- Sulphuric acid

Nitrobenzene Workers (see nitrobenzol workers)

Nitrobenzol Workers
- Benzol and its homologues (toluol and xylol)
- Nitric acid
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol
- Nitrous fumes
- Sulphuric acid

Nitrocellulose Workers (see also pyroxylin plastics workers)
- Acetone
- Amyl acetate
- Amyl aclohol
- Arsine
- Benzol and its homologues (toluol and xylol)
- Ethyl acetate
- Ethylene glycol monomethyl ether
- Nitric acid
- Nitrous fumes
- Sulphuric acid

Nitroglycerine Makers
- Arsine
- Lead and its compounds
- Nitric acid
- Nitroglycerine
- Nitrous fumes
- Sulphuric acid

Nitrous Oxide Workers
- Nitrous fumes

Oilcloth Makers (see linoleum makers)

Oilers
- Petroleum oils

Oil Extractors (see extractors) (oils and fats)

Oil Flotation Plant Workers (see also concentrating mill workers)
- Hydrogen sulphide
- Petroleum oils
- Sulphur dioxide

Oil Purifiers
- Sulphuric acid

Oil Refiners (see petroleum refiners)

Oil Well Workers
- Hydrogen sulphide
- Petroleum oils

Open Hearth Department Workers (iron and steel)
- Carbon monoxide
- Heat

Ore Concentrating Mill Workers (see concentrating mill workers)

Oxyacetylene Cutters (see welders)

Ozonators
- Ozone

Packing House Employees (see also slaughterhouse workers)
- Alkalis
- Dampness
- Erysipeloid
- Heat
- Hydrogen sulphide
- Septic infections
- Sudden variations of temperature
- Undulant fever
- Weil's disease

Painters
- Acetone
- Amino compounds of benzol, toluol and xylol
- Amyl acetate
- Amyl alcohol
- Aniline
- Antimony and its compounds
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Carbon disulphide
- Chromium compounds
- Ethyl acetate
- Ketones
- Lead and its compounds
- Manganese compounds
- Mercury and its compounds
- Methyl alcohol
- Nitric acid
- Nitrous fumes
- Petroleum hydrocarbons
- Repeated motion, pressure and vibration
- Titanium dioxide
- Turpentine

Painters (luminous watch and instrument dials)
- X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Painters (tar)
- Tar and pitch

Paint Makers
- Acetone
- Alkalis
- Amino compounds of benzol, toluol and xylol
- Amyl acetate
- Amyl alcohol
- Aniline
- Antimony and its compounds
- Arsenic compounds
- Barium compounds
- Benzol and its homologues (toluol and xylol)
- Cadmium compounds
- Carbon disulphide
- Chlorinated diphenyls
- Chlorinated naphthalenes
- Chromium compounds
- Dampness
- Ethyl acetate
- Hydrochloric acid
- Lead and its compounds
- Magnesium compounds
- Manganese compounds
- Mercury and its compounds
- Methyl alcohol
- Petroleum hydrocarbons
- Phenol
- Pyridine
- Selenium compounds
- Sulphuric acid
- Tar and pitch
- Titanium dioxide

Turpentine
Uranium compounds

Paint Remover Makers
Ammonia
Benzol and its homologues (toluol and xylol)
Butanone
Cresol
Dichloroethyl ether
Diethylene dioxide
Furfural
Ketones
Methyl alcohol
Methylene chloride
Petroleum hydrocarbons
Phenol

Paint Removers
Ammonia
Acetone
Amyl acetate
Benzol and its homologues (toluol and xylol)
Dichloroethyl ether
Inorganic dust (no free silica)
Ketones
Lead and its compounds
Methylene chloride
Petroleum hydrocarbons
Phenol

Pair Heaters (tin plate)
Heat

Paper Box Makers
Repeated motion, pressure and vibration

Paper Glazers
Arsenic and its compounds

Paperhangers
Arsenic and its compounds
Chromium compounds
Inorganic dust (no free silica)
Lead and its compounds

Paper Makers (see also particular occupation)
Alkalis
Ammonia
Amyl acetate
Arsine
Chlorine
Chromium compounds
Dampness
Excessive noise
Formaldehyde
Fungus infections
Heat
Hydrochloric acid
Hydrofluoric acid
Hydrogen sulphide
Lead compounds
Magnesium compounds
Sulphur dioxide
Sulphuric acid
Sudden variations of temperature
Titanium dioxide

Paper Money Makers
Chromium compounds

Paraffin Workers
Acetone
Benzol and its homologues (toluol and xylol)
Carbon disulphide
Carbon tetrachloride
Ethylene dichloride
Petroleum oils

Parakeet Handlers
Psittacosis

Paris Green Workers
Arsenic and its compounds

Parrot Handlers
Psittacosis

Patent Leather Makers
Amyl acetate
Carbon monoxide
Lead and its compounds
Methyl alcohol
Oxalic acid
Ozone
Sudden variations of temperature
Sulphuric acid
Turpentine

Pavers
- Heat
- Repeated motion, pressure and vibration
- Tar and pitch

Pencil Makers
- Acetone
- Alkalis
- Amino compounds of benzol, toluol and xylol
- Aniline
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Chromium compounds
- Pyridine

Perfume Makers
- Acetone
- Amino compounds of benzol, toluol and xylol
- Ammonia
- Amyl acetate
- Aniline
- Benzol and its homologues (toluol and xylol)
- Brominated hydrocarbons (ethyl bromide)
- Butyl alcohol
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Cresol
- Dimethyl sulphate
- Ethyl acetate
- Formic acid
- Hydrochloric acid
- Methyl alcohol
- Methyl chloride
- Methylene chloride
- Naphthols
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol
- Petroleum hydrocarbons
- Phenol
- Sulphuric acid
- Trichloroethylene

Petroleum Refiners
- Acetone
- Acrolein
- Alkalis
- Amino compounds of benzol, toluol and xylol
- Ammonia
- Aniline
- Benzol and its homologues (toluol and xylol)
- Carbon monoxide
- Chlorinated hydrocarbons
- Dampness
- Dichloroethyl ether
- Heat
- Hydrochloric acid
- Hydrogen sulphide
- Lead and its compounds
- Methylene chloride
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol
- Petroleum hydrocarbons
- Petroleum oils
- Sulphur dioxide
- Sulphuric acid
- Trichloroethylene
- Turpentine

Pewter Makers
- Antimony and its compounds
- Lead and its compounds

Pharmaceutical Workers
- Acetone
- Acrolein
- Alkalis
- Amino compounds of benzol, toluol and xylol
- Aniline
- Antimony and its compounds
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Brominated hydrocarbons (ethyl bromide, ethylene dibromide. methyl bromide)
- Bromine
- Calcium cyanamide

Carbon dioxide
Carbon tetrachloride
Chloride of lime
Chlorinated hydrocarbons
Diethylene dioxide
Dinitrophenol
Ethylene dichloride
Formic acid
Magnesium compounds
Manganese compounds
Mercury and its compounds
Methyl chloride
Methylene chloride
Naphthols
Nitric acid
Nitroglycerine
Nitrous fumes
Organic dust
Petroleum hydrocarbons
Phenol
Phenyl hydrazine
Phosgene
Phosphorus and its compounds
Sulphuric acid
Tellurium compounds
Tetrachloroethane
Tetrachloroethylene
Trichloroethylene
Trinitrophenol
Turpentine
Uranium

Phenol Makers
Benzol and its homologues (toluol and xylol)
Phenol
Sulphuric acid

Phenyl Hydrazine Workers
Phenyl hydrazine

Phosphate Extractors
Hydrochloric acid

Phosphate Mill Workers (see also fertilizer makers)
Dampness
Hydrofluoric acid
Inorganic dust (no free silica)
Phosphorus and its compounds
Sudden variations of temperature

Phosphide Makers
Carbon monoxide
Phosphine

Phosphor-Bronze Workers
Phosphorus and its compounds

Phosphoric Acid Makers
Cyanides
Nitric acid
Nitrous fumes
Sulphuric acid

Phosphorus Compound Makers
Hydrogen sulphide
Phosphorus and its compounds

Phosphorus Extracters
Hydrofluoric acid
Phosphine
Phosphorus and its compounds

Phosphorus (red) **Makers**
Phosphine
Phosphorus and its compounds

Phosphorus Refiners
Phosphine
Phosphorus and its compounds

Photoengravers
Alkalis
Ammonia
Amyl acetate
Benzol and its homologues (toluol and xylol)
Chromium compounds
Methyl alcohol
Nitric acid
Nitrous fumes

Photographers (see also photographic material workers)
Defective illumination
Methly alcohol
Ultraviolet and infrared rays

Photographic Film Makers (see also pyroxylin plastics workers)
- Amyl acetate
- Bromine
- Butyl alcohol
- Defective illumination
- Ethyl acetate
- Nitric acid
- Nitrous fumes
- Silver and its compounds

Photographic Material Workers (see also photographic film makers)
- Acetaldehyde
- Acetone
- Amino compounds of benzol, toluol and xylol
- Ammonia
- Aniline
- Barium compounds
- Benzol and its homologues (toluol and xylol)
- Chlorine
- Chromium compounds
- Cresol
- Cyanides
- Formaldehyde
- Hydrochloric acid
- Mercury and its compounds
- Metol
- Phenol
- Sulphuric acid
- Tellurium compounds
- Trichloroethylene
- Trinitrophenol
- Turpentine
- Uranium compounds
- Vanadium compounds

Photograph Retouchers
- Lead and its compounds

Photogravure Workers
- Chromium compounds
- Nitric acid
- Nitrous fumes

Picklers
- Arsine
- Cyanides
- Dampness
- Heat
- Hydrochloric acid
- Hydrofluoric acid
- Nitric acid
- Nitrous fumes
- Sulphuric acid

Picric Acid Makers
- Benzol and its homologues (toluol and xylol)
- Nitric acid
- Nitrous fumes
- Phenol
- Sulphuric acid
- Trinitrophenol

Pigment Makers (see colour makers)

Pile Drivers
- Repeated motion, pressure and vibration

Pipe Fitters (see also particular liquid piped)
- Lead and its compounds

Pitch Workers
- Arsenic and its compounds
- Cresol
- Heat
- Tar and pitch

Planer Men (stone)
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Plasterers
- Anthrax
- Dampness
- Inorganic dust (no free silica)

Plaster of Paris Workers
- Inorganic dust (no free silica)

Plastics Makers (see pyroxylin plastics workers, also resin (synthetic) makers)

Platers (see electroplaters and metallizers)

Platinum Extractors
- Bromine

Plumbers (see also particular substance piped)
- Arsine
- Carbon monoxide
- Lead and its compounds

Pneumatic Tool Workers
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Repeated motion, pressure and vibration

Policemen
- Carbon monoxide
- Dampness

Polishers and Cleaners (metal) (see also degreasers)
- Benzol and its homologues (toluol and xylol)
- Cyanides
- Hydrochloric acid
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Lead and its compounds
- Methyl alcohol
- Organic dust
- Oxalic acid
- Petroleum hydrocarbons
- Pyridine
- Repeated motion, pressure and vibration
- Silver and its compounds
- Trichloroethylene
- Turpentine

Polish Makers
- Amino compounds of benzol, toluol and xylol
- Amyl acetate
- Aniline
- Benzol and its homologues (toluol and xylol)
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Chlorobenzols
- Cyanides
- Diethylene dioxide
- Inorganic dust (no free silica)
- Methyl alcohol
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol
- Oxalic acid
- Petroleum hydrocarbons
- Trichloroethylene
- Turpentine

Porcelain Makers (see pottery workers)

Porters
- Repeated motion, pressure and vibration

Potassium Hydroxide Makers
- Alkalis

Pot Fillers (glass)
- Heat

Potlifters (iron and steel)
- Heat

Pot Pullers (foundry)
- Heat

Pot Room Workers (aluminum foundry; carbide plant)
- Heat

Pot Setters
- Heat

Pottery Workers (see also particular occupation)
- Arsenic and its compounds
- Carbon dioxide
- Carbon monoxide
- Chromium compounds
- Cobalt compounds
- Dampness
- Heat
- Hydrochloric acid
- Hydrofluoric acid

Inorganic dust (free silica)
Inorganic dust (no free silica)
Lead and its compounds
Manganese compounds
Mercury and its compounds
Selenium compounds
Sulphur dioxide

Pourers (foundry)
Heat
Ultraviolet and infrared rays

Powder Makers (see smokeless powder makers)

Preparers (tannery)
Anthrax
Dampness
Septic infections

Preservative Makers and Handlers
Formaldehyde

Pressers
Carbon monoxide
Repeated motion, pressure and vibration

Pressmen (oil refining)
Dampness
Petroleum oils

Pressmen (printers) (see printers)

Pressroom Workers (rubber)
Amino compounds of benzol, toluol and xylol
Aniline
Antimony and its compounds
Arsenic and its compounds
Benzol and its homologues (toluol and xylol)
Petroleum hydrocarbons
Sudden variations of temperature

Primers (explosives)
Mercury and its compounds

Printers
Amino compounds of benzol, toluol and xylol
Aniline
Antimony and its compounds
Arsenic and its compounds
Benzol and its homologues (toluol and xylol)
Carbon monoxide
Carbon tetrachloride
Chlorinated hydrocarbons
Chlorobenzols
Cyanides
Defective illumination
Glycols
Inorganic dust (no free silica)
Lead and its compounds
Mercury and its compounds
Methyl alcohol
Petroleum hydrocarbons
Tetrachloroethylene
Turpentine

Printers Textile (see textile printers)

Puddlers (iron and steel)
Carbon monoxide
Heat
Manganese and its compounds

Pulp Mill Workers (see also paper makers)
Dampness
Excessive noise
Heat

Putty Makers
Carbon disulphide
Inorganic dust (no free silica)
Lead and its compounds
Petroleum hydrocarbons

Putty Polishers (glass)
Inorganic dust (no free silica)
Lead and its compounds

Pyrites Burners
Arsenic and its compounds
Heat
Hydrogen sulphide
Inorganic dust (no free silica)
Selenium compounds
Sulphur dioxide

Pyroxylin Plastics Workers
- Acetaldehyde
- Acetone
- Acrolein
- Amino compounds of benzol, toluol and xylol
- Amyl acetate
- Amyl alcohol
- Aniline
- Arsine
- Benzol and its homologues (toluol and xylol)
- Brominated hydrocarbons (ethylene dibromide)
- Butyl alcohol
- Carbon monoxide
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Cyanides
- Diethylene dioxide
- Ethyl acetate
- Ethylene glycol monomethyl ether
- Hydrogen sulphide
- Ketones
- Lead and its compounds
- Methyl alcohol
- Nitric acid
- Nitrous fumes
- Organic dust
- Petroleum hydrocarbons
- Propyl alcohol
- Sulphuric acid
- Tetrachloroethane
- Triorthocresyl phosphate

Quarrymen
- Excessive noise
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Repeated motion, pressure and vibration

Quartz Workers
- Inorganic dust (free silica)

Radioactive Paint Makers
- X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Radiologists
- X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Radio Tube Makers
- Mercury and its compounds

Radium Miners
- X-rays, radium and other radioactive substances, (radiothorium, mesothorium, etc.)

Radium Ore Reduction Workers
- X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Radium Specialists
- X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Rag Workers
- Anthrax
- Organic dust
- Septic infections

Rayon Makers
- Alkalis
- Ammonia
- Amyl acetate
- Arsine
- Butyl alcohol
- Carbon disulphide
- Chlorinated diphenyls
- Chlorinated hydrocarbons
- Chlorine
- Cyanides
- Dampness
- Diethylene dioxide
- Formaldehyde
- Heat
- Hydrochloric acid
- Hydrogen sulphide
- Methyl alcohol
- Methylene chloride
- Nitric acid
- Nitrous fumes
- Oxalic acid
- Sulphuric acid
- Tetrachloroethane

Reclaimers (rubber)
- Amino compounds of benzol, toluol and xylol
- Aniline
- Benzol and its homologues (toluol and xylol)
- Carbon disulphide
- Hydrochloric acid
- Lead and its compounds
- Phenol
- Sulphuric acid

Red Lead Workers
- Lead and its compounds

Refiners (metals) (see also particular occupation)
- Arsenic and its compounds
- Arsine
- Carbon monoxide
- Heat
- Hydrochloric acid
- Lead and its compounds
- Mercury and its compounds
- Nitric acid
- Nitrous fumes
- Selenium compounds
- Sulphur dioxide
- Sulphuric acid

Refiners (sugar) (see sugar refiners)

Refrigerating Plant Workers
- Ammonia
- Brominated hydrocarbons
- Carbon dioxide
- Chlorinated hydrocarbons
- Dampness
- Freon
- Methyl chloride
- Ozone
- Sudden variations of temperature

Refrigerator (mechanical) **Makers and Repair Men**
- Acrolein
- Brominated hydrocarbons (ethyl bromide, methyl bromide)
- Chlorinated hydrocarbons
- Freon
- Methyl chloride
- Methyl formate
- Sulphur dioxide

Resins (synthetic) **Makers**
- Acetaldehyde
- Acetone
- Alkalis
- Amino compounds of benzol, toluol and xylol.
- Aromatic hydrocarbons (naphthalene, anthracene)
- Benzol and its homologues (toluol and xylol)
- Chlorinated diphenyls
- Chlorinated hydrocarbons
- Chlorinated naphthalenes
- Cresol
- Dichloroethyl ether
- Diphenyl
- Ethyl acetate
- Ethylene glycol monomethyl ether
- Formaldehyde
- Furfural
- Heat
- Hexamethylene tetramine
- Methyl alcohol
- Nitrobenzols
- Organic dust
- Oxalic acid
- Phenol
- Selenium compounds
- Styrene
- Trichloroethylene
- Triorthocresyl phosphate
- Vinyl chloride

Riveters
- Excessive noise
- Lead and its compounds
- Repeated motion, pressure and vibration

Road Repairers
- Heat
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Tar and pitch

Roentgenologists
X-rays, radium and other radioactive substances, (radiothorium, mesothorium, etc.)

Roller Coverers (cotton mill)
Heat
Organic dust

Rollers (metals)
Heat

Roll Setters (iron and steel)
Heat

Roll Wrenchers (iron and steel)
Heat

Roofers
Lead and its compounds
Sudden variations of temperature
Tar and pitch

Roofing Material Workers
Asbestos dust
Heat
Inorganic dust (no free silica)
Tar and pitch

Rope Makers
Organic dust
Tar and pitch

Rotogravure Workers
Acetone
Amino compounds of benzol, toluol and xylol
Benzol and its homologues (toluol and xylol)

Roughers (iron and steel)
Heat

Rubber Cement Makers (see cement mixers, rubber)

Rubber Glove Makers
Petroleum hydrocarbons

Rubberized Asbestos Board Makers
Petroleum hydrocarbons

Rubber (synthetic) **Makers**
Acetaldehyde
Acrylonitrile
Amino compounds of benzol, toluol and xylol
Amyl alcohol
Aniline
Butadiene
Chlorine
Chlorobutadiene
Cresol
Isoprene
Naphthols
Naphthylamines
Nitrous fumes
Styrene
Sulphur chloride

Rubber Tire Builders
Benzol and its homologues (toluol and xylol)
Heat
Petroleum hydrocarbons
Repeated motion, pressure and vibration

Rubber Workers (see also particular occupation)
Acetaldehyde
Acetone
Alkalis
Amino compounds of benzol, toluol and xylol
Aniline
Antimony and its compounds
Arsenic and its compounds
Barium compounds
Benzol and its homologues (toluol and xylol)
Carbon disulphide
Carbon tetrachloride
Chlorinated hydrocarbons
Chromium compounds
Ethylene dichloride
Formaldehyde
Formic acid
Heat

Inorganic dust (free silica)
Inorganic dust (no free silica)
Lead and its compounds
Magnesium compounds
Methyl alcohol
Naphthols
Naphthylamines
Nitrous fumes
Organic dust
Petroleum hydrocarbons
Phenol
Pyridine
Repeated motion, pressure and vibration
Sudden variations of temperature
Synthetic rubber
Tellurium compounds
Tetrachloroethane
Trichloroethylene
Triorthocresyl phosphate
Turpentine

Sagger Makers
Dampness
Inorganic dust (no free silica)
Lead and its compounds

Sailors
Carbon monoxide
Sudden variations of temperature
Repeated motion, pressure and vibration

Salt Extractors (coke oven by-products)
Ammonia
Sulphuric acid

Salt Preparers
Heat
Inorganic dust (no free silica)
Sudden variations of temperature

Sand Blasters
Inorganic dust (free silica)
Inorganic dust (no free silica)

Sand Cutters
Inorganic dust (free silica)

Sanders
Inorganic dust (free silica)
Inorganic dust (no free silica)

Sanding Machine Operators
Inorganic dust (free silica)
Inorganc dust (no free silica),

Sandpaperers (enameling and painting auto bodies, etc.)
Inorganic dust (no free silica)
Lead and its compounds

Sandpaper Makers
Inorganic dust (free silica)
Inorganic dust (no free silica)

Sand Pulverizers
Inorganic dust (free silica)

Saw Filers
Defective illumination
Inorganic dust (no free silica)

Sawmill Workers
Organic dust

Sawyers
Repeated motion, pressure and vibration

Sawyers (stone)
Inorganic dust (free silica)
Inorganic dust (no free silica)

Scissors Sharpeners
Inorganic dust (no free silica)
Repeated motion, pressure and vibration

Scourers (belts)
Benzol and its homologues (toluol and xylol)

Scourers (metals)
Carbon tetrachloride
Chlorinated hydrocarbons
Nitric acid
Nitrous fumes
Petroleum hydrocarbons
Sulphuric acid
Trichloroethylene

Scourers (wood lasts) (shoes)
Organic dust

Scouring Powder Makers
Alkalis
Inorganic dust (free silica)
Inorganic dust (no free silica)

Scrapers (foundry)
Inorganic dust (free silica)
Inorganic dust (no free silica)

Screen Tenders (pulp mill)
Dampness

Screen Workers (lead and zinc smelting)
Inorganic dust (no free silica)
Lead and its compounds

Sealers (incandescent lamps)
Carbon monoxide

Sealing Wax Makers
Arsenic and its compounds
Turpentine

Seamstresses
Defective illumination
Repeated motion, pressure and vibration

Seed Treating Workers
Fungus infectons
Mercury salts
Organic mercury compounds
Phosphorus compounds

Selenium Refiners
Selenium and its compounds

Sewage Purification Workers
Chlorine

Sewer Workers
Aliphatic hydrocarbons (methane, etc.)
Ammonia
Carbon dioxide
Carbon monoxide
Dampness
Hydrogen sulphide
Petroleum hydrocarbons

Sewing Machine Operators
Defective illumination
Repeated motion, pressure and vibration

Shade Cloth Makers
Benzol and its homologues (toluol and xylol)
Petroleum hydrocarbons

Shale Oil Workers (see petroleum refiners)

Shavers (felt hats; fur; tannery)
Anthrax
Dampness
Organic dust
Septic infections

Shearers
Anthrax
Fungus infections
Undulant fever

Sheep Dip Makers
Arsenic and its compounds

Sheet Metal Workers
Lead and its compounds
Metal fume fever
Zinc and its compounds

Shellackers
Amyl acetate
Benzol and its homologues (toluol and xylol)
Butyl alcohol
Lead and its compounds
Methyl alcohol
Petroleum hydrocarbons
Turpentine

Shellac Makers
- Ammonia
- Amyl acetate
- Benzol and its homologues (toluol and xylol)
- Butyl alcohol
- Lead and its compounds
- Methyl alcohol
- Petroleum hydrocarbons
- Turpentine

Shell Fillers
- Dinitrophenol
- Nitrobenzols
- Nitro compounds of benzol, toluol and xylol
- Nitroglycerine
- Tetryl
- Trinitrophenol
- Trinitrotoluol

Shepherds
- Anthrax
- Fungus infections

Sherardizers
- Metal fume fever
- Zinc and its compounds

Shingle Stainers
- Petroleum hydrocarbons

Shipyard Workers
- Lead and its compounds
- Repeated motion, pressure and vibration
- Tar and pitch
- Ultraviolet and infrared rays

Shoddy Workers
- Anthrax
- Arsine
- Chlorine
- Hydrochloric acic
- Organic dust
- Septic infections
- Sulphuric acid

Shoe Dyers
- Amino compounds of benzol, toluol and xylol
- Lead and its compounds
- Nitrobenzols
- Nitro compounds of benzol, toluol and xylol

Shoe Factory Operatives (see also particular occupation)
- Acetone
- Amyl acetate
- Anthrax
- Benzol and its homologues (toluol and xylol)
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Organic dust
- Petroleum hydrocarbons
- Tetrachloroethane
- Trichloroethylene
- Turpentine

Shoe Finishers
- Ammonia
- Amyl acetate
- Amyl alcohol
- Benzol and its homologues (toluol and xylol)
- Ethyl acetate
- Methyl alcohol
- Petroleum hydrocarbons
- Sudden variations of temperature

Shoe Heel (wood) **Coverers**
- Acetone
- Amyl acetate
- Benzol and its homologues (toluol and xylol)
- Ethyl acetate
- Methyl alcohol
- Petroleum hydrocarbons

Shoemakers (see cobblers)

Shooting Gallery Workers
- Lead and its compounds
- Mercury and its compounds
- Nitrous fumes

Shot Makers
Antimony and its compounds
Arsenic and its compounds
Lead and its compounds

Silicon Alloy Makers
Inorganic dust (free silica)

Silk Weighters
Lead and its compounds
Tin compounds

Silk Workers
Organic dust
Septic infections

Silo Workers
Carbon dioxide

Silverers (mirror) (see mirror silverers)

Silver Foil Makers
Silver and its compounds.

Silver Melters
Carbon monoxide
Cyanides
Silver and its compounds
Sudden variations of temperature

Silver Miners (see also miners)
Arsenic and its compounds

Silver Nitrate Makers
Silver and its compounds
Nitric acid
Nitrous fumes

Silver Platers (see also electroplaters)
Silver and its compounds

Silversmiths
Silver

Singers (cloth)
Carbon monoxide

Sintering Plant Workers
Inorganic dust (no free silica)

Skimmers (glass)
Heat
Ultraviolet and infrared rays

Slag Workers
Heat
Inorganic dust (no free silica)

Slate Workers
Inorganic dust (free silica)
Inorganic dust (no free silica)

Slaughter House Workers
Anthrax
Dampness
Erysipeloid
Fungus infections
Glanders
Septic infections
Undulant fever
Weil's disease

Slip Makers (pottery)
Dampness
Inorganic dust (no free silica)
Lead and its compounds

Slushers (porcelain enameling)
Lead and compounds

Smelters (see also particular metal)
Heat
Inorganic dust (free silica)
Inorganic dust (no free silica)
Sulphur dioxide

Smokeless Powder Makers
Acetone
Amyl acetate
Amyl alcohol
Benzol and its homologues (toluol and xylol)
Carbon disulphide
Nitric acid
Nitrobenzol
Nitro compounds of benzol, toluol and xylol
Nitrous fumes
Nitroglycerine
Phenol
Trinitrophenol

Smoothers (glass)
- Dampness
- Inorganic dust (no free silica)

Soap (abrasive) **Workers**
- Inorganic dust (no free silica)
- Inorganic dust (free silica)

Soap Makers
- Acrolein
- Alkalis
- Amyl acetate
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Carbon tetrachloride
- Chlorinated hydrocarbons
- Dampness
- Dichloroethyl ether
- Ethyl acetate
- Ethylene dichloride
- Formaldehyde
- Formic acid
- Hydrochloric acid
- Hydrogen sulphide
- Manganese compounds
- Methyl alcohol
- Nitrobenzol
- Nitro compounds of benzol, toluol and xylol
- Organic dust
- Petroleum hydrocarbons
- Septic infections
- Sudden variations of temperature
- Sulphuric acid
- Tar and pitch
- Tetrachloroethane
- Tetrachloroethylene
- Trichloroethylene

Soda Makers
- Ammonia
- Arsine
- Carbon dioxide
- Chlorine
- Dampness
- Hydrogen sulphide
- Nitric acid
- Nitrous fumes
- Sulphuric acid

Sodium Hydroxide Makers
- Alkalis
- Chlorine
- Dampness

Sodium Silicate Makers
- Inorganic dust (free silica)

Sodium Sulphide Makers
- Hydrogen sulphide

Softeners (tannery)
- Organic dust

Solderers
- Arsine
- Cadmium and its compounds
- Carbon monoxide
- Cyanides
- Hydrochloric acid
- Lead and its compounds
- Solder
- Ultraviolet and infrared rays
- Zinc and its compounds

Solder Makers
- Antimony and its compounds
- Cadmium and its compounds
- Lead and its compounds
- Solder
- Zinc and its compounds

Spice Makers
- Organic dust

Spinners (asbestos)
- Asbestos dust

Spinners (textile)
- Organic dust
- Repeated motion, pressure and vibration

Spongers
- Dampness
- Heat

Sprayers (metals) (see metallizers)

Sprayers (paint) (see painters)

Sprayers (tree) (see also insecticide makers)
Arsenic and its compounds
Cyanides
D.D.T.
Lead and its compounds
Phosphorus compounds

Spreaders (rubber works)
Carbon tetrachloride
Sudden variations of temperature

Stablemen
Anthrax
Ammonia
Fungus infections
Glanders
Septic infections
Tetanus
Undulant fever

Stamp Mill Workers
Dampness
Heat
Inorganic dust (free silica)
Inorganic dust (no free silica)

Starch Makers
Carbon dioxide
Hydrogen sulphide
Organic dust

Statuary Workers
Inorganic dust (free silica)
Inorganic dust (no free silica)

Steam Fitters (see pipe fitters)

Stearic Acid Makers
Acrolein
Sudden variations of temperature

Steel Alloy Makers (see alloy makers)

Steel (chrome) **Makers**
Chromium compounds

Steel Engravers (see engravers)

Steeple Jacks
Carbon monoxide
Sulphur dioxide

Stereotypers
Antimony and its compounds
Lead and its compounds
Sudden variations of temperature

Still (coal tar) **Cleaners**
Benzol and its homologues (toluol and xylol)
Heat
Tar and pitch

Stillmen (carbolic acid)
Heat
Phenol

Stillmen, Operating (see also particular chemical)
Heat

Stitchers (shoes)
Methyl alcohol

Stockmen
Anthrax
Glanders
Septic infections
Tetanus
Undulant fever

Stockyard Workers (see slaughter house workers)

Stokers
Carbon monoxide
Heat
Inorganic dust (no free silica)
Sudden variations of temperature
Ultraviolet and infrared rays

Stone (artificial) **Makers**
Inorganic dust (free silica)
Inorganic dust (no free silica)
Tar and pitch

Stone Cleaners
- Hydrofluoric acid
- Oxalic acid

Stone Cutters
- Dampness
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Repeated motion, pressure and vibration

Stone Masons
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Stone Workers
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Storage Battery Makers
- Amyl acetate
- Antimony and its compounds
- Arsine
- Cadmium and its compounds
- Carbon monoxide
- Lead and its compounds
- Mercury and its compounds
- Nickel and its compounds
- Sulphur dioxide
- Sulphuric acid

Straw Cutters
- Fungus infections

Straw Hat Makers
- Acrolein
- Amyl acetate
- Chloride of lime
- Chlorinated hydrocarbons
- Ethyl acetate
- Formaldehyde
- Methyl alcohol
- Organic dust
- Petroleum hydrocarbons
- Sudden variations of temperature
- Tetrachloroethane

Street Cleaners
- Inorganic dust (no free silica)
- Organic dust
- Septic infections

Street Repairers
- Excessive noise
- Heat
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Repeated motion, pressure and vibration
- Tar and pitch

Subway Construction Workers
- Inorganic dust (free silica)

Sugar Refiners
- Ammonia
- Barium compounds
- Carbon dioxide
- Chlorine
- Dampness
- Heat
- Hydrochloric acid
- Hydrogen sulphide
- Inorganic dust (no free silica)
- Organic dust
- Septic infections
- Sudden variations of temperature
- Sulphur dioxide
- Sulphuric acid

Sulphates Makers
- Sulphuric acid

Sulphides Makers
- Hydrogen sulphide

Sulphite Cooks (pulp mill)
- Heat
- Sudden variations of temperature
- Sulphur dioxide

Sulphur Dioxide Makers
- Carbon monoxide
- Sulphur dioxide

Sulphuric Acid Workers
- Ammonia
- Arsenic and its compounds
- Arsine
- Hydrogen sulphide

Lead and its compounds
Nitric acid
Nitrous fumes
Selenium and its compounds
Sulphur dioxide
Sulphuric acid
Vanadium and its compounds

Sumackers (tannery)
Anthrax
Dampness
Septic infections

Surgical Dressing Makers
Phenol

Tablehands (tannery)
Anthrax
Dampness

Table Operatives (iron and steel)
Heat

Table Turners (enamelling)
Inorganic dust (no free silica)
Lead and its compounds
Sudden variations of temperature

Tailors
Repeated motion, pressure and vibration

Talc Workers
Inorganic dust (no free silica)

Tallow Refiners
Acrolein
Carbon disulphide
Chlorinated hydrocarbons
Septic infections
Sulphuric acid

Tank Cleaners (see also particular chemical)
Arsine
Benzol and its homologues (toluol and xylol)
Hydrofluoric acid
Petroleum hydrocarbons
Tar and pitch
Tetraethyl lead

Tank Men
Dampness
Heat

Tannery Workers
Alkalis
Amino compounds of benzol, toluol and xylol
Ammonia
Amyl acetate
Aniline
Anthrax
Arsenic and its compounds
Chloride of lime
Chromium and its compounds
Cyanides
Dampness
Formaldehyde
Formic acid
Hydrochloric acid
Hydrogen sulphide
Lead and its compounds
Mercury and its compounds
Oxalic acid
Petroleum hydrocarbons
Septic infections
Sulphur dioxide
Sulphuric acid
Trinitrophenol

Tapers (aeroplanes)
Chlorinated hydrocarbons
Tetrachloroethane

Tappers (smelting) (see also particular metal)
Heat
Ultraviolet and infrared rays

Tar (distillery) **Workers** (see also coal tar workers)
Arsenic and its compounds
Cresol
Heat
Tar and pitch

Taxidermists
- Anthrax
- Arsenic and its compounds
- Mercury and its compounds
- Organic dust
- Psittacosis
- Septic infections

Teasers (glass)
- Carbon monoxide
- Heat

Telegraphers
- Repeated motion, pressure and vibration

Telephone Linemen (trench work) (see also cable splicers)
- Carbon monoxide
- Cresol
- Dampness
- Electrical burns; electrical shock
- Lead and its compounds
- Petroleum hydrocarbons
- Phenol
- Poison ivy

Temperers
- Calcium cyanamide
- Carbon monoxide
- Cyanides
- Heat
- Lead and its compounds
- Mercury and its compounds
- Petroleum oils
- Sulphuric acid

Tetraethyl Lead Makers
- Bromine
- Lead and its compounds
- Tetraethyl lead

Textile (asbestos) **Workers**
- Asbestos dust

Textile Comb Makers
- Inorganic dust (no free silica)

Textile Finishers (see particular occupation)

Textile Printers
- Amino compounds of benzol, toluol and xylol
- Amyl acetate
- Aniline
- Antimony and its compounds
- Aromatic hydrocarbons
- Arsenic and its compounds
- Cadmium compounds
- Carbon monoxide
- Chlorinated hydrocarbons
- Chlorine
- Chlorobenzols
- Chromium and its compounds
- Cyanides
- Ethyl acetate
- Formaldehyde
- Glycols
- Heat
- Hydrochloric acid
- Lead and its compounds
- Manganese compounds
- Mercury and its compounds
- Methyl alcohol
- Nitric acid
- Nitrous fumes
- Petroleum hydrocarbons
- Phenol
- Sudden variations of temperature
- Sulphuric acid
- Turpentine
- Vanadium compounds

Textile Workers (see particular occupation)
- Dampness
- Heat
- Organic dust
- Sudden variations of temperature

Thallium Workers
- Thallium and its compounds

Thermometer Makers
- Mercury and its compounds
- Methyl chloride
- Thallium and its compounds

Thread Glazers
Heat
Sudden variations of temperature

Tile Makers (see also pottery workers)
Dampness
Heat
Inorganic dust (free silica)
Lead and its compounds
Sudden variations of temperature
Uranium compounds

Tin Foil Makers
Heat
Lead and its compounds

Tinners
Acrolein
Ammonia
Arsenic and its compounds
Arsine
Dampness
Heat
Hydrochloric acid
Lead and its compounds

Tin Plate Mill Workers (see iron and steel workers)

Tin Recovery Workers
Chlorine

Tire Builders (see rubber tire builders)

Tobacco Denicotinizers
Ethylene dichloride
Nicotine and tobacco
Trichloroethylene

Tobacco Moisteners
Carbon dioxide
Dampness

Tobacco Seedling Treaters
Benzol and its homologues (toluol and xylol)

Tobacco Workers
Nicotine and tobacco
Organic dust

Tongsmen (iron and steel)
Heat

Tool Makers
Inorganic dust (no free silica)

Top Fillers (foundry)
Carbon monoxide
Heat
Inorganic dust (no free silica)

Towermen (sulphuric acid) (see also sulphuric acid workers)
Arsine
Nitric acid
Nitrous fumes
Sulphur dioxide
Sulphuric acid

Toy Makers
Acetone
Amyl acetate
Arsenic and its compounds
Lead and its compounds

Train Despatchers
Defective illumination
Repeated motion, pressure and vibration

Transfer Workers (pottery)
Lead and its compounds
Turpentine

Transparent Wrapping Materials Workers
Acetone
Alkalis
Carbon disulphide
Chlorinated hydrocarbons
Hydrochloric acid
Hydrogen sulphide
Sudden variations of temperature
Sulphuric acid

Treaders (rubber)
Benzol and its homologues (toluol and xylol)
Petroleum hydrocarbons
Repeated motion, pressure and vibration

Tree Sprayers (see sprayers, tree)

Trichloroethylene Workers
- Trichloroethylene

Trinitrotoluol Makers
- Benzol and its homologues (toluol and xylol)
- Nitric acid
- Nitro compounds of benzol, toluol and xylol
- Nitrous fumes
- Trinitrotoluol

Tube Makers (glass)
- Heat

Tubulators (incandescent lamps)
- Carbon monoxide

Tumbling Barrel Workers
- Inorganic dust (free silica)
- Inorganic dust (no free silica)

Tunnel Workers
- Aliphatic hydrocarbons (methane, etc.)
- Carbon dioxide
- Compressed air (increased atmospheric pressure)
- Dampness
- Defective illumination
- Hydrogen sulphide
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Nitrous fumes

Turners-Out (glass)
- Heat

Turpentine Extractors
- Heat
- Turpentine

Type Cleaners
- Benzol and its homologues (toluol and xylol)
- Methyl alcohol
- Petroleum hydrocarbons

Type Founders
- Antimony and its compounds
- Lead and its compounds

Type Melters
- Acrolein
- Lead and its compounds

Type Setters (see compositors)

Typists
- Repeated motion, pressure and vibration

Upholsterers
- Anthrax
- Methyl alcohol
- Organic dust

Uranium Miners
- Uranium and its compounds
- X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Uranium Workers
- Uranium and its compounds
- X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Vanadium Steel Workers
- Heat
- Vanadium and its compounds

Vapour Curers (see vulcanizers)

Varnishers
- Acetaldehyde
- Acetone
- Amino compounds of benzol, toluol and xylol
- Amyl acetate
- Amyl alcohol
- Aniline
- Benzol and its homologues (toluol and xylol)
- Butyl alcohol
- Ethyl acetate

Ethylene chlorohydrin
Formic acid
Ketones
Lead and its compounds
Manganese compounds
Methyl alcohol
Paraldehyde
Petroleum hydrocarbons
Turpentine

Varnish Makers
Acetaldehyde
Acetone
Acrolein
Alkalis
Amino compounds of benzol, toluol and xylol
Ammonia
Amyl acetate
Amyl alcohol
Aniline
Barium compounds
Benzol and its homologues (toluol and xylol)
Butyl alcohol
Carbon disulphide
Chlorinated diphenyls
Chlorinated naphthalenes
Diethylene dioxide
Ethyl acetate
Ethylene chlorohydrin
Ethylene glycol monomethyl ether
Formic acid
Furfural
Ketones
Lead and its compounds
Manganese compounds
Methyl alcohol
Ozone
Petroleum hydrocarbons
Phenol
Sulphur chloride
Sulphur dioxide
Sudden variations of temperature
Turpentine

Varnish Remover Makers
Benzol and its homologues (toluol and xylol)
Dichloroethyl ether
Ketones
Methylene chloride
Petroleum hydrocarbons

Varnish Removers
Benzol and its homologues (toluol and xylol)
Dichloroethyl ether
Ketones
Methylene chloride
Petroleum hydrocarbons

Vatmen
Carbon dioxide
Dampness
Heat

Vat Varnishers (see varnishers)

Vault Workers
Carbon dioxide

Velvet Makers
Arsenic and its compounds
Heat

Veterinarians
Anthrax
Glanders
Septic infections
Undulant fever

Vignetters
Hydrochloric acid

Vinegar Workers
Carbon dioxide
Heat

Vintners
Carbon dioxide

Vinyl Chloride Makers
Vinyl chloride

Vulcanizers
Amino compounds of benzol, toluol and xylol
Ammonia
Aniline
Antimony and its compounds

Benzol and its homologues (toluol and xylol)
Carbon dioxide
Carbon disulphide
Carbon tetrachloride
Chlorinated hydrocarbons
Chromium compounds
Dampness
Heat
Hydrogen sulphide
Methyl alcohol
Petroleum hydrocarbons
Selenium compounds
Sudden variations of temperature
Sulphur chloride
Sulphur dioxide

Vulcanizers (steam)
Heat
Dampness

Wallpaper Printers
Amino compounds of benzol, toluol and xylol
Arsenic and its compounds
Chromium compounds
Formaldehyde
Heat
Lead and its compounds
Phenol
Sudden variations of temperature

Warehouse Workers
Anthrax

Washers
Dampness

Washers (metal) (see also degreasers)
Chlorinated hydrocarbons
Petroleum hydrocarbons

Washwomen
Dampness
Repeated motion, pressure and vibration

Watch Dial (luminous) **Painters**
X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Watchmakers
Defective illumination
Repeated motion, pressure and vibration

Waterproofers (paper and textile)
Benzol and its homologues (toluol and xylol)
Carbon tetrachloride
Chromium compounds
Formaldehyde
Petroleum hydrocarbons
Tar and pitch

Water Purifiers
Barium compounds
Chloride of lime
Chlorine

Wax Makers (see also petroleum)
Benzol and its homologues (toluol and xylol)
Chlorinated diphenyls
Chlorinated hydrocarbons
Chlorinated naphthalenes
Ozone
Sulphuric acid
Tetrachloroethane
Turpentine

Wax Ornament Makers
Acrolein
Arsenic and its compounds
Chromium compounds

Weavers
Organic dust
Repeated motion, pressure and vibration

Weavers (asbestos)
Asbestos dust

Weighers
Inorganic dust (free silica)
Organic dust

Welders
- Arsenic and its compounds
- Benzol and its homologues (toluol and xylol)
- Cadmium and its compounds
- Carbon monoxide
- Chromium and its compounds
- Copper
- Electrical burns; electrical shock
- Heat
- Hydrofluoric acid
- Lead and its compounds
- Manganese and its compounds
- Mercury and its compounds
- Metal fume fever
- Nitric acid
- Nitrous fumes
- Ozone
- Phosphine
- Phosphorus and its compounds
- Selenium and its compounds
- Ultraviolet and infrared rays
- Zinc and its compounds

Well Workers
- Aliphatic hydrocarbons (methane, etc.)
- Carbon dioxide
- Hydrogen sulphide

White Lead Workers
- Carbon dioxide
- Lead and its compounds

Window Shade Makers
- Benzol and its homologues (toluol and xylol)
- Petroleum hydrocarbons

Wire Drawers
- Arsenic and its compounds
- Hydrochloric acid
- Sulphuric acid

Wirers (incandescent lamps)
- Amyl acetate

Wood Alcohol Distillers
- Acetone
- Carbon monoxide
- Methyl alcohol

Wooden Heel Workers
- Anthrax

Wood Last Scourers (shoes)
- Organic dust

Wood Polishers (see furniture polishers)

Wood Preservers
- Arsenic and its compounds
- Cresol
- Dinitrophenol
- Mercury and its compounds
- Phenol
- Sulphuric acid
- Tar and pitch

Wood Stainers
- Chromium compounds
- Lead and its compounds

Woodworkers
- Methyl alcohol
- Organic dust
- Petroleum hydrocarbons

Wool Carders
- Anthrax
- Organic dust

Wool Scourers
- Acetone
- Ammonia
- Anthrax
- Dampness
- Sudden variations of temperature

Wool Spinners
- Anthrax
- Organic dust

Wool Workers (see also particular occupation)
- Anthrax
- Organic dust

X-Ray Machine Makers
- X-rays, radium and other radio-active substances (radiothorium, mesothorium, etc.)

X-Ray Photographers
- X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

X-Ray Technicians
- X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

X-Ray Tube Makers
- X-rays, radium and other radioactive substances (radiothorium, mesothorium, etc.)

Yeast Makers
- Acetaldehyde
- Carbon dioxide
- Hydrofluoric acid
- Sulphuric acid

Zinc Chloride Makers
- Arsine
- Chlorine
- Hydrochloric acid
- Zinc compounds

Zinc Electrode Makers
- Mercury and its compounds

Zincers
- Cyanides

Zinc Miners (see also miners)
- Arsenic and its compounds
- Lead and its compounds
- Manganese and its compounds

Zinc Smelters and Refiners
- Antimony and its compounds
- Arsenic and its compounds
- Cadmium and its compounds
- Carbon monoxide
- Heat
- Inorganic dust (free silica)
- Inorganic dust (no free silica)
- Lead and its compounds
- Manganese and its compounds
- Metal fume fever
- Selenium and its compounds
- Sulphur dioxide
- Tellurium and its compounds
- Zinc and its compounds

Zoological Technicians
- Anthrax
- Glanders
- Psittacosis
- Septic infections
- Tetanus
- Undulant fever

CHAPTER III

HARMFUL CONDITIONS AND SUBSTANCES

Section A. Abnormalities of Air Pressure

Exposure to abnormally high atmospheric pressures occurs in occupations where compressed air shafts and caissons are used, as in the construction of bridge piers, building foundations and traffic tunnels, and in diving operations. Careful, slow compression and decompression are essential. Too sudden an increase in pressure may cause rupture of the eardrums with haemorrhage from the ears. While under compression, the blood and tissue fluids take up increased amounts of air. When the pressure is decreased too rapidly, bubbles of nitrogen form gas emboli which block off the blood supply to various parts of the body, producing the symptoms of "bends", "compressed air illness", or "caisson" disease. Usually symptoms appear within the first few hours following too rapid decompression but may not develop for 12 to 48 hours. The most common symptom is severe pain in the muscles and joints of the arms and legs. Itching and rash, vertigo, nausea, vomiting, epigastric pain, fatigue, dyspnoea and shock may occur. In some cases symptoms related to the central nervous system are apparent, such as paralysis of the skeletal muscles or bladder. In others, symptoms due to cardiac failure or pulmonary emboli are evident. Death may occur rapidly or may follow secondary complications.

First aid measures involve the patient's return to the compression chamber where he is slowly and carefully decompressed. In certain provinces, there are regulations limiting the periods of exposure permitted at various atmospheric pressures, and requiring all compressed air workers to wear identification badges. These badges direct that, in an emergency, the wearer be taken to the nearest compressed air lock, the location of which is given on the badge.

Exposure to abnormally low atmospheric pressures occurs in aviation and in work at high altitudes. As such exposures are the concern of specialized medical departments, they are not discussed here.

Section B. Abnormalities of Temperature and Humidity

1. Heat

Excessively high temperature and humidity occur in many industries, principally as a result of the heat and moisture produced by industrial processes. This may be accentuated during the summer months by a high external temperature. Very high temperatures may bring about heat cramps, heat exhaustion or heat stroke. Temperatures which are not high enough to cause these acute conditions may result in loss of efficiency, increased liability to accidents, and increased absenteeism.

HEAT CRAMPS

Heat cramps occur in persons who are sweating profusely as a result of heavy physical work performed in hot environments. The characteristic symptoms are due to spasmodic contractions of the muscles of the extremities and abdominal wall. The body temperature is usually normal or slightly elevated, the pulse rate slightly increased, the blood pressure normal and the skin moist. Nausea and vomiting may occur. The cramps disappear completely and rapidly following intravenous injection of physiological saline and can be prevented by oral administration of salt and water. Further evidence that the loss of salt and water from the body is the responsible factor is presented by experiments in which human subjects on salt-free diets experienced cramps on being subjected to temperatures sufficiently high to induce sweating.

HEAT EXHAUSTION

This term is applied to many complaints occurring in hot weather, the symptoms varying from a sensation of fatigue to complete collapse. The majority of clinical heat cases fall into this group. Usually the symptoms begin with a feeling of dizziness, fatigue and headache, sometimes accompanied by nausea. These may proceed to a state of unconsciousness, or complete collapse may occur without warning. The body temperature may be low or elevated, usually not above 102°F. The skin is moist and is hot or cold depending on the state of the patient. The pulse is rapid and weak, the blood pressure usually being low. The clinical picture is that of shock. Emergency treatment should be directed toward controlling shock and replacing chloride loss.

HEAT STROKE

This is often called sunstroke when the cause is the radiant heat from the sun. In industrial workers, heat stroke occurs most frequently during prolonged heat waves. The onset may be sudden without any marked prodromal symptoms, although in some cases a period of mental excitement precedes the coma. The outstanding symptom is the high body temperature which is usually between 107°F. and 110°F. The pulse is rapid and full except in the late stages, and the blood pressure is somewhat elevated. The respiration is laboured and rapid. The face is flushed and the skin is usually hot and dry. The nervous system is depressed, incontinence is common and vomiting may occur. The state of coma may last for hours or days. Emergency measures are aimed at lowering body temperature.

2. Sudden Variations of Temperature

Rapid changes in temperature may contribute to an increase in respiratory disease, particularly pneumonia, and rheumatic and neuralgic conditions. Air movement is desirable, but uncomfortable drafts should be avoided, especially if the temperature of the draft is such that it will cause a rapid change in room temperature.

Section C. Dampness

Exposure to dampness occurs in many occupations. Tankmen, vatmen, and washers, for example, may be required to work in wet clothes continually. Such exposures may be contributing factors to rheumatic and respiratory conditions.

When dampness is a feature of any industrial process, work places should be supplied with drain channels, and adequate waterproof clothing should be provided. Where the occupation makes wet conditions unavoidable, workers should change to dry clothing before leaving the plant.

Section D. Defective Illumination

Defective illumination, characterized by insufficient light, glare, improper diffusion and distribution of light and unsuitability of colour may cause eye fatigue and headache. In addition, defective illumination reduces working efficiency and is a very frequent cause of industrial accidents.

In some industries, it may be a comparatively simple matter to provide good illumination while in others one may require the advice of illuminating engineers. For those who have need for technical knowledge on this subject, a useful reference work is "Recommended Practice of Industrial Lighting," prepared by the Illuminating Engineering Society and available from the Department of Labour, Ottawa. It should be borne in mind that, in fine work, magnification with good illumination may be more effective than high intensity alone.

Nystagmus has been reported in British coal miners and in train despatchers in America. In the case of coal miners, the condition is attributed to the low illumination of the coal face combined with the necessity of working in cramped positions. In train despatchers, the condition is thought to be due to the constant shifting of the eyes associated with the recording of data on the train sheet.

Ultraviolet light is an occupational hazard which frequently causes acute conjunctivitis and photophobia. This hazard is discussed in Section F, under "Radiant Energy."

Section E. Excessive Noise

The effects produced by excessive noise include fatigue, impaired hearing, decreased efficiency and emotional disturbances.

Noise can be measured by the sound level meter, which determines the intensity of a sound in the immediate environment in terms of decibels. The noise level produced by a riveting machine is about 100 decibels; in a large stenographic room it is about 70 decibels; the intensity of sound in ordinary conversation varies from 35 to 65 decibels. The normal threshold of audibility is 0 decibels. Intensities above 80 decibels are annoying and uncomfortable, and noise levels of 100 to 130 decibels have been found to cause temporary and permanent deafness. Levels above 130 decibels are painful to the ear.

Intensity is not the only factor in the evaluation of a noise problem. Industrial noise is usually a combination of sounds of different frequencies. By measuring the intensity of sound produced at each frequency and determining the frequency of vibration of various parts of a machine, it is often possible to establish the main sources of a noise and take steps to eliminate or reduce it.

By means of an audiometer, the hearing loss in decibels may be determined at each frequency. Investigations of deafness due to noise have shown that the initial loss in acuity occurs for sounds of

relatively high frequency, particularly in the range between 2896 and 5792 cycles per second. Following brief exposures to high noise levels the loss is temporary, recovery taking place over a period of several days or possibly weeks. With repeated exposure on successive days, the loss is cumulative, and though improvement may be expected following removal from exposure, some permanent loss may occur. Exposure during the work day to sustained noise of 100 decibels or higher for several months is usually considered capable of causing permanent partial impairment of hearing. As long as the impairment is limited to frequencies above the speech range, the loss in acuity does not constitute a serious handicap. However, with continued exposure to high noise levels the range of frequencies for which there is decreased acuity spreads. The ability to hear sounds of lower frequency decreases and the individual finds it increasingly difficult to hear the conversational voice. The greater the loss in acuity for conversation, the more seriously is the person handicapped.

In assessing the importance of noise in producing deafness, a careful search must be made for those organic conditions which are recognized as causing deafness. There is considerable individual susceptibility to the effects of noise. Allowance should also be made for the normal deterioration in hearing which occurs with advancing age. This loss has been estimated to average 5 to 10 decibels, for sounds having frequencies of 4096 and 5792 cycles per second, for each decade after the age of 30. As the loss at these frequencies increases in magnitude, the neighboring frequencies become involved.

Noise may be prevented, or eliminated by one or more of the following methods:

(a) The elimination of noise at its source.

(b) Isolation of noisy operations.

(c) Reduction of noise by sound insulation.

(d) Use of personal protective devices against noise.

Section F. Radiant Energy

Radiations may be divided into two types, corpuscular and electro-magnetic radiations. In general, corpuscular radiations exhibit properties commonly associated with particles, whereas electro-magnetic waves exhibit wave-like properties. However, the two types of radiations demonstrate certain properties in common.

Corpuscular Radiations: These are produced by the natural or artificial disintegration of atomic nuclei. The particles emitted may be of three kinds—alpha particles, beta particles and neutrons.

Alpha particles are the comparatively large, positively charged nuclei of helium. They are relatively slow-moving (about 1/20th of the speed of light) and have low penetrating power, being stopped by a few centimetres of air, a sheet of paper, or 1/20th of a millimetre of living tissue. However, because of their heavy mass and great momentum, they can disrupt molecules with which they collide, and can kill tissue cells lying in their path.

Beta particles are very small, negatively charged electrons, having velocities approaching the speed of light. They can penetrate 1 metre of air, or 1 cm. of water.

Neutrons are uncharged particles having a mass equal to that of the hydrogen atom, and possessing great penetrating power. Their industrial application is limited, and they are not ordinarily encountered as a hazard to health.

Electro-magnetic Radiations: These are radiations possessing wave-like properties such as reflection, refraction, dispersion, interference and diffraction. The most important electro-magnetic radiations are gamma rays, X-rays, ultraviolet and actinic rays, visible radiation, infrared or heat rays, and radio waves of various lengths. These radiations differ primarily in their wave-lengths.

Gamma rays are the shortest electro-magnetic radiations commonly encountered, having wave-lengths ranging from 0·005 to 1·3 angstrom units. (An angstrom unit is one ten-millionth of a millimetre.) Their penetrating power is very great, the shorter gamma rays passing through many inches of steel. By knocking electrons out of atoms through which they pass, they produce secondary beta radiations.

The common X-rays possess wave-lengths ranging from 0·01 to 10 angstrom units. They are less penetrating than gamma rays, being stopped by several centimetres of lead. They are also capable of producing secondary beta radiation, and owe their therapeutic use in medicine to this property.

Ultraviolet rays and actinic rays have wave-lengths of 2,500 to 4,500 angstrom units.

Visible radiation, or light rays, have wave-lengths ranging from 4,000 to 8,000 angstrom units.

Infrared, or heat rays have wave-lengths of over 7,000 angstrom units.

Radio waves possess wave-lengths ranging from several centimetres (*e.g.*, radar) to around 3,000 metres in length. In ultra-short-wave diathermy, wave-lengths of 3 to 12 metres are used; in short-wave diathermy the wave-length ranges from 12 to 30 metres; and in long-wave diathermy the wave-lengths vary from 200 to 600 metres. Recently, high-frequency machines have been introduced into industry for the drying of glues in plywoods, surface glazing of pottery and ceramics, and the fireless cooking of foods. These machines, for the most part, produce radiations having wave-lengths ranging from several centimetres to 30 metres. In other processes, as in the surface hardening of steel, lower frequency machines may be used, producing radiations with wave-lengths up to 3,000 metres.

1. X-Rays, Radium and Other Radioactive Substances

X-RAYS

X-rays are used industrially for fluoroscopy and radiographic purposes. As yet, their use in Canadian industry is limited, but undoubtedly will be expanded in various fields including the examination of metal castings for flaws, diffraction analyses, screening of materials for foreign bodies, and in other methods for controlling the quality of the product. The use of X-rays for fluoroscopic screening involves the greater risk of injury; the danger from scatter radiation during radiography, though not as great, is less obvious, and is usually less well guarded against.

Physiological Action: X-rays act on the body tissues by displacing electrons from the atoms through which they pass. This property, when properly controlled, is utilized in X-ray therapy; when not controlled, damage to the tissues may result. The commonest injury produced consists of damage to the skin; this may be only an area of acute erythema, which gradually disappears in the course of a few weeks; if severe it may develop into necrosis and ulceration. Such lesions are very slow to heal. Acute X-ray burns probably never initiate cancer; on the other hand, repeated exposure can give rise to a chronic dermatitis, occurring usually on the backs of the hands, with loss of the hair and atrophy of hair follicles, obliteration of the small blood vessels, hyperkeratosis and warty growths, cracking of the epithelium, areas of pigmentation, and, later, ulcerative changes and

destruction of the finger nails. The condition may progress to cancer, though no cases of X-ray cancer have yet been reported in industry.

Other effects of X-rays on the body consist of depression of the bone marrow, characterized by a leucopenia, and may culminate in severe and even fatal aplastic anaemia if allowed to progress. Sterility also has been reported in both male and female radiographers.

Constitutional symptoms, consisting of fever, nausea, vomiting, diarrhoea, abdominal pain, prostration and dyspnoea, may follow severe exposures. Death has been reported from such radiation sickness, occurring usually about the fourth day.

Recommended M.A.C.: The dose of X-radiation which is considered safe for repeated exposure has been set at 0·1 to 0·2 roentgen per day by various authorities. The lower figure is the one generally accepted at the present time.

RADIUM AND OTHER RADIOACTIVE SUBSTANCES

The use of radium and radioactive materials and their salts in industry has been confined chiefly to the painting of luminous dials for watches and other instruments. Exposure also occurs in the manufacture of radioactive paints, in the mining of radioactive ores and the handling of their products, in the making of mantles for gasoline lamps, and in the manufacture of radon seeds. With the recent development of radioactive isotopes of metallic and non-metallic elements, there is a definite possibility that their use in industry, at least by industrial research workers, will result in an increased hazard from these materials.

There are three main groups of naturally-occurring radioactive elements, the best known being the uranium group. By the process of radioactive disintegration, uranium passes through a long series of decay products, of which radium is probably the most familiar, and finally becomes lead. During the decay process, alpha, beta and gamma rays are produced. At one stage in the process following the disintegration of radium, a gas is formed known as radon, or radium emanation. It, in turn, gives off alpha particles and becomes radium-A, a solid. The other two groups of radioactive materials are derived from thorium and actinium, both of which furnish similar series of disintegration products.

Mode of Entry into Body: Radioactive materials may act locally on exposed surfaces. The alpha rays have low penetrating power, being

stopped by 1/20th mm. of tissues. Beta rays have slightly greater penetration power but their chief action is on the skin or nearby tissue. Gamma rays possess great penetrating power, and exert their action chiefly on the blood forming organs. Radon, being a gas, can be inhaled. Poisoning also has occurred following the ingestion of radioactive paint through the practice of pointing the paint brush with the lips, and through inhalation of radioactive dusts.

Physiological Action and Toxicity: Locally, exposure of the skin for several hours to radium results in the production of a dermatitis. After a latent period of several days, an erythema develops which may progress to form a slow-healing ulcer. Repeated exposures may result in a chronic atrophic dermatitis. The effect is probably due to beta rays. The gamma rays, because of their greater penetrating power, affect the bone marrow, causing a leucopenia and an anaemia. If exposure is continued, aplastic anaemia, which may be fatal, may result. Ingested radium is largely excreted through the gastro-intestinal tract. A small proportion is absorbed and stored as the insoluble sulphate in the bones where it continues to give off radiations, consisting of 92% alpha rays, the remainder beta and gamma rays. After several years' bombardment of the surrounding tissues, the alpha rays may produce severe or fatal aplastic anaemia, rarefying osteitis with spontaneous fracture, bone sarcoma, or necrosis of the mandible. From 2 to 10 micrograms (0·002 to 0·01 mgm) of radium distributed over the whole skeleton is sufficient to produce fatal results. Once stored in the bones, there is no known method by which it can be eliminated. It has been suggested that the high incidence of lung cancer in European miners of uranium and radioactive cobalt ores is due to the inhalation of radon gas. This theory has not been proven.

Recommended M.A.C.: The recommended maximum allowable dose of gamma radiation is 0·1 roentgen per day. The M.A.C. for radon and thoron is 10^{-8} curies per cubic metre of air. The maximum amount of radium fixed in the body which is tentatively regarded as harmless (the tolerance value) is 0·1 microgram.

2. Ultraviolet Rays

Ultraviolet and actinic rays ("black light") are invisible, having shorter wave-lengths than blue and violet light. The "near" ultra-

violet rays which have wave-lengths of over 3,000 angstrom units are much less harmful than the shorter ultraviolet rays having wave-lengths of 2,400 to 3,000 angstrom units.

Uses and Occurrence: Exposure to ultraviolet rays occurs chiefly in arc and electrowelding. The intensity of the ultraviolet radiations increases with the temperature of the welding process. Exposure may also occur in the use of ultraviolet rays for inspecting razor blades, blue prints and golf balls, and in the examination of bedding for presence of used materials; ultraviolet rays are also used in the diagnosis and treatment of skin diseases.

Physiological Action: The effects of ultraviolet radiations are exerted chiefly on the eyes, skin, superficial blood vessels and the haemopoietic system. Symptoms are not produced at the time of exposure, but follow a latent period of 4 to 12 hours. The skin develops an erythema resembling sunburn, which is followed in several days by freckling and pigmentation, the latter being a protective reaction. The blood shows an increase in the number of red cells, and a lymphocytosis. Exposure of the eyes to ultraviolet rays even for a few seconds may result, after the latent period, in the development of an ophthalmia, with acute conjunctivitis, lachrymation, photophobia and burning, and sometimes chemosis. In severe cases, there may be corneal ulceration and iritis. A few authorities believe that the lens may be injured, with cataract as a final result. The intensity of the radiations decreases rapidly as the distance from the source is increased, and harmful effects, either on the skin or the eyes, are not usually observed when the individual is more than 25 feet from the source.

Recommended M.A.C.: No maximum intensity values have been set. Prevention lies in screening the worker by the use of suitable goggles, face shields and protective clothing.

3. Infrared Rays

Infrared radiations (so-called "heat" rays) are invisible, having longer wave-lengths than those of visible light. On penetrating the skin for a short distance they are converted into heat.

Uses and Occurrence: Infrared rays are given off in such high temperature processes as welding, glassblowing, foundry work and other occupations where metal and glass are heated to the molten

state. In these processes, there may be a concomitant exposure to ultraviolet rays and to excessive light radiations. Infrared rays are utilized in industry in the heating and drying of many materials, *e.g.*, painted and lacquered objects.

Physiological Action: Exposure of the body to infrared radiation causes a vasodilation of the skin vessels and stimulation of cellular activity, thus promoting tissue repair and healing. In excessive doses, it produces an erythema resembling sunburn, which may be followed by pigmentation and freckling. Workers doing hard physical labour under exposure to intense heat radiations and high temperatures, may be subject to violent cramps of the abdomen and limbs. Heat cramps result from a severe metabolic disturbance which includes dehydration of the tissues and loss of salt, and are prevented by replacement of the body chlorides which have been excreted in the sweat. The most serious effect of prolonged exposure to infrared radiations is the development of cortical cataract at the posterior pole of the crystalline lens. The character of the cataract differs from that of ordinary senile cataract, which develops in the form of radiating lines from the periphery, the central pupillary area remaining free. The posterior cortical form of cataract is usually found in workers over 35 years of age who have been exposed for some years to the glare from molten glass or metal, or from furnaces.

Recommended M.A.C.: No value for the safe intensity of infrared rays has been set.

4. Ultra High Frequency Radiations

Though of relatively little importance in industry at the present time, short waves, or high frequency radiations will be more widely used in industrial processes requiring surface heating and internal heating of manufactured objects.

Physiological Action: Beyond their use in medicine, little study has been devoted to the effects of high frequency fields upon the body. Their application in medicine depends upon their heating effect on those parts of the body within the field. The resulting local increase in blood circulation probably assists in reducing oedema and promotes resolution of septic processes. A selective effect of the short wave field upon various organs has not been conclusively proven.

Occasionally, the use of short wave machines in industry has been reported to cause complaints of tiredness, exhaustion, headache, and heat. Other symptoms complained of are chills, numbness and dizziness. Complaints disappeared within several hours after leaving the short wave field, and no serious effects have been reported. Investigations into the effect of radar waves on the haemopoietic system and reproductive organs have been reported as showing no evidence of either depression or stimulation.

Section G. Electrical Burns; Electrical Shock

The effects of electrical currents upon the body depend upon a variety of external and individual conditions. Generally speaking, the higher the voltage involved, the greater will be the current through the body. Wetness or dampness of the skin also increases the strength of current through the individual by lowering the skin resistance. The longer the duration of contact, the greater will be the damage to the tissues through which the current passes. In its passage through the body, the current takes the shortest route between the contacts and affects chiefly the organs through which it passes, the severity of the damage depending mainly on the strength and duration of the current.

When the current passes through the brain, the patient may be rendered unconscious, either briefly or for some hours. If the respiratory centre is affected, there will be depression or even paralysis of respiration. Voltages of more than 1,000 tend to produce paralysis of the respiratory centre alone; voltages less than 220 tend to produce ventricular fibrillation, while voltages between 220 and 1,000 may affect both the heart and the respiratory centre. Death is caused by *(a)* ventricular fibrillation, *(b)* failure of the respiratory centre, *(c)* a combination of the first two causes, or *(d)* prolonged tetanus of the respiratory muscles resulting in suffocation.

When the current does not pass through any of the vital organs causing immediate death, other parts of the body may be affected, resulting in functional or structural damage which may be temporary or permanent. Temporary total disability is not uncommon while permanent total disability is rare. Permanent disability is usually the result of burns in tissues directly affected by the current. Electrical burns vary in degree and sometimes cause destruction of deep tissue which is not apparent upon superficial examination.

Other effects of electrical shock include injuries sustained by mechanical trauma when the worker is thrown to the ground by the spasmodic contraction of his muscles.

In the emergency treatment of electrical shock, the first step is the removal of the victim from the electrical contact. At the same time, the rescuer himself must take suitable precautions to avoid contacting the current through the body of the victim. When separation from the contact has been effected, if breathing has ceased, artificial respiration should be started immediately by the Schäfer or other reliable method and continued without interruption.

Section H. Repeated Motion and Pressure; Vibration

In many industrial processes, certain muscles or groups of muscles are used to a greater extent than in ordinary life, causing new employees to develop muscular stiffness and soreness. Certain occupations which subject the skin of the hands, feet, or other parts of the body to excessive friction, may produce blisters and abrasions. In these cases, the body must gradually adjust itself to more severe muscular effort, or must develop compensatory hardening and thickening of the skin. Occasionally, the employee has to be given lighter work during the period of adjustment.

In certain trades such as carpentry and brick-laying, the resumption of work in the spring after the winter lay-off requires the use of poorly conditioned muscles in the repetitive movements of hammering, sawing, etc. At the same time, these muscles must maintain some degree of contraction in gripping the tool and are subject to the effects of repeated shocks or blows. These conditions frequently lead to the development of tenosynovitis, bursitis, and myositis, causing pain and swelling in the tendons, joints or muscles used. The affected part may require complete rest until the condition clears.

"Occupational cramps" are associated with the prolonged performance of repeated muscle movements and occur among telegraphers, writers, typists, pianists, cotton twisters and drapers. Cramps or spasm, tremor and weakness may develop in the muscles concerned when attempting to do the repetitive task. Function is unimpaired for the performance of other muscular movements. Evidence of muscular wasting, or other signs of paralysis are rarely present. In the absence of muscular or nerve lesions, occupational muscular

cramps are believed to be a form of psychoneurosis related to emotional instability and precipitated by such factors as prolonged fatigue, and unsatisfactory adjustment to the job.

Portable Vibrating Tools

These tools are of two main types—*(a)* the piston-operated pneumatic tools, such as road and rock drills, chipping hammers or pneumatic chippers, and riveting hammers, *(b)* the newer rotating tools which consist essentially of a handle through the centre of which is a rotating spindle carrying a head, and driven by an electric motor or compressed air; the heads are of various kinds, and may be used for polishing, cutting or grinding. Both types of tools may be carried in the hands. In some arrangements, the rotating tool may be stationary while the article being worked upon is carried in the hands. In either case, the vibrations of the tool are transmitted to the hands and arms of the operator.

The clinical effects resulting from prolonged use of piston-operated tools have been recognized for some years. The commonest effect is a disturbance of the blood vessels of the fingers which is clinically indistinguishable from Raynaud's disease, the fingers becoming white or "dead", numb when exposed to cold, and later developing hyperaemia, cyanosis and a burning sensation when removed from the cold. The condition may develop after two years of exposure to the vibrations, but usually requires longer exposure. In rare cases, the muscles, fasciae and peripheral nerves may be affected, resulting in wasting of muscles of the thenar and hypothenar eminences. Injury to the joints, particularly the right elbow, with limitation of extension and pain may also occur in a few cases. The factors responsible for these effects have not been clearly established; the frequency and amplitude of vibration, strength of grip, manner in which the tool is held, exposure of the hand to air blown from the tool and other factors have all been incriminated. At present, it appears that the development of "dead fingers" is most often associated with the use of tools which vibrate with frequencies ranging between 2,000 and 7,500 beats per minute.

During the past war, a condition associated with the use of high-speed rotating tools was reported in the United States. The tools were used for grinding burrs from aluminum alloy castings, and the emery head rotated at about 25,000 revolutions per minute. About 12 per cent of the workers exposed were affected, the chief complaints

being burning, throbbing pain in the fingers holding the tool, continuous sharp pain radiating up the forearm, sometimes to the shoulder, stiffness and weakness of the hand and fingers, usually with swelling, and loss of sensation in that part of the hand in contact with the tool. The skin of the affected hand was atrophic and glossy; there were no colour changes in the skin and the response to heat was usually normal, but the capillaries of the nail beds were decreased in numbers and showed morphological changes. The condition developed in from 1 to 72 weeks after starting the job, the average being 19 weeks. The prognosis was poor, only a few mild cases showing improvement after a year's removal from exposure.

Section I. Infections

Infections, both localized and systemic, are frequently of occupational origin. Anthrax, fungus infections, undulant fever, and "septic infections" are typical examples. A brief summary of these and certain other infectious diseases which may be occupational in origin is given below.

1. Anthrax

(Malignant Pustule, Wool-Sorter's Disease)

This disease occurs in sheep and cattle and may be transmitted to man by direct contact through the skin and mucous membrane. Clinically, the disease takes an external (malignant pustule) or internal (pulmonary and rarely intestinal) form.

In man the most important portal of entry of the infection is through cuts and abrasions in the skin. The primary lesion is usually on the face or hands. It begins as a small red spot which soon forms a vesicle with surrounding induration. The bacillus of anthrax can usually be isolated from the vesicle fluid. The vesicle then develops a black gangrenous centre with spreading oedema and adjacent crops of vesicles which coalesce and eventually form a large, hard and necrotic area from which a slough separates. There is no suppuration. General symptoms include moderate fever, headache, nausea and muscular pain.

In the respiratory form of the infection (wool-sorter's disease) the prognosis is grave. Intestinal anthrax is not considered an industrial hazard.

2. Erysipeloid

Erysipeloid is caused by the bacillus of swine erysipelas. Risk of exposure is greatest among abattoir workers and persons handling fish and meat products. The most frequent form of the infection in man is mild, manifesting itself as a small red spot on the hands or fingers, which spreads slowly to adjacent parts and clears in the centre. The colour is characteristically violaceous or purplish-red. The infection is usually accompanied by itching and burning sensations. Low-grade fever, headache, malaise and lymphangitis may be present. The condition clears up spontaneously within four to six weeks.

3. Glanders

(Farcy)

This is primarily a disease of the horse, ass, or mule but may be conveyed to man by the nasal and other secretions of affected animals. It is caused by B. mallei, which enters the human system through a break in the skin or mucous membrane. The disease is seen in two forms, the acute and the chronic, both of which are characterized by fever, glandular enlargement, cutaneous lesions, and inflammation of the respiratory tract.

The distinctive skin lesion is a red, painful swelling which arises at the site of entry and breaks down to form a rapidly spreading ulcer. The adjacent glands become swollen and tender and frequently develop abscesses. As the disease progresses, similar lesions appear on other parts of the skin. Characteristic nodules or "farcy buds" form along the lymphatics at a distance from the site of inoculation and tend to break down into discharging ulcers. In acute cases there is a characteristic bloody and offensive nasal discharge. Very few acute cases recover. The organism may be isolated from the pustular lesions or nasal discharge. The serum gives a positive test for complement fixation.

4. Fungus Infections

Many fungus infections are of minor clinical importance while others may produce marked disability and death.

Skin diseases are caused by monilia, yeasts, dermatophytes, (ringworm fungi), sporotrichum, blastomycetes, as well as a great many other fungi.

Fungus disease of the lungs may be due to blastomycetes, actinomyces, monilia, coccidioides immitis, cryptococcus, and aspergillus. The symptoms of chronic cough, fatigue, loss in weight and slight fever resemble those of pulmonary tuberculosis, from which it is often difficult to differentiate. The radiographic appearance varies, but may resemble tuberculosis, secondary carcinoma or other pulmonary lesions. The sputum usually reveals large numbers of fungi of predominantly one type.

5. Psittacosis

(Ornithosis)

This is an infectious disease occurring among parrots, parakeets and other species of birds. It is due to a filterable virus which is transmitted directly from the infected bird to man. The characteristic lesion in man is an atypical bronchopneumonia which runs a typhoid-like course.

The onset may be abrupt or insidious. The initial symptoms are malaise, anorexia, fever, headache and backache. Chills may occur. There is cough, which is usually non-productive. The respiratory symptoms, although present, are not severe, and the physical signs of pulmonary consolidation appear gradually and with irregular distribution. In the acute phase, the virus may be isolated from the sputum. In the later stages the serum gives a positive test for complement fixation.

The prognosis is influenced by the source of the virus, the age of the patient and the extent of the pneumonia.

6. Rabies

(Hydrophobia)

This is an acute infectious virus disease, transmitted in the saliva of a rabid animal. Man is usually infected by the bite of a rabid dog. The disease is characterized by a variable incubation period, usually of 3 to 6 weeks, symptoms of great excitability, mental derangement and hyperaesthesia, followed by paralysis and death. Diagnosis is established by a history of being bitten and the recognition of Negri bodies in the brain of the suspected animal.

7. Tetanus

(Lockjaw)

Tetanus is an infectious disease in which the symptoms are due to the toxin produced by the causative agent Cl. tetani. This anaerobic organism is found in the soil and gains entrance to the body through wounds, particularly penetrating ones.

The incubation period is from one to three weeks. Symptoms begin with stiffness of the jaw and a sense of apprehension and restlessness. As the disease progresses, the muscles of the neck and back become rigid and stiffness in the jaws is replaced by actual difficulty in opening them. One of the most characteristic features is the development of painful convulsions which are precipitated by relatively slight stimuli. The terminal stage usually develops during the second week of symptoms. Diagnosis is confirmed by isolation of the organism from the local lesion. An active immunity may be produced by the prophylactic use of tetanus toxoid.

8. Tularaemia

This acute infectious disease is caused by the B. tularense which is transmitted to man usually from rabbits, ground squirrels, mice or other rodents. There is a characteristic primary cutaneous lesion with enlargement of the neighbouring lymph glands.

The incubation period averages three and a half days. The onset is sudden, accompanied by chills and fever. The primary lesion is usually found on the hands or fingers, being a shallow ulcer covered with a greyish membrane. It is indolent in healing. The lymph glands rapidly become enlarged and painful and widespread lymphangitis may occur. This has been called the ulceroglandular type and accounts for the majority of the cases. The sera of infected persons agglutinate the bacilli. The casual organisms may be isolated from the ulcer or glandular exudate.

The course of the disease is irregular, the fever usually lasting from three to four weeks. The immediate prognosis is good.

9. Undulant Fever

(Brucellosis, Malta Fever)

Undulant fever is an acute infectious disease characterized by a prolonged febrile course with irregular remissions and exacerbations. It is caused by a member of the Brucella group of micro-organisms.

The causative agent is ingested from contaminated food and drink, particularly milk, and butter and cheese made from the milk of infected goats and cows. It is also contracted by those who handle infected meat and meat products. The majority of cases occur in farmers, milkers and abattoir workers. The average incubation period is two weeks. The onset is usually gradual with a progressive increase in the evening temperature which returns to normal in the morning. In about ten days, elevation of the morning temperature occurs as well. Anorexia and constipation are the rule. Loss of weight is pronounced. Generalized muscle and joint pains are frequent complaints.

The course may last from a few weeks to many months and is characterized by recurrences of the fever, each bout usually lasting from two to three weeks. The organism may be isolated from the blood. The serum agglutinates the organisms. However, sera giving positive agglutination tests may be found in individuals who give no history of clinical symptoms.

10. Weil's Disease

(Acute Spirochaetal Hepatitis, Spirochaetal Jaundice)

This is a disease characterized by jaundice and intestinal haemorrhages. It is caused by the Leptospira icterohaemorrhagiae which is present in rats and is excreted in their urine. Transmission to man is probably through the skin, or by the gastro-intestinal tract.

The incubation period is about a week. The onset is usually sudden with chills, fever and prostration, followed in several days by jaundice. General muscular pains are usual and nausea with vomiting occurs. The temperature remains irregularly raised and falls by lysis, reaching normal about the eighteenth day. It may subsequently rise again for a few days. The organism may be isolated from the blood and urine during the first week of symptoms. Subsequently the diagnosis is made by serum agglutination tests. Relapses are fairly common. Fatal cases show severe liver destruction.

11. Septic Infections

Under this heading are included boils, carbuncles, folliculitis, cellulitis, localized lymphangitis, erysipelas and other septic infections commonly occurring in industry. In any occupation, the incidence of infection from these organisms depends to a large extent on the prevailing risk of injury to the skin.

Section J. Dusts and Fumes

Certain industrial processes are associated with the production of considerable quantities of dusts and fumes. The effect of such dusty environments upon the health of the worker is frequently very difficult to assess. At one time, most dusts were thought to be at least potentially harmful to the body. With the discovery of the effect of silica on the lungs, the view was finally adopted that only silica (and possibly asbestos) was dangerous, and dusts were classified as harmful or inert, depending upon their silica content. In recent years, with the discovery of lung conditions associated with non-siliceous dusts and fumes, and with the realization that the reaction of the lung to silica is probably modified by the inhalation of concommitant materials, it has become necessary to reconsider the effects on the body of exposure to dusty environments. Though silica still remains the chief cause of pulmonary disease resulting from occupational exposure, some other dusts and fumes are now known to produce disability or even death. It is doubtful whether any dust can be regarded as entirely harmless, under conditions of extreme exposure.

From the hygienist's point of view, the question is whether a particular dust or fume exposure may be harmful to the health of workers. The practicing physician must decide whether a worker's disability or impairment of function is the result of exposure to dust or fumes associated with his work, and whether an affected worker should be allowed to return to his job. To answer these questions requires not only a broad knowledge of the effects on the body of dusts and fumes generally, but also detailed information regarding the particular exposure and its relation to the worker. Thus, in assessing the exposure, information is required concerning such factors as: (1) chemical composition of the dust or fume; (2) the size of the dust or fume particles; (3) the concentration or quantity of the respirable dust or fume in the worker's breathing zone; (4) intermittency of exposure and total length of exposure; (5) severity of the work (as it affects depth of respiration); and (6) efficiency of protective devices and measures aimed at dust control.

1. *Chemical Composition of the Dust or Fume.*

This information is frequently difficult to secure from the patient. Dusts, which are produced by mechanical action, such as grinding, crushing, drilling and cutting, are, for the most part, similar in

composition to the substance from which they are derived. The same chemical constituents are present in the dust though sometimes in different proportions than in the parent substance. Fumes, on the other hand, being the result of condensation from the gaseous state, as after volatilization of metals, are frequently oxidized during the condensation process and therefore differ chemically from the substance from which they originate. In order to determine accurately the composition of the dust or fume, it may be necessary to secure samples for analysis.

2. *Size of Dust or Fume Particle.*

It has been conclusively established that the vast majority (over 90 per cent) of the particles which reach the alveoli are less than 3 microns (3/1,000ths of a millimetre) in diameter. Most particles larger than 3 microns either settle out of the air before it is breathed, or are trapped in the secretions of the nose and throat and eliminated or swallowed. As a rule, dust particles vary greatly in size, depending to some extent upon the mechanical processes involved in their production. The dust produced by the wet drilling of hard rock contains a higher *percentage* of respirable dust than that produced by dry drilling, since most of the larger particles are trapped in the wet process while many of the finer particles escape. The *number* of particles of respirable size produced by dry drilling is, of course, very much greater than that produced by wet drilling. In the case of fumes, the particles are usually more uniform in size and their diameter such that the greater proportion of the fume is respirable. However, fume particles tend to flocculate, producing larger particle masses.

3. *Concentration of Respirable Dust or Fume in Air.*

A healthy respiratory system is capable of handling a certain amount of dust or fume without impairment of function or systemic poisoning resulting. However, the inhalation of amounts beyond this threshold is harmful. The concentration of dust or fume which can safely be inhaled varies widely according to the composition of the material inhaled and the duration of the exposure. There is also a wide variation in individual susceptibility. Of two men, working at the same job and in the same exposure over a period of years, one may develop occupational dust disease while the other remains apparently unaffected. This individual susceptibility is often observed by the industrial physician dealing with groups of workers in similar exposures. For most of the mineral dusts and fumes,

values have been set for the concentrations of the material in air which are regarded as permissible for repeated 8-hour exposures.

4. *Intermittency of Exposure and Length of Exposure.*

In the case of certain acutely irritant dusts and fumes, (*e.g.*, cadmium), harmful effects may result from single, massive exposures. With the majority of dusts however, months or years may be required before the worker is affected. Frequently the man concerned has changed his job before symptoms develop. For this reason, a careful occupational history should start from the time the patient left school, and account for his employment during the subsequent years up to the time of his present illness.

The securing of adequate information concerning the above four factors in the worker's exposure, and the assessment of other factors such as the efficiency of the measures taken to control dust within the plant, frequently require laboratory and other facilities not readily available to the general practitioner. In such cases, assistance should be requested from the Provincial Department of Health or its Division of Industrial Hygiene.

1. Organic Dusts

Organic dusts contain carbon and are largely derived from substances of animal or plant origin. Common examples are dusts and fibres arising from the handling or manufacture of: wood, bone and shell; fur, skins, hides and leather; feathers; brooms and straw; flour and grain; tobacco; jute, flax (linen), hemp, cotton, wool (worsted, etc.); felts and carpets; rags and paper; sweepings.

Though most organic dusts do not cause pneumoconiosis or pulmonary fibrosis of a specific disabling nature, many are irritant to the upper respiratory passages and to the conjunctivae and skin, causing bronchitis, conjunctivitis and dermatitis. Many organic dusts may elicit allergic reactions in sensitized persons, with production of asthma, urticaria, etc. Such irritative and allergic effects are usually relieved by termination of the exposure, and tend to recur when the patient returns to the job. A few dusts of the organic type have been reported to cause rather specific illnesses, outlined below. In some cases, it is doubtful whether the dust itself is responsible, bacteria and fungi present in the dust having been incriminated by certain investigators.

COTTON DUST

The term "byssinosis" has been applied to a respiratory affection occurring in certain workers exposed to cotton dust. The condition has been recognized in England for some years. The persons affected were employed in carding rooms and were mainly the operators of carding engines. As yet, no definite cases have been reported in Canada.

The clinical picture usually takes the following course: After working for some time in the dusty atmosphere, the worker develops a feeling of irritation in the throat and chest, sneezing, and a dry, short cough. At first, complaints are noticed only on Monday mornings or after a short absence from work and subside after only a few hours or a day or two, even though the worker remains in exposure. Symptoms return each Monday morning and a low-grade fever may be present in a few of the affected workers. The condition may gradually wear off and not recur, or it may never entirely clear, the symptoms recurring upon each return to work after a short absence. In the early stages there is no disability. However, after 10 or more years' exposure, the symptoms of fever, cough and tightness in the chest return, in greater severity than in the early stage. The worker tires easily, loses weight, and suffers from asthma and bronchitis. Removal from work may produce some improvement in his condition, but return to work results in further recurrence of symptoms. As the condition progresses, dyspnoea becomes marked, the cough is more productive, with expectoration of mucoid or mucopurulent sputum, and there is marked emphysema. In the later stages, the condition is disabling and incurable. Physical findings in the early stages are not remarkable. In the later stages the usual clinical findings are those of chronic bronchitis and emphysema. The radiographic appearances are consistent with chronic bronchitis and emphysema though the hilar shadows are somewhat more prominent than is usually found in these conditions. Autopsies have revealed only a non-specific chronic bronchitis and emphysema. The condition is regarded by most authorities as the result of allergy occurring in sensitized persons.

In addition to the fairly distinct entity described above, there have been several reports of an acute febrile illness, with cough, dyspnoea, tightness in the chest, and general systemic symptoms, occurring in cotton weavers in England and in mattress makers, cotton seed treaters and cotton millworkers in the United States.

These illnesses were regarded as being a form of acute bronchitis. Residual symptoms lasted for one or two months. The American investigators believed the illness to be caused by a gram-negative, rod-shaped bacillus; the English workers attributed their cases to mycotic agents in the cotton dust.

"THRESHER'S LUNG" AND "FARMER'S LUNG"

Men exposed to dust from hay, straw and grain have been reported to suffer from a group of diseases which are often called "asthma", "bronchitis" or "pneumonia". Investigators in England, Switzerland and Scandinavia consider the condition to be a fairly discrete entity, probably due to mycotic infection of the lungs. Exposure occurs when grain has stood out in wet weather before threshing. Under these conditions the grain is frequently heavily contaminated with fungi, and when dried, the dust contains the spores. The condition is characterized by gradually increasing dyspnoea, slight fever, unproductive cough and an increased sedimentation rate. Clinical findings include areas of dullness to percussion and moist crepitations in the lungs. In more severe cases the shortness of breath is marked, the fever is higher, and there may be muco-purulent sputum or haemoptysis. Radiologically there is a fine reticulation in the early stages, later followed by a soft mottling through both lungs. As the disease progresses, the mottling becomes more dense, the hilar shadows enlarge, and eventually the lungs show large, dense patches due to coalescent areas of fibrosis, and there are considerable areas of emphysema. In the early stages, the condition responds to potassium iodide therapy. In the later stages response to therapy is poor, and the fibrosis advances. One case coming to autopsy showed emphysema of the lung, with bands of fibrosis traversing the lung substance.

WHEAT DUST

Large amounts of grain dust, when inhaled, may produce asthma and bronchitis in sensitive individuals. The fine hairs of the wheat grain have been reported to cause asthma in workers exposed to the dust arising from the first cleaning of the wheat. Injection of minute amounts of the wheat hair extract gave rise to severe local reactions and to asthma in sensitive millers.

BAGASSE

The term "bagassosis" is applied to a lung condition occurring in workers handling bagasse—a fibrous material which remains after the

sugar has been extracted from sugar cane. The condition has been reported in England, United States and Mexico, and consists of an acute bronchiolitis and pneumonia, with symptoms beginning usually about 8 weeks after first commencing work with the bagasse. There is usually fever, extreme dyspnoea, cough with scanty, black stringy sputum, and signs scattered throughout both lungs. Radiography shows the presence of miliary shadows throughout both lung fields. Symptoms gradually lessen over a period of 6 weeks, with the lung fields clearing completely. Occasionally the pneumonia fails to resolve and fibrosis of the lung may develop. The exact cause of the condition has not been definitely established, the vegetable dust and fungi both being suspect. No cases have been noted in Canada, as yet.

2. Inorganic Dusts and Fumes

These materials are metallic or mineral in origin. Included in the mineral dusts are coal and graphite, though strictly speaking the latter are organic materials. The inorganic dusts and fumes may be divided into five main groups, according to their action upon the body.

Group I. Inert Dusts and Fumes:

These substances produce no systemic symptoms when inhaled, and prolonged exposure does not result in the development of any disability. Though the lungs may show radiographic changes after some years of exposure, such changes are not accompanied by symptoms or measurable impairment of function, and there is no predisposition to pulmonary tuberculosis. The radiographic shadowing is caused, for the most part, by the deposition of radio-opaque materials in relation to the lymphatic system of the lung, where they remain, causing no injury. The lung changes produced by the inert dusts are of importance chiefly because of the difficulty they present in differential diagnosis, since their radiographic appearances sometimes closely resemble those due to silica. Included in the group of inert dusts and fumes are:

CARBON DUSTS

Graphite is a crystalline form of carbon; ash-free anthracite coal is an amorphous form of the same element. In the pure state, these dusts cause no fibrosis of the lung; as they occur in nature, however, graphite and anthracite frequently contain considerable amounts of

free silica, and workers exposed may develop a modified form of silicosis, or anthracosilicosis. Like true silicosis, anthracosilicosis may produce a disabling fibrosis of the lung. The incidence of the disease has been shown to be related to the concentration of silica dust in the air and to the length of the exposure.

IRON DUSTS AND FUMES

Exposure to these materials occurs in haematite miners, welders, polishers (who use the red oxide of iron, or rouge), tool makers, cutlery makers, and many other occupations. The deposition of these dusts or fumes in the lungs produces a non-disabling condition known as siderosis, which is characterized radiologically by a fine, generalized mottling of the lung fields. There are usually no symptoms or impairment of health. Certain exposures to iron dusts and fumes are associated with exposure to silica. Thus silicosis is a recognized hazard in iron and steel foundries, and the mining of iron ores in certain regions, particularly in England, is associated with the production of a modified form of silicosis which may be disabling if infected.

DUSTS OF CALCIUM AND MAGNESIUM COMPOUNDS

Calcium carbonate occurs naturally as limestone, marble and chalk. Gypsum is calcium sulphate. Calcined magnesium carbonate is used in making bricks for furnace linings and other types of insulating material. Inhalation of the dusts of these materials may produce radiographic changes, but there is no evidence that any disabling lung fibrosis results, even after prolonged exposures. Talc, a hydrated magnesium silicate, is discussed below (see Group V).

CEMENT DUST

Cement is essentially a mixture of calcium silicate and calcium aluminate, with an excess of lime. Though there may be up to 23 per cent of silica in cement, this silica is present as silicate, combined with the calcium. There is no definite evidence that prolonged exposure to cement dust causes any disability. Characteristic radiographic changes do not occur.

SILICATE DUSTS

Most of the silicate dusts including clay and kaolin (aluminum silicate), sodium silicate (or "water glass", an adhesive used in sealing cardboard cartons), and the dusts of fibrous glass insulating materials (composed of silicates of sodium, potassium, calcium and

other metals) produce no fibrosis of the lungs even after long exposure. The only silicate which at present is definitely known to cause disabling lung fibrosis is asbestos, a fibrous silicate of magnesium and other metals (see Group IV). In the case of two other silicates, mica and talc, their role in producing pulmonary disease is still under investigation (see Group V).

Recommended M.A.C.: For inert dusts, such as cement, limestone, chalk, iron, etc., containing no free silica, the recommended maximum allowable concentration is regarded by most authorities as being 50 M.P.P.C.F. (million particles per cubic foot of air).

Group II. Dusts and Fumes Producing Systemic Effects:

Some metallic dusts and fumes are absorbed through the lungs, causing systemic effects in other parts of the body while the lungs escape damage. Inhalation of lead dust and fumes plays an important part in the causation of lead poisoning, and mercury poisoning may follow the inhalation of mercury vapour. The chronic form of manganese poisoning usually follows inhalation. While the lungs are generally not affected in such cases, mice exposed to manganese dust have developed a pneumonitis, and British and European reports have indicated a higher incidence of pneumonia and other respiratory infections in persons exposed to manganese dusts and fumes than in the general population. The inhalation of fumes of certain other metals causes a general systemic effect known as "metal fume fever". While zinc oxide is regarded as the commonest cause, the oxides of magnesium, copper, lead, antimony, arsenic, nickel, silver and cobalt have been reported as capable of producing the condition. Some investigators think that the fever is a reaction to proteids liberated following the destruction of epithelial cells of the respiratory tract by the action of the metallic oxide. The only respiratory symptoms reported are a dry cough and tightness of the chest. In any case, the effects on the respiratory system cause little discomfort, and no permanent disability is produced. See Section K, for further details regarding systemic poisons and metal fume fever.

Group III. Dusts and Fumes Producing Inflammatory Lung Changes:

Inhalation of dusts or fumes of cadmium, vanadium, osmium and beryllium may produce acute inflammatory changes in the lungs. Exposure to certain compounds of beryllium may result in the development of an acute pneumonitis, or the worker may apparently escape injury and have no symptoms while in exposure, only to develop a

disabling and sometimes fatal lung condition after leaving exposure. The latent period may be as long as five years. Cadmium, beryllium and vanadium are discussed in Section K, since their effect upon the body is not restricted to the respiratory system.

Group IV. Dusts and Fumes Producing Disabling Lung Fibrosis:

Two dusts and one fume are known to produce disabling pulmonary fibrosis—silica, asbestos, and the fume produced during the fusion of bauxite in the manufacture of artificial alumina abrasives.

SILICA

From the point of view of numbers of men exposed and cases of disability produced, silica is still the chief cause of pulmonary dust disease. The distinction between "free" silica (silicon dioxide, SiO_2) and "combined" silica (or silicate, having the radicle $-SiO_4$) is most important, since the silicates, with the exception of asbestos, are probably inert. The prolonged inhalation of dusts containing free silica, on the other hand, may result in the development of a disabling pulmonary fibrosis known as silicosis. The Committee on Pneumoconiosis of the American Public Health Association defines silicosis as "a disease due to the breathing of air containing silica (SiO_2), characterized by generalized fibrotic changes and the development of miliary nodules in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present), and characteristic X-ray findings."

Silica occurs in the pure state in nature as quartz. It is the main constituent of sand, sandstone, tripoli and diatomaceous earth, and is present in high amounts (up to 35%) in granite. Several ores, especially iron and coal, may contain a variable amount of silica. Exposure to silica occurs in hard rock mining, in foundries, in the manufacture of porcelain and pottery, in the spraying of vitreous enamels, in sandblasting, in granite-cutting and tombstone-making, in the manufacture of silica firebrick and other refractories, in grinding and polishing operations where natural abrasive wheels are used, and other occupations.

The duration of exposure which is associated with the development of silicosis varies widely for different occupations. Thus, the average duration of exposure required for the development of silicosis in sand-blasters is 2 to 10 years, in moulders and granite cutters

about 30 years, and in hard rock miners 10 to 15 years. There is, also, much variation in individual susceptibility, certain workers showing radiological evidence of the disease years before their fellow workmen who are similarly exposed. Such susceptible individuals are fortunately rather rare.

In the absence of pulmonary tuberculosis, silicosis does not commonly cause symptoms or disability. The action of silica on the lungs results in the production of a diffuse, nodular fibrosis in which the parenchyma and the lymphatic system are involved. This fibrosis is, to a certain extent, progressive, and may continue to increase for several years after exposure is terminated. It would appear that the fibrosis is commonly self-limiting, a point of equilibrium being reached where the reparative processes of the lungs effectively counteract the irritant action of the retained and dissolved silica. The exact point of equilibrium is determined chiefly by the amount of silica retained in the lungs, and by the effectiveness of the response elicited in the individual's lung tissue. In most instances, the pulmonary reserve greatly exceeds the amount of lung tissue which is replaced by the fibrotic process, and no symptoms or disability result. Where the pulmonary reserve is sufficiently reduced, the worker complains of shortness of breath on exertion. This is the first and most common symptom in cases of uncomplicated silicosis. If severe, it may incapacitate the worker for heavy, or even light, physical exertion, and in extreme cases there may be shortness of breath even while at rest. The most common physical sign of silicosis is a limitation of expansion of the chest. There may be a dry cough, sometimes very troublesome. The characteristic radiographic appearance is one of diffuse, discrete nodulation, scattered throughout both lung fields. Where the disease advances, the shortness of breath becomes worse, and the cough more productive and troublesome. There is no fever or other evidence of systemic reaction. Further progress of the disease results in marked fatigue, extreme dysnopea and cyanosis, loss of appetite, pleuritic pain and total incapacity to work. If tuberculosis does not supervene, the condition may eventually cause death either from cardiac failure or from destruction of lung tissue, with resultant anoxaemia. In the later stages, the X-ray may show large, conglomerate shadows, due to the coalescence of the silicotic nodules, with areas of emphysema between them. Such advanced cases are rare, and are usually the

result of prolonged massive exposures occurring in individuals who are unduly susceptible.

The most serious feature of silicosis lies in the fact that its presence predisposes to tuberculous infection, and that tuberculosis in persons with silicosis tends to progress regardless of any therapeutic measures taken, although rest may delay the process. In some silicotics the tuberculosis runs an acute course; more commonly, the combination of tubercle bacilli and inhaled quartz produces chronic fibrosis of the lungs, which slowly spreads over a period of many years. Contraction of the masses of fibrosis results in the formation of areas of compensatory emphysema. Such disease causes shortness of breath, and if severe, may produce total disability. The usual symptoms of tuberculosis, such as fever, night sweats, loss of weight and productive cough may make their appearance only a year or so before death. The dyspnoea and disability which were formerly believed to be characteristic of silicosis were in most instances due to the massive fibrosis and emphysema which followed the combined action of tubercle bacilli and silica. Silicosis, alone, rarely causes significant emphysema, with its resultant shortness of breath and disability.

In making a diagnosis of silicosis, it is essential to ascertain whether the man worked in a silica exposure, and if he has, whether he had sufficient exposure to cause the disease. Though this seems elementary, cases have been labelled silicosis where there was never any exposure to silica, or where there had been exposure for only several months. The tendency to rely solely upon a single chest film, with no enquiry made as to past exposure, frequently results in such mistakes in diagnosis. Much harm can be done a man in telling him that his disability is due to silicosis, if it should be shown subsequently that he does not have the disease.

Recommended M.A.C.: For dusts containing over 50% free silica, the recommended maximum allowable concentration for prolonged exposure is 5 million particles per cubic foot (M.P.P.C.F.)

For dusts containing 5 to 50% free silica, the recommended M.A.C. is 20 M.P.P.C.F.

For dusts containing less than 5% free silica, the recommended M.A.C. is 50 M.P.P.C.F.

ASBESTOS

Canada is one of the principal countries producing asbestos. This material is chiefly hydrated magnesium silicate, occurring as a white

fibrous ore. The fibres, when separated, may be spun into yarn and woven in much the same manner as ordinary textiles or they may be ground and mixed with other materials to form insulating boards or sheets. Exposure to the dust occurs in the crushing, carding, spinning and weaving of the material, and in the manufacture of brake linings and asbestos insulating products.

The essential lesion produced is a diffuse fibrosis which probably begins as a "collar" about the terminal bronchioles. Usually, at least 4 to 7 years of exposure are required before the production of a serious degree of fibrosis. There is apparently less predisposition to tuberculosis than is the case with silicosis.

Clinically, the most striking sign is shortness of breath of gradually increasin intensity, often associated with a dry cough. In the early stages physical signs are absent or slight; in the later stages râles may be heard, the diastolic blood pressure is raised, the second pulmonary sound is accentuated, and in long-standing cases there is frequently clubbing of the fingers. In the early stages of the disease the chest X-ray reveals a ground-glass or granular change, chiefly in the lower lung fields; as the condition progresses the heart outline becomes "shaggy" and irregular patches of mottled shadowing may be seen.

At autopsy, the pleurae are thickened and adherent and thick subpleural fibrous plaques are often present. Where the disease is far advanced there are usually large areas of fibrosis, with emphysematous changes in the apices and bases. The alveolar walls are thickened, and the characteristic "asbestos bodies" are found.

Recommended M.A.C.: 5 to 10 M.P.P.C.F.

FUME PRODUCED DURING THE FUSION OF BAUXITE

In 1942 an occupational lung disease caused by the fumes produced during the fusion of bauxite in the manufacture of artificial alumina abrasives was discovered by Dr. C. G. Shaver, Superintendent of the Niagara Peninsula Sanatorium. Bauxite is a natural hydrated alumina or aluminum oxide (Al_2O_3) containing small amounts of silica. When fused, a crystalline alumina or artificial corundum is formed. In the manufacture of corundum the bauxite is heated in open metal pots by means of an electric arc. During the fusion, dense white fumes are given off. The fumes consist chiefly of alumina (40 to 60%) and amorphous silica (30 to 45%). The greatest exposure occurs among men operating overhead cranes and among those who shovel mix into the furnaces.

The lesion produced is essentially a diffuse interstitial fibrosis. The nodule formation, which characterizes silicosis, is absent. In the early stages there are no symptoms or clinical signs. The radiographic changes consist of an indefinite granular or lace-like appearance in the apices and sub-apical regions. As the condition progresses, the lace-like shadowing extends into the lower lung filelds In the later stages the whole lung field may present a granular, or irregular, nodular appearance. The diaphragm is distorted and the mediastinum widened. Up to this point, the patient has usually had no complaints, and the only physical sign is a decreasing vital capacity. In such cases, spontaneous pneumothorax, frequently bilateral, may occur, with complaint of sudden extreme dyspnoea and/or severe chest pain. With the occurrence of the pneumothorax the man is nearly always totally incapacitated. The heart is retracted to the uncollapsed side, and the periphery of the collapsed lung may show large emphysematous blebs or bullae. The lungs of cases coming to autopsy have shown massive strands of interstitial hyaline fibrosis, with areas of compensatory emphysema. The pleurae are thickened or adherent, and sub-pleural bullae are usually present. There is no nodule formation. Extensive lung fibrosis may follow as little as 2 or 3 years of exposure, though usually the period of exposure is longer. The exact factor in the fumes which is responsible for causing the condition has not yet been isolated. The possibility that the fibrosis may be due to silica, modified in its action by the presence of alumina or of other materials present in the fumes, has to be considered. On the other hand, exposure of workmen in Germany during the past war to aluminum powder resulted in the development of a lung condition which, from the reports published, appears to be essentially the same as that discovered by Shaver. There is some suggestion that similar cases have also occurred in Sweden following exposure to aluminum powder. These exposures are being investigated.

Recommended M.A.C.: No values have been set, as yet. Periodic chest X-ray is essential to the early detection of cases.

Group V. Dusts and Fumes of Undetermined Pathogenicity to the Lung:

The effect of certain dusts and fumes on the body and the role they play in causing lung disease has not yet been clearly determined.

TALC

Talc or soapstone, a hydrated magnesium silicate, is used as a filler in rubber, plastics, paints, insulating materials, cosmetics and dusting powders. Exposure may occur in the mixing, crushing and milling of the material, and in its use as a filler. Reports published indicate that exposure to talc dust may be associated with the development of a diffuse fibrosis, with symptoms of dyspnoea, cough and fatigue, and with the production of disability. Radiologically there may be a diffuse, fine granular shadowing throughout both lung fields, or the shadowing may be of a nodular type, resembling silicosis. Autopsy reports describe irregular areas and bands of cellular fibrosis surrounding the vessels and bronchi, producing thickening of the alveolar walls. No silicotic nodules were found. Though some investigators believe that talc, per se, is responsible, the possibility that the condition is produced by a modified action of free silica present in the talc has to be borne in mind. Further investigation is needed to decide the question.

Recommended M.A.C.: It is felt by some authorities that the concentration of talc dust in air should not exceed 20 M.P.P.C.F. where men are exposed repeatedly for 8 or more hours each day.

MICA

A potassium-aluminum or magnesium-aluminum silicate. There is no definite evidence that exposure to mica dust causes pulmonary fibrosis. The possibility of free silica being present in the earth from which mica is obtained must be remembered.

Recommended M.A.C.: 50 M.P.P.C.F., where the mica contains less than 5% silica.

DIATOMACEOUS EARTH

Natural diatomaceous earth is an amorphous form of silica. In the amorphous state, the material does not cause disabling lung fibrosis. When calcined with certain fluxes, a portion of the amorphous silica is converted into crystalline silica, and men exposed to this form of the earth have been reported to develop a lung fibrosis which does not exhibit the nodule formation characteristic of silicosis. Pneumothorax has been reported to occur frequently in the men affected.

Recommended M.A.C.: No limit has been set.

ARSENIC, CHROMIUM AND RADIOACTIVE DUSTS

There is some evidence that exposure to dusts of arsenic, chromium and radioactive materials has been associated with a higher incidence

of pulmonary cancer than occurs in the general population. In the case of arsenic and the radioactive dusts, the evidence that these materials may cause carcinoma of the lung is fairly strong. However, in the case of an individual with lung cancer, it is as yet impossible to say that the carcinoma is the result of his exposure, even where there has been prolonged exposure to one or more of these materials. The role played by exposure to dusts and fumes in the development of pulmonary carcinoma is extremely difficult to assess and it may be many years before the relationship is clear.

Section K. Harmful Chemicals

Acetaldehyde (Acetic Aldehyde; Acetyl Hydride; Aldehyde; Ethanal; Ethyl Aldehyde).

Formula: CH_3CHO.

Properties: A colourless liquid, having a strong, pungent, fruity odour. Inflammable.
Specific Gravity: 0·78 times as heavy as water.
Boiling Point: 69·4° F.
Vapour Density: 1·5 times heavier than air.
Volatility: Is quite volatile at ordinary room temperatures. Acetaldehyde is miscible with water, alcohol, benzol, ether, acetone and gasoline.

Uses and Occurrence: Used in the manufacture of chemicals and dyes, plastics, synthetic rubber, disinfectants and preservatives, drugs and perfumes. Is employed in photography and in the silvering of mirrors.

Mode of Entry into Body: As a vapour, through inhalation.

Physiological Action and Toxicity: Acetaldehyde is eliminated in the bile by the liver. The vapour is irritant to the mucous membranes of the eyes, mouth and respiratory tract. The majority of subjects find 50 ppm irritating to the eyes, and a concentration of less than 200 ppm is definitely irritating to the nose and throat. Acetaldehyde possesses anaesthetic properties, but this action is masked by the more intense irritant action on the upper respiratory tract. Continued exposure to very high concentrations may result in death from respiratory paralysis. Acetaldehyde is less toxic than

formaldehyde and decidedly less so than acrolein. In an occasional hypersensitive individual, acetaldehyde has been known to act as a specific irritant, producing urticaria.

Signs and Symptoms: Exposure to the vapour may cause cough, dyspnoea, rhinitis, bronchitis, tachycardia, conjunctivitis, and night sweats. In hypersensitive individuals, exposure to the liquid or vapour may result in urticaria, asthmatic attacks, etc.

Recommended M.A.C.: The intense irritating effect of the vapour gives warning long before a dangerous concentration is reached. The maximum allowable concentration recommended by various authorities ranges from 50 ppm to 200 ppm; the latter figure is considered by most to be quite safe for an 8-hour exposure.

Fire Hazard: Dangerous: is a class 1 flammable liquid. The minimum explosive limit is 4%, the maximum 57%, by volume, of the vapour in air. Flash point: −17°F.

Acetone (Dimethylketal; Dimethyl Ketone; Methyl-Acetyl; Propanone)

Formula: CH_3COCH_3.

Properties: A colourless liquid having a fragrant odour. Inflammable. Specific Gravity: 0·792 times as heavy as water. Boiling Point: 134°F. Vapour Density: 2 times heavier than air. Very volatile. Miscible in all proportions with water, alcohol and ether.

Uses and Occurrence: Is used widely in industry as a solvent.

Mode of Entry into Body: Through inhalation of the vapour. Small amounts can be absorbed through the skin.

Physiological Action and Toxicity: Acetone is narcotic to animals in very high concentrations. Cats are anaesthetized by concentrations of 8,000 to 17,000 ppm and above, but repeated exposures to concentrations of 2,000 ppm cause no ill effects other than slight irritation of the eyes and nose. In industry, no injurious effects from its use have been reported, other than the occurrence of skin irritations resulting from its de-fatting action.

Signs and Symptoms: There are rarely any complaints. Occasionally there may be drying and cracking of the skin.

Recommended M.A.C.: 500 ppm for an 8-hour working day.

Fire Hazard: Dangerous. Flash point: 0°F. Minimum explosive limit is 2%, maximum explosive limit 13%, by volume, of the vapour in air. Is a class 1 flammable liquid.

Acetylene (Ethine)

Formula: $CH \equiv CH$.

Properties: A colourless gas. Highly inflammable.
Vapour Density: 0·9 times as heavy as air.
Soluble in water, acetone and alcohol.
May contain the following impurities, all of which are toxic: phosphine, arsine, hydrogen sulphide, carbon disulphide and carbon monoxide.

Uses and Occurrence: Is widely used as a fuel in the welding and cutting of metals. Is also employed in organic synthesis and the manufacture of chemicals.

Mode of Entry into Body: Through inhalation of the gas.

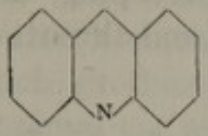
Physiological Action and Toxicity: When mixed with oxygen, in proportions of 40 per cent or more acetylene, acetylene acts as a narcotic, and has been employed in surgical anaesthesia. Acetylene acts as a simple asphyxiant, by diluting the oxygen in the air to a level which will not support life. However, the presence of impurities in commercial acetylene may result in the production of symptoms before an asphyxiant concentration is reached.

Signs and Symptoms: Dizziness, headache, mild gastric symptoms, and in high concentrations, semi-asphyxia and brief loss of consciousness have all been reported. In general industrial practice however, acetylene does not constitute a serious hazard.

Recommended M.A.C.: None has been suggested, since asphyxiant concentrations are seldom reached in industrial practice.

Fire Hazard: Acetylene is an explosive gas when mixed with air in concentrations above 2·5 per cent and below 80 per cent, by volume, of the vapour in the air.

Acridine

Formula: $C_{13}H_9N$ or 

Properties: Small, colourless, needle-like crystals.
Sublimes at 212°F. Melting point: 232°F.
Soluble in alcohol, ether and carbon disulphide. Slightly soluble in hot water.

Uses and Occurrence: Used in the manufacture of dyes.

Mode of Entry into Body: Through inhalation of the vapour or dust. Acts locally on the skin.

Physiological Action and Toxicity: Inhalation of the dust or vapour causes violent sneezing. Acridine is irritating to the eyes and the mucous membranes of the upper respiratory tract. Solutions of acridine and its salts irritate the skin.

Recommended M.A.C.: No value has been set.

Acrolein (Acrylic Aldehyde; Acraldehyde; Allyl Aldehyde; Propenal)

Formula: $CH_2=CHCHO$.

Properties: A colourless or yellowish liquid, with a disagreeable, choking odour. Inflammable.
Specific Gravity: 0·84 times as heavy as water.
Boiling Point: 126°F.
Vapour Density: 1·94 times heavier than air.
Soluble in water, alcohol and ether.

Uses and Occurrence: Used in the organic synthesis of plastics, in the manufacture of rubber, and in the refining of lubricating oils. Industrial exposure commonly occurs through the heating to dryness, and decomposition, of oils, fats and glycerine.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Acrolein is an intense irritant to the eyes and mucous membranes of the upper respiratory tract. It possesses narcotic action, but the irritant effect is so strong that one is forced to leave the exposure before narcosis has time to occur. A concentration of 1 ppm produces marked irritation of the eyes and nose in 5 minutes. 10 ppm is said to be fatal in a short time. Prolonged exposure to high concentrations may induce oedema of the lungs.

Signs and Symptoms: Weeping and burning of the eyes, irritation of the nose and throat, cough, shortness of breath, dizziness.

Recommended M.A.C.: 0·5 ppm for an 8-hour work period.

Fire Hazard: Liquid acrolein is classed as a dangerous fire hazard. No data are given as to flash point or explosive limits. However, since 1 ppm of the vapour in air is easily detectable, producing marked irritation in 5 minutes, there is little chance of explosive concentrations being reached without detection.

Acrylonitrile (Vinyl Cyanide)

Formula: $CH_2=CHCN$.

Properties: Colourless liquid, with a mild ether-like odour. Inflammable. Boils at 171°F, and has a vapour density of almost twice that of air. Is about 7% soluble in water, is completely soluble in all common organic solvents.

Uses: In the manufacture of synthetic (Buna S, oil-resistant) rubber and plastics; in organic synthesis.

Mode of Entry into Body: As vapour, through inhalation; as the liquid, through skin absorption.

Physiological Action and Toxicity: Acrylonitrile closely resembles hydrocyanic acid in its toxic action. By inhibiting the respiratory enzymes of tissues, it renders the tissue cells incapable of oxygen absorption. Poisoning is acute; there is little evidence of cumulative action on repeated exposure.

Signs and Symptoms: Exposure to low concentrations is followed by flushing of the face and increased salivation; further exposure results in irritation of the eyes, photophobia, irritation of the nose, deepened respiration, and if exposure continues, respirations become shallow. Nausea, vomiting, weakness and oppressive feeling in the chest, occasionally headache and diarrhoea, are other complaints. Several cases of mild jaundice have been reported, accompanied by mild anaemia and leucocytosis. Urinalysis is generally negative, except for an increase in bile pigment. Serum and bile thiocyanates are raised.

Recommended M.A.C.: The maximum allowable concentration recommended for an 8-hour day is 20 ppm. This is half the concentration which produces a toxic effect on dogs, the most susceptible animal yet studied.

Fire Hazard: Dangerous; flash point: 39°F. Minimum explosive limit: 3·05%. Maximum explosive limit: 17%. Class 2 flammable liquid.

Alcohols

Alcohols are widely used in industry. All are narcotic, and in addition, possess a direct poisoning action. Theoretically, their toxicity increases as the number of carbon atoms in the chain is increased. However, as one ascends the series, the boiling point rises, solubility decreases, and volatility decreases. Because of their low volatility, those alcohols above ethyl in the series, (that is, the propyl, butyl, and amyl alcohols) are of relatively little importance as a cause of illness in industry, even though their use is fairly widespread. Alcohols higher in the series than amyl alcohol are much less widely used, and are even less volatile. For further details, refer to: methyl alcohol, ethyl alcohol, propyl alcohol, butyl alcohol and amyl alcohol.

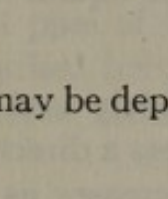
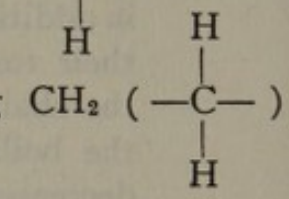
Aldehydes

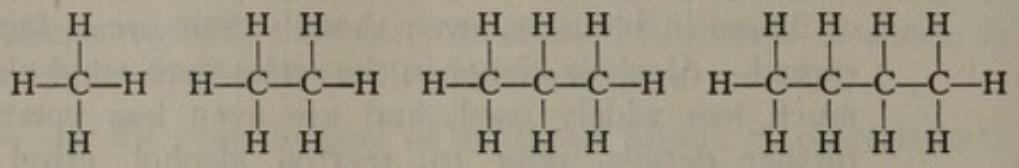
The aldehydes are oxidation products of the primary alcohols. Formaldehyde, acetaldehyde, croton aldehyde, paraldehyde and acrolein, or allyl aldehyde, are all used or occur in industry, particularly in the manufacture of rubber. In addition to these, there are other aldehydes which are also employed in industry, though less widely. Formaldehyde and acrolein present the most important hazards to health. These two aldehydes, together with acetaldehyde and paraldehyde, are discussed in more detail elsewhere in this section.

All the aldehydes possess anaesthetic properties, but this is masked by their marked irritant action on the eyes and mucous membranes of the respiratory tract. The lower aldehydes are very soluble in water, and act chiefly on the eyes and tissues of the upper respiratory tract. The higher aldehydes, being less soluble in water, tend to penetrate more deeply into the respiratory system and may affect the lungs.

Aliphatic Hydrocarbons

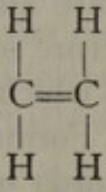
Hydrocarbons are chemicals containing carbon and hydrogen atoms. Each carbon atom has four valencies or bonds. Each bond is linked to another atom, which may be another carbon or hydrogen atom. Where each carbon atom is linked to four other atoms, the chemical is called a saturated hydrocarbon.

Thus Methane (CH_4) may be depicted as  , and is the simplest hydrocarbon. By adding CH_2  groups to methane, a series of saturated hydrocarbons may be built up, as follows:



Methane, CH_4 Ethane, C_2H_6 Propane, C_3H_8 Butane, C_4H_{10}

Where two carbon atoms share more than one bond, the hydrocarbon is said to be unsaturated. Thus:

Ethylene is C_2H_4, or $CH_2{=}CH_2$, and may be written as  . Acetylene is C_2H_2, or $CH{\equiv}CH$.

Hydrocarbons in which the carbon atoms occur in chains, either straight as in butane, or branched, are spoken of as aliphatic hydrocarbons. Aliphatic hydrocarbons may be classified according to the degree of saturation of the carbon bonds. Where all the carbon bonds are saturated, the hydrocarbon is known as a paraffin or alkane. Where two carbon bonds are shared by adjacent carbon atoms, the hydrocarbon is called an olefine or alkene; where three bonds are shared by adjacent carbon atoms, the hydrocarbon is an alkyne, or member of the acetylene series.

The Paraffin Series: The lower members of the paraffin series of aliphatic hydrocarbons, in ascending order, are methane, ethane, propane, butane, pentane, hexane, heptane, octane, nonane, and decane. The series may be continued, but the above list contains the more important members. The first four members of the series are gases at ordinary room temperatures. Pentane and the higher members, up to $C_{15}H_{32}$, are all liquids; members higher than these are solids. In general, the higher the member of the series, the higher the boiling point and the lower the volatility.

Methane occurs as the fire-damp of coal mines. Ethane and propane are used as fuels, and propane and butane as refrigerants. The higher homologues occur as distillation products of petroleum oil, and mixtures of them are commonly known as petroleum ether, naphtha, benzine, gasoline, kerosene, etc. (See Petroleum Hydrocarbons).

The Olefine Series: The lower members of the olefine series are: ethylene (ethene), propylene (propene), butylene (butene), amylene (pentene), hexylene (hexene), etc. Ethylene is used in welding; the other members are not widely used in industry. Amylene and hexylene are liquids, the lower members are gases.

The Acetylene Series: The chief member of this series is the first, acetylene. It is widely used in welding.

Physiological Action and Toxicity. Methane and Ethane are simple asphyxiants. Propane and butane are narcotic in high concentrations, but there is no evidence that toxic effects result from exposures lasting several hours to concentrations of 3% to 5%. The higher members of the paraffin series are anaesthetic and irritant. Further details of their action are given under "Petroleum Hydrocarbons." The members of the series higher than decane are not sufficiently volatile to form physiologically active concentrations in air.

Ethylene and propylene, in high concentrations, are anaesthetics; however, to have a narcotic effect, very high concentrations, of 60% or more, are required. In industrial practice, where they are diluted with air, they act as simple asphyxiants, producing symptoms only when the oxygen in the air has been diluted to a level where anoxia results.

Details concerning the toxicity of acetylene are given elsewhere in this section.

Recommended M.A.C.'s: No maximum allowable concentrations have been recommended for methane, ethane, propane, butane, ethylene or propylene, since their toxicity is so low that they seldom cause industrial poisoning.

Fire Hazard: Methane, ethane, propane, butane, ethylene, propylene, butylene, and acetylene are all explosive gases, having minimum explosive limits of around 2% and maximum explosive limits of 9 to 11%, by volume, of the gas in air. The minimum explosive limit of amylene is 1·6%.

Alkalis

The most important of the alkaline materials used in industry are: sodium hydroxide (caustic soda), anhydrous sodium carbonate (soda ash), sodium sulphide, trisodium phosphate, potassium hydroxide (caustic potash), potassium carbonate, ammonium hydroxide and carbonate, calcium oxide (quicklime), calcium sulphide and cyanamide, barium oxide, hydroxide and carbonate, arsenic sulphide and the alums.

Mode of Entry into Body: Most of the alkalis act locally on the skin; gaseous ammonia may be inhaled.

Physiological Action and Toxicity: The alkalis, as a group, constitute one of the commonest causes of occupational dermatitis. They act on the skin as primary irritants. Alkaline solutions soften and dissolve the keratin layer, and the skin becomes white, soggy, wrinkled and macerated. Repeated exposure frequently results in the development of chronic eczematous skin conditions. The stronger caustics may produce chemical burns which are often deep and slow in healing.

Systemically, the only alkali presenting any hazard is ammonia, which is discussed separately.

Aluminum

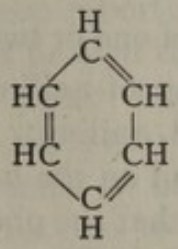
Symbol: Al.

Aluminum is found very widely in nature, always in combination with other elements. The commonest minerals containing it are the felspars, the micas, kaolon (clay), cryolite ($3NaF, AlF_3$), and bauxite ($Al_2O_3, 2\ H_2O$).

Aluminum, in the form of the silvery, light metal, is not generally regarded as an industrial poison. Following animal experiments, the inhalation of fine dusts of aluminum and of aluminum hydroxide ($Al(OH)_3$) was introduced in the prevention and treatment of silicosis. To date there has been no clear-cut evidence that the inhalation of these materials, in uncomplicated cases of silicosis, has resulted in any damage to the body under present conditions of administration.

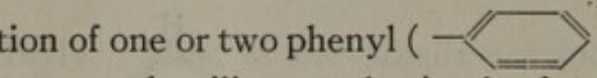
On the other hand, a form of acute lung fibrosis associated with the manufacture of aluminum powder was discovered in Germany during the war. In 1942 a similar lung disease was discovered independently in Ontario, in connection with the fusing of bauxite in the manufacture of aluminum oxide abrasive materials. This lung condition is discussed in Section J, under Inorganic Dusts, Group IV.

Amino Compounds of Benzol, Toluol and Xylol

Benzol, the simplest of the aromatic hydrocarbons, has the structural formula  . When one of the hydrogen atoms is replaced by a methyl ($—CH_3$) group, Toluol, (methyl benzol or phenyl methane) is formed. When two hydrogen atoms of the benzol ring are replaced with methyl groups, Xylol (dimethyl benzol) results. These three substances are starting points in the manufacture of many organic chemicals; their action on the body is discussed elsewhere in this section.

When one of the hydrogen atoms in the benzol ring is replaced by an amino ($—NH_2$) group, Aniline, or amino benzol, is formed (See "Aniline"). Further derivatives may be formed from aniline by the introduction of various groups into the benzol ring, or by the replacement of the hydrogen atoms in the amino radicle. Thus, the replacement by the methyl ($–CH_3$) radicle of one or two hydrogen

atoms in the ring portion of the aniline molecule results in the formation of methyl aniline and dimethyl aniline. Replacement by two ethyl ($—CH_2CH_3$) groups gives us diethyl aniline. In general, aniline derivatives of this type have much the same action on the body as has aniline, producing a methaemoglobinaemia and a depressant effect on the nervous system. If one of the hydrogens in the amino group of aniline is replaced by the acetyl radicle ($—OC—CH_3$), acetanilide is formed. This substance was formerly used as an analgesic and antipyretic drug; its use in industry is chiefly confined to organic synthesis and the manufacture of celluloid. Industrially, acetanilide has caused little trouble, but its abuse as a drug in certain countries resulted in cases of human poisoning and death, its chief effects being the production of methaemoglobinaemia and an acute antipyretic action. The symptoms in acute poisoning by ingestion were: cyanosis, fatigue, dizziness and palpitation, with sometimes nausea and vomiting, gastric pain, twitchings, visual disturbances, rigor and delirium. Occasionally urticarial rashes and eczema were produced.

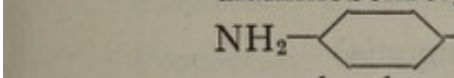
The introduction of one or two phenyl () radicles into the amino group of aniline results in the formation of mono- and diphenyl aniline. These substances appear to have a different action on the body than do aniline and most other derivatives, in that the phenyl anilines do not produce a methaemoglobinaemia. They may produce depression of the central nervous system.

Substitution of one or more chlorine atoms for hydrogen atoms in the aniline ring yields chloroanilines. In general, the clinical picture of chloroaniline poisoning is very similar to that of aniline poisoning, and the main portal of entry of the poison is absorption through the skin.

The hydrogen atoms of the aniline ring may be replaced by nitro ($—NO_2$) groups, forming the various nitro anilines. The three mononitroaniline isomers appear to be much more toxic than tetranitroaniline. The latter may cause dermatitis. All the nitroanilines are absorbed through the skin and by breathing the dust. The mononitroanilines differ slightly from aniline in their action upon the body,

affecting the nervous system less and the circulation more than is the case with aniline. Following absorption, the mononitroanilines produce methaemoglobinaemia, cyanosis, anaemia, weakness, headache and cardiac disturbances. If the poisoning is severe, the patient may become dyspnoeic and lapse into unconsciousness and ultimately die. Persons who recover from severe exposure may have kidney and liver damage.

Another nitroaniline derivative is Tetryl, which is discussed separately.

Introduction of two amino groups into the benzol ring yields diaminobenzol, better known as phenylene diamine; *e.g.*  . There are three isomers, all occurring as colourless crystals at ordinary room temperatures. They are used chiefly in the manufacture of dyes, and in the dyeing and rubber industries. The para-phenylene diamine appears to be the most toxic of the three isomers, and causes dermatitis in workers using it or handling furs treated with para-phenylene diamine dyes. Para-phenylene diamine has also been reported as causing attacks of asthma. Some investigators believe that it acts locally as a primary irritant. On the other hand, animal experiments have shown that para-phenylene diamine is a true sensitizing agent, and most cases of dermatitis and asthma which it causes appear to be due to this effect. Moderate gastro-intestinal upset has been occasionally reported following para-phenylene diamine poisoning.

The most important of the amino derivatives of toluene are the mono-amino toluenes or toluidines. The ortho and meta isomers are liquids, the para-toluidine is a solid. All are absorbed through the skin and from the gastro-intestinal tract, their action on the body being essentially the same as that of aniline, with depression of the nervous system and formation of methaemoglobin. The para-toluidine is apparently the most toxic of the three, and may cause destruction of the red blood cells, anaemia, haematuria, jaundice and irritation of the mucous membranes and skin, in addition to the effects on the nervous system and blood already mentioned.

Chlorotoluidine is formed by introducing a chlorine atom into the toluidine molecule. All four isomers are readily absorbed through the skin, the 5-chloro-2-toluidine probably being the most toxic. In addition to causing depression of the nervous system and formation of methaemoglobin, it may cause icterus, degenerative changes in the liver, and irritation of the bladder, with supra-pubic pain, haematuria and cells and casts in the urine.

When an amino group is introduced into the xylene molecule, amino xylene, or xylidine (dimethyl amino benzol) is formed. The six xylidine isomers resemble aniline in their effect upon the body.

Ammonia (Anhydrous Ammonia, Liquid Ammonia)

Formula: NH_3

Properties: A colourless gas having a characteristic pungent odour. Vapour Density: 0·597. It is easily liquefied by compression (liquid ammonia) and is extremely soluble in water, forming ammonium hydroxide (aqua ammonium, water of ammonia, ammonium hydrate), NH_4OH.

Uses and Occurrence: Occurs and is used widely in industry.

Mode of Entry into Body: Through inhalation of the gas.

Physiological Action and Toxicity: Ammonia is a strong irritant to the eyes and upper respiratory tract. This effect is so marked that serious exposure seldom occurs where the workman is able to leave the exposure. The irritant action of the gas on the eyes may temporarily render the employee incapable of making his escape. A concentration of 50 ppm produces a detectable odour, and concentrations of 500 to 700 ppm cause irritation to the eyes, nose and throat. Concentrations over 2,500 ppm may be dangerous if exposure continues beyond ½ hour. Death results from spasm or oedema of the larynx. In non-fatal acute exposure, serious damage does not generally result, as the action of the gas is confined chiefly to the upper respiratory passages. Permanent disability is rare.

Signs and Symptoms: Irritation of the eyes, conjunctivitis, swelling of the eyelids, irritation of the nose and throat, coughing, dyspnoea and vomiting. Irritation of the skin

may be experienced, especially if it is moist. Corneal ulcers have been reported following splashing of ammonia water in the eye.

Recommended M.A.C.: 100 ppm for an 8-hour working period.

Fire Hazard: The minimum explosive limit is 16%; the maximum explosive limit 27% by volume, of the gas in air. Concentrations of 16% are seldom reached under practical conditions, so that ammonia is not a serious fire hazard.

Amyl Acetate (Amyl Acetic Ester; Banana Oil; Pear Oil)

Formula: $CH_3COOC_5H_{11}$

Properties: A colourless liquid, having a banana-like, or pear-like odour. The commercial product is a mixture of the various amyl acetate isomers.
Specific Gravity: 0·879 times as heavy as water.
Boiling Point: 290 to 300°F.
Vapour Density: 4·5 times heavier than air.
Volatility: Is about 25% less volatile than butyl acetate, and is probably 8 or 10 times less volatile than ether.
Soluble in alcohol and ether; very slightly soluble in water.

Uses and Occurrence: Is widely used as a solvent in the manufacture of paints, varnishes and lacquers, lacquer thinners, plastics, linoleum, cements, and as a solvent for nitrocellulose. Is used in the manufacture of photographic film, and in the textile and leather industries.

Mode of Entrance into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: When inhaled in high concentrations, amyl acetate is irritating to the mucous membranes; it also possesses a narcotic effect, and from animal experiments it appears to be more toxic than butyl acetate. A concentration of 1,000 ppm, breathed for half an hour, has caused headache, fatigue, oppression in the chest, and irritation of the eyes and mucous membranes of the nose and throat, with excessive salivation. 5,000 ppm produces deep narcosis in cats in 30 minutes.

Signs and Symptoms: Burning of the eyes, lacrimation, headache, irritation and dryness of the throat, "dopiness", fatigue, and occasionally vague nervousness. There is no definite evidence of organic injury occurring in industrial exposures.

Recommended M.A.C.: 200 ppm for an 8-hour working day.

Fire Hazard: Flash Point: 76 to 92°F. Minimum explosive limit is 1·1%, by volume, of the vapour in air. Class 3 flammable liquid.

Amyl Alcohol (Pentanol)

Formula: All the amyl alcohols contain 5 carbon atoms in their chain. There are at least seven isomers.

Properties: All are liquids, and have boiling points above that of water. Their volatility is low and their vapour densities are 3 to 4·5 times that of air.

Uses and Occurrence: The amyl alcohols are used widely in industry, as solvents in the manufacture of lacquers and varnishes and synthetic rubber.

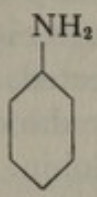
Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: According to animal experiments, the amyl alcohols are about four times as toxic as ethyl alcohol. However, because of their low volatility and their low solubility in the body fluids, they are absorbed slowly and only to a small extent. There is little definite evidence that their use in industry has resulted in poisoning. The vapour may, however, be irritant to the eyes and the upper respiratory tract.

Recommended M.A.C.: A concentration of 100 to 200 ppm has been generally accepted by most authorities as the maximum allowable concentration for isoamyl alcohol for an 8-hour working day.

Fire Hazard: The amyl alcohols all have flash points above 90°F. The minimum explosive limits are slightly higher than 1%, by volume, of the vapour in air. They are class 3 flammable liquids.

Aniline (Aminobenzene; Aminobenzol; Aniline Oil; Phenylamine; Aminophen)

Formula: $C_6H_5NH_2$, or 

Properties: A colourless or yellowish-brown, oily liquid.
Specific Gravity: 1·022 times heavier than water.
Boiling Point: 363°F.
Vapour Density: 3·2 times heavier than air.
Volatility, at ordinary room temperatures, is fairly low.
Soluble in alcohol and ether; slightly soluble in water.

Uses and Occurrence: Aniline may be prepared by the reduction of nitrobenzene; certain of the reduction processes may liberate arsine. Aniline is also prepared by the direct introduction of the NH_2 group into benzol. In the latter process there is no formation of arsine. Aniline is used in the preparation of dyes, inks and pharmaceutical products. It is employed in the synthesis of many organic compounds and in the rubber and textile industries.

Mode of Entry into Body: The most important route of entry, in industrial exposure, is by absorption through the skin, though poisoning may occur through inhalation of the vapour.

Physiological Action and Toxicity: The most important action of aniline on the body is the formation of methaemoglobin, with resulting anoxaemia and depression of the central nervous system. Some investigators believe that in addition to this action, aniline may have a direct toxic action, resulting in a fall in blood pressure and cardiac arrhythmia. In acute exposures, which usually result from spilling the liquid on the skin and clothes, but which may also follow the inhalation of the vapour given off when aniline is heated, the signs and symptoms produced are, for the most part, the results of the methaemoglobinaemia and anoxaemia. In less acute exposure which has been prolonged over some weeks or months, there is usually haemolysis of the red blood

cells, followed by stimulation of the bone marrow and attempts at regeneration. The red cells may show stippling; immature cells may be present. The white blood cells usually show little change either in number of morphology. The liver may be affected, with production of jaundice. The urine is frequently dark brown or wine coloured, and may contain haemoglobin, haematoporphyrin, and, in some cases, excretion products of aniline, such as para-aminophenol. Long continued employment in the manufacture of aniline dyes has been associated with the development of papillomatous growths of the bladder, some of which became malignant. Aniline, and certain other amino compounds, particularly benzidine and beta-naphthylamine, have been suspected of causing the condition (see Naphthylamines). The least concentration of aniline in air having a detectable odour is 0·5 ppm. Concentrations of 10 to 50 ppm may cause symptoms after several hours of exposure.

Signs and Symptoms: Following acute exposure, the signs and symptoms are those of anoxaemia, the workman becoming pale, then deeply cyanosed. He may have throbbing headache, weakness and lassitude, and occasionally nausea, vomiting, abdominal cramps and stumbling gait. Where the exposure has been more severe, there may be low blood pressure and weak, thready pulse, shortness of breath and an increase in the respiratory rate. Coma or delirium may follow, then death. The symptoms in chronic poisoning are weakness, loss of appetite, pallor, anaemia, loss of weight, dark coloured urine, and various nervous manifestations. In cases of bladder tumor, the presenting sign is often the finding of red blood cells in the urine.

Recommended M.A.C.: 5 ppm for an 8-hour working day.

Fire Hazard: The flash point is 168°F.

Antimony and Its Compounds

Symbol: Sb.

Antimony is a silver-gray, crystalline metal having a melting point of 1166°F. It is used in the manufacture of alloys in combination with lead, tin and copper. These alloys are commonly used in making type metal and storage

battery grids. The yellow or orange antimony sulphides are used in the rubber industry and as pigments in the manufacture of paints, glass and pottery glazes, and enamels. Exposure to antimony or its compounds may also occur in the printing, dyeing and textile printing trades, and in the smelting and refining of antimony ore.

Antimony and antimony compounds used in industry commonly contain some arsenic and are generally used in association with compounds of lead.

Mode of Entry into Body: Through inhalation of the fumes; through inhalation and ingestion of the dust. Acts locally on the skin.

Physiological Action and Toxicity: Because of its association with lead and arsenic in industry, it is difficult to accurately assess the toxicity of antimony and its compounds. Animals exposed to fumes of antimony oxide have developed pneumonitis, fatty degeneration of the liver, a decreased leucocyte count affecting in particular the polymorphonuclears, and damage to the heart muscle. In humans, complaints referable to the nervous system have been reported. In assessing human cases however, the possibility of lead or arsenical poisoning must always be borne in mind. Locally antimony compounds are irritant to the skin and mucous membranes.

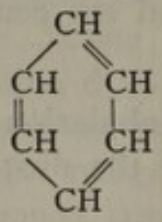
Signs and Symptoms: Irritation and eczematous eruption of the skin, inflammation of the mucous membranes of the nose and throat, metallic taste and stomatitis, gastro-intestinal upset, with vomiting and diarrhoea, and various nervous complaints, such as irritability, sleeplessness, fatigue, dizziness and muscular and neuralgic pains.

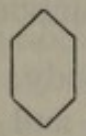
Recommended M.A.C.: 1·0 mgm of antimony per 10 cubic metres of air is generally regarded as the maximum allowable concentration for an 8-hour working day.

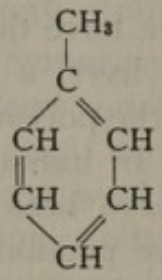
Fire Hazard: Antimony pentasulphide (Sb_2S_5) or "golden sulphide" is combustible and is readily ignited by a small flame. It is hazardous if in contact with oxidizing materials or acids.

Aromatic Hydrocarbons

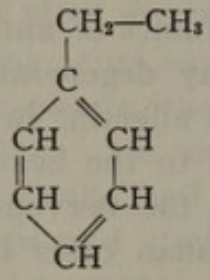
Aromatic hydrocarbons are characterized by the arrangement of their carbon atoms in a particular molecular pattern containing a number of double bonds per molecule. The simplest molecule of the aromatic series is that of benzene containing 6 carbons and 3 double bonds. This molecule may be represented graphically as



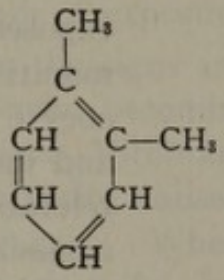
or simply  and the arrangement is known as a "benzene ring". By the substitution of an alkyl group for one or more of the hydrogen atoms, a series of chemicals (the "benzene series") may be built up, thus:



Methyl Benzene or Toluene

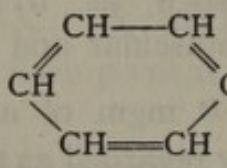


Ethyl Benzene



Dimethyl Benzene or Xylene

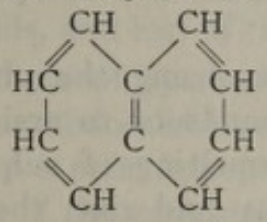
Because of the confusion which sometimes arises in distinguishing the word "benzine" (a petroleum distillate) from "benzene" (a coal-tar distillate) it is considered advisable to refer to the latter throughout this book as benzol and to its derivatives as toluol and xylol.

The radicle  is known as the phenyl radicle, and toluol might therefore be termed phenyl methane. The linking of two phenyl groups results in the compound diphenyl.

By the substitution of the —OH or hydroxy group for one or more of the hydrogen atoms, phenol and the cresols are formed. Similarily, amine radicles (—NH_2), nitro radicles

($—NO_2$), chlorine atoms and other radicles or atoms may be substituted for the hydrogens in the benzol ring, to produce a large number of industrially important derivatives.

The more important members of the benzol series and their derivatives are discussed individually in this section.

In addition to the benzol series, there are two other groups of aromatic hydrocarbons. The naphthalene series is characterized by the presence of a condensed or double-ring structure, in which two carbon atoms are shared by each ring. The simplest of the series, naphthalene, has the formula  . This material is widely used in the manufacture of moth repellents, dyes, synthetic resins and certain organic acids. It is a white, crystalline, volatile solid having a melting point of 176°F. Dermatitis has been reported in susceptible individuals handling the crystals, and inhalation of the vapour may produce headache, nausea and vomiting. However, cases of industrial poisoning are rare. The chlorinated naphthalenes are discussed separately.

Members of the anthracene series contain molecules made up of three condensed rings.

Arsenic and Its Compounds

Symbol: As.

From the point of view of their effect upon health, arsenic and arsenic compounds may be divided into two groups: *(a)* the gas arsine, or hydrogen arsenide; and *(b)* solid compounds of arsenic. Of these, the first is the more serious. For further details concerning its effect, refer to "Arsine".

Arsenic is a silvery, brittle metal, turning black in air. As such, it does not constitute a hazard. The compounds of arsenic most commonly met with in industry are:

1. Arsenic trioxide (white arsenic; arsenious acid; arsenous anhydride; arsenious oxide) As_2O_3. A white, amorphous powder, used as an insecticide, rat poison, weed killer, cattle dip, hide preservative and textile mordant.

2. Copper acetoarsenite (cupric acetoarsenite; paris green; schweinfurth green; imperial green, king's green, patent green; emerald green, etc.) $(CuOAs_2O_3)_3 .Cu(CH_3COO)_2$. An emerald green powder used chiefly as an insecticide.

3. Lead arsenate, $Pb(AsO_4)_2$. A white crystalline compound used as an insecticide.

4. Calcium arsenate, $Ca(AsO_4)_2$. A white powder, used as an insecticide.

5. Copper arsenite (Cupric arsenite; Scheele's green) $CuHAsO_3$. A light green powder used as a pigment in making wallpaper, paints, etc.

In addition to industries using the above compounds, exposure to arsenic compounds or to arsine may occur in many occupations. The smelting of sulphide ores, as of lead, iron, copper, antimony and zinc, the manufacture of sulphuric acid from iron pyrites, the manufacture of brass, dyes, colours, glass enamels, and ferro-silicon may all carry an exposure to arsenic.

Mode of Entry into Body: As a dust, through ingestion and inhalation; acts locally on the skin.

Physiological Action and Toxicity: The occurrence of systemic effects as the result of exposure to arsenic compounds, other than arsine, is very rare in industry. The solid compounds, however, commonly cause local lesions of the skin and mucous membranes. In poisoning with lead arsenate, the clinical picture is usually one of lead poisoning, rather than of arsenic. In the rare cases of systemic arsenic poisoning occurring in industry, as the result of exposure to solid arsenic compounds, the poisoning is usually not severe, and affects the gastro-intestinal and central nervous systems, producing a peripheral neuritis. Arsenic is excreted in the urine and faeces. An urinary excretion of over 0·5 mgm per litre may be accompanied by symptoms of systemic poisoning.

There is some evidence that exposure to dusts of arsenic is associated with an increased incidence of carcinoma of the lung.

Signs and Symptoms: The light arsenic dusts, falling on the skin, tend to remain in the skin folds and on moist areas,

where an itchy dermatitis is set up; this may progress to ulceration. Accompanying the skin eruption there may be conjunctivitis, irritation of the nose and throat, ulceration of the mucous membrane of the nasal septum, hoarseness and cough. The ulceration of the nasal septum may progress to perforation in a matter of a month or so. Long exposure to arsenic dusts may result in eczema, brown pigmentation of the skin of the temples, eyelids and neck, bronzing of the skin of the chest, abdomen and back, keratoses, and epitheliomata. There may be trophic changes in the nails, and loss of the hair.

Symptoms of systemic poisoning are nausea, vomiting, diarrhoea, abdominal pains and a peripheral polyneuritis, with severe neuralgic pains, and slight motor paralysis or weakness of the extensors of the fingers and toes.

Arsenic may be found in the hair and nails for many months after exposure has ceased.

Recommended M.A.C.: 1 mgm per 10 cu. metres of air per 8-hour working day.

Fire Hazard: None, for the solid arsenic compounds.

Arsine (Hydrogen Arsenide; Arsenic Hydride; Arseniuretted Hydrogen)

Formula: AsH_3

Properties: A colourless gas having a rather metallic odour. Vapour density is 2·68 times that of air. Soluble in water. Inflammable.

Occurrence: Arsine is not used in industry, but 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. Industries which may have an arsine hazard include electro-plating, gold extraction, ore smelting, dye works, fertilizer works, manufacture of paper, and many others.

Mode of Entry into Body: By inhalation of the gas.

Physiological Action and Toxicity: The toxicity of arsine is due to its haemolytic action. On entering the blood stream,

it combines with the haemoglobin of the red blood cells; gradually the arsenic in this haemoglobin-arsenic complex is oxidized, and the oxidation process is accompanied by haemolysis of the cell. The resulting anaemia is responsible for the production of many of the symptoms accompanying arsine poisoning; other symptoms result from the haemolysis itself, and occur during the excretion of the haemoglobin. Haemoglobin and methaemoglobin are commonly found in the urine. Less commonly whole blood may be passed. Occasionally, the renal tubules may be plugged by debris, with resultant suppression of urine. Jaundice, which may be severe, is a common result of the haemolysis. Frequently there is oedema of the lungs, which may be accompanied by cyanosis. Kidney damage is common in patients surviving the acute affects of the gas.

Concentrations of 3 to 10 ppm will produce slight symptoms after several hours' exposure. Concentrations of 16 to 30 ppm are dangerous after one half to one hour, and 250 ppm fatal after 30 minutes.

Signs and Symptoms: Signs of poisoning usually develop within several hours of exposure. Headache, dizziness, nausea and vomiting, epigastric pain and weakness occur early, followed by tea-coloured urine, or bloody urine in the more severe cases. Some time later, albumen and casts may appear in the urine, or, in serious cases, there may be suppression of urine. Jaundice and tenderness over the liver may appear about the same time. Blood examination, shows an anaemia which may be marked. In fatal cases, the patient may develop delirium, followed by coma and death. During the acute stage of the poisoning and for some weeks after, arsenic may be demonstrated in the urine.

Recommended M.A.C.: The maximum allowable concentration recommended by most authorities is from 1 ppm to 0·5 ppm for an 8-hour working day.

Fire Hazard: Arsine is flammable, but no data on explosive limits can be found in the literature. It is doubtful that sufficiently high concentrations would accumulate under the conditions of its evolution in industry to constitute an explosive hazard.

Babbitt Metal (White Metal)

There are many different compositions for babbitt metal. For the most part they fall into two main groups: (1) Tin-base metals, containing 80 to 93% tin, 3 to 10% copper, and 5 to 10% antimony. (2) Lead-base metals, containing 70 to 90% lead, 9 to 20% antimony, 0·5 to 9% tin, and occasionally traces of copper.

The hazards associated with the production of babbitt metal are therefore lead and antimony poisoning. For details, refer to "Lead" and Antimony".

Barium and Its Compounds

Symbol: Ba.

Exposure to the salts of barium occurs in the manufacture of paints and lacquers, fireworks, dyes and inks, depilatories, insecticides and rat poisons, glass, and linoleum. Barium compounds may also be used in the rubber industry.

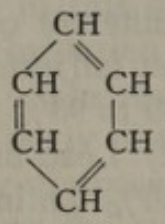
Mode of Entry into Body: Through inhalation and ingestion of the dust. Acts locally on the skin.

Physiological Action and Toxicity: The soluble barium salts, such as the chloride and sulphide, are poisonous when taken by mouth. The insoluble sulphate is non-poisonous, and is used in radiography. Few cases of industrial systemic poisoning have been reported, but one investigator describes a fatal case of poisoning attributed to barium oxide, the symptoms being severe abdominal pain with vomiting, dyspnoea, rapid pulse, paralysis of the right arm and leg, and eventually cyanosis and death. The same investigator produced paralysis in animals with barium oxide and carbonate. The usual result of exposure to the sulphide, oxide and carbonate is irritation of the eyes, nose and throat, and of the skin, with production of dermatitis. The salts mentioned are somewhat caustic.

Recommended M.A.C.: 5 mgm per 10 cubic metres of air for an 8-hour working day.

Fire Hazard: Barium chlorate, nitrate and peroxide are all oxidizing materials. When mixed with combustible materials, the chlorate and the peroxide are explosive and ignite easily by friction or on contact with a small amount of water.

Benzol (Benzene, Phenyl Hydride, Coal Naphtha)

Formula: C_6H_6 or 

Properties: A clear, colourless liquid having a pleasant odour. Inflammable. Specific gravity: 0·878. Boiling point: 176°F. Is highly volatile at room temperatures, the evaporation rate being $\frac{1}{3}$ that of ethyl ether. The vapour is 2·73 times as heavy as air. Benzol is soluble in alcohol and ether, and is slightly soluble in water. It is an excellent solvent, and has been widely used in industry. Because of its extreme toxicity however, its use is discouraged wherever possible. Benzene (benzol) is derived from coal-tar; it must not be confused with benzine, a fraction of crude petroleum, which does not possess the toxic properties of benzol.

Mode of Entry into Body: Poisoning occurs through inhalation of the vapour. Though benzol can penetrate the skin, this route is unimportant in the development of benzol poisoning.

Physiological Action and Toxicity: Locally, benzol has a comparatively strong irritating effect, producing erythema and burning, and in more severe cases, oedema and even blistering. Exposure to high concentrations of the vapour (3,000 ppm or higher) results from accidents such as failure of equipment or spillage. Such exposure, while rare in industry, may result in acute poisoning, characterized by the narcotic action of benzol on the central nervous system. The anaesthetic action of benzol is similar to that of other anaesthetic gases, consisting of a preliminary stage of excitation (benzol jag) followed by depression and, if exposure is continued, death through respiratory failure.

The chronic, rather than the acute form of benzol poisoning is important in industry and is due to a toxic action on the blood-forming tissues. There is no specific blood picture occurring in cases of chronic benzol poisoning.

The bone marrow may be hypoplastic, normal, or hyperplastic, the changes being reflected in the peripheral blood.

Anaemia, leucopenia, macrocytosis, reticulocytosis, thrombocytopenia, high colour index, and prolonged bleeding time may or may not be present. For the supervision of the worker, repeated blood examinations are necessary, including haemoglobin determinations, white and red cell counts and differential smears. Where a worker shows a progressive drop in either red or white cells, or where the white count remains below 5,000 per cu. mm., or the red count below 4·0 million per cu. mm, on two successive examinations, he should be immediately removed from exposure. With this method of supervision of the worker, no permanent damage will result to the blood-forming system.

Following absorption of benzol, elimination is chiefly through the lungs, when fresh air is breathed. The portion that is absorbed, is oxidized, and the oxidation products are combined with sulphuric and glycuronic acids and eliminated in the urine. Benzol has a definite cumulative action, and exposures to relatively high concentrations are not serious from the point of view of causing damage to the blood-forming system, provided that the exposure is not repeated. On the other hand, daily exposure to concentrations of 100 ppm or less may cause damage if continued over a protracted period of time. Benzol may cause degenerative changes in the heart, liver and kidneys, varying in intensity with the concentration and duration of exposure.

Signs and Symptoms: In acute poisoning, the worker becomes confused and dizzy, complains of tightening of the leg muscles and of pressure over the forehead, then passes into a stage of excitement (benzol jag). He laughs, sings, swears, and usually becomes very obstinate. If allowed to remain in exposure, he quickly becomes stupefied and lapses into coma. In non-fatal cases, recovery is usually complete and no permanent disability occurs.

In chronic poisoning the onset is slow, with the symptoms vague; fatigue, headache, dizziness, nausea and loss of appetite, loss of weight, and weakness are common complaints in early cases. Later, pallor, nose-bleeds, bleeding gums, menorrhagia, petechiae and purpura may develop.

There is great individual variation in the signs and symptoms of chronic benzol poisoning, and much of this variability

is undoubtedly due to variation in individual susceptibility, to variation in the concentration of fumes to which individuals are exposed, and to variation in duration of exposure.

Recommended M.A.C.: The recommended maximum allowable concentration is 50 ppm for an 8-hour working period.

Fire Hazard: Benzol flashes at 12°F; its minimum explosive limit is 1·4%, its maximum explosive limit 8%, by volume, of the vapour in air. Class 1 flammable liquid.

Beryllium and Its Compounds

Symbol: Be.

Beryllium is a light, hard, greyish-white metal which in recent years has become increasingly important in the production of copper alloys having high electrical conductivity and high fatigue-resisting properties. The extraction of beryllium from its ore is attended by exposure to acid salts of the metal, particularly the fluoride (BeF_2), the oxyfluoride, and the sulphate ($BeSO_4$), and to beryllium oxide (BeO). Exposure to the oxide also occurs in the casting of beryllium alloys. In the manufacture of fluorescent powders, lamps and sign tubes there may be exposure to beryllium carbonate and to more complex salts, such as zinc manganese beryllium silicate.

Mode of Entry into Body: Through inhalation of the dusts and fumes. Beryllium compounds may act locally on the skin.

Physiological Action and Toxicity: Exposure to beryllium compounds encountered in the extraction of the metal or its oxide from the ore has been attended, in certain individuals, by the development of dermatitis of an oedematous and papulo-vesicular type, chronic skin ulcers, rhinitis, nasopharyngitis, epistaxis, bronchitis and in severe cases, by the development of an acute pneumonitis, with cough, scanty sputum, low-grade fever, râles, dyspnoea and substernal pain. The haemoglobin and white cell counts were usually decreased, the sedimentation rate elevated. Radiographs showed diffuse haziness throughout both lungs, followed by the appearance of soft, ill-defined opacities which were either large or small. The condition usually occurred while the

worker was in exposure, sometimes within one or two months of starting work, and recovery occurred within two months as a rule, though radiographic changes sometimes persisted for 4 months. Certain investigators have reported occasional failure of complete resolution, followed by fibrosis. In severe cases of pneumonitis the patient may die. Necropsies have revealed diffuse pulmonary oedema, haemorrhagic extravasation, large numbers of plasma cells and a relative absence of polymorphonuclear infiltration. On the basis of experimental work with animals, certain investigators are of the opinion that the acute upper and lower respiratory effects are due chiefly to the acid radicle (fluorine or sulphate) present in the dust or fume.

More recently, a delayed form of lung disease, characterized by the occurrence of granulomatous areas in the lung tissue, has been reported in workers manufacturing fluorescent powders, lamps and sign tubes, and casting beryllium alloys. The condition appeared to bear little relation to the intensity of the exposure. Symptoms started either during exposure or might be delayed up to 5 years after leaving the work. The commonest complaints were cough, shortness of breath, loss of appetite, loss of weight, and fatigue. Râles were usually present in the bases and axillae, and the red cell count was frequently elevated. Cyanosis was common and the pulse and respiratory rates were often increased. Radiographically, three stages of the disease were described: (1) a diffuse, uniform granular shadowing extending throughout both lung fields; (2) a diffuse reticular pattern on the granular background; (3) the appearance of distinct nodules scattered through the lungs, with some enlargement and blurring of the hilar shadows. The intensity of the shadowing was usually greater in the middle third of the lung fields. Sufficient time has not yet elapsed to permit accurate estimation of the incidence of the disease, but to date it appears that the incidence is low, probably of the order of 2 to 5%. The prognosis is poor. Clinical improvement may occur gradually over a period of several years, but there appears to be little tendency for the radiographic shadowing to clear. In certain cases, the disease has progressed gradually for some months or years, with death resulting from respir-

atory and cardiac failure. In several instances necropsies have shown the presence of a diffuse fibrosis with coarse strands of hyalinized collagen between the alveoli and, in some places, replacing them. The hyalinized areas contained granulomatous foci, the alveolar walls were thickened and fibrosed, the blood vessels being engorged and dilated. In some cases the hilar lymph nodes showed granulomatous change and fibrosis. Granulomatous change has also been noted in the liver and hyaline fibrosis in the spleen. Two cases of delayed lung disease not coming to autopsy have presented papular lesions on the dorsum of the hands; on biopsy these showed "sarcoid-like" lesions with central necrosis.

During 1948 four cases were reported in the United States in which localized granulomatous lesions developed following penetrating wounds caused by splinters of glass from broken fluorescent light tubes. Several weeks or months following accident, swellings were noted in the injured areas and excision revealed granulomatous tumours, which in one case was shown to contain beryllium.

Recommended M.A.C.: No limits have been set. The exact aetiology of the lung diseases occurring in beryllium workers has not yet been entirely clarified.

Fire Hazard: The nitrate is an oxidizing material and in contact with organic materials may cause violent combustion.

Brass and Bronze

Brass is a copper-zinc alloy of varying composition. It usually contains some lead or tin (sometimes both) and occasionally traces of other metals.

Bronze is an alloy of copper and tin, usually containing some zinc or lead (sometimes both), and small percentages of other metals.

The chief hazards associated with the production and machining of brass and bronze are lead poisoning and metal fume fever, which are described elsewhere in this section.

Brominated Hydrocarbons

The bromine derivatives of the aliphatic hydrocarbons which are most commonly used in industry are methyl bromide, ethyl bromide and ethylene dibromide. Of these, the methyl bromide is the most important due to its higher toxicity and the greater frequency of its use.

Formulae:

CH_3Br	CH_3CH_2Br	$CH_2Br\text{-}CH_2Br$
Methyl Bromide	Ethyl Bromide	Ethylene Dibromide
(Bromomethane)	(Bromoethane)	(Dibromoethane)
	(Hydrobromic ether)	

Properties: All three materials are clear, colourless liquids which are soluble in most organic solvents and insoluble or sparingly soluble in water.

	Specific Gravity	*B.P. °F*	*Vapour Density*	*Min. Exp. Limit*	*Max. Exp. Limit*
Methyl Bromide...	1·732	40	3·27	Practically non-inflammable	
Ethyl Bromide...	1·430	100	3·76	6·75%	11·25%
Ethylene Dibromide.	2·17	268	6·5	Non-inflammable	

Uses and Occurrence: All three liquids are used in organic synthesis. The methyl and ethyl bromides are used as refrigerants; the methyl bromide is used as a fire extinguisher and insecticide; the ethylene dibromide is used as a solvent for oils, waxes, resins, etc., and in the manufacture of ethyl gasoline.

Mode of Entry into Body: Through inhalation of the vapour. May act locally on the skin.

Physiological Action and Toxicity: The bromine derivatives are less narcotic but much more toxic than the corresponding chlorine compounds. Methyl bromide and ethylene dibromide are reported to be 8 times more toxic on inhalation than ethyl bromide. However, because of its greater volatility, methyl bromide is a much more frequent cause of

poisoning. Death following acute poisoning is usually caused by its irritant effect on the lungs. In chronic poisoning, death is due to injury to the central nervous system. Fatal poisoning has always resulted from exposures to relatively high concentrations of methyl bromide vapour (from 8,600 to 60,000 ppm). Non-fatal poisoning has resulted from exposures to concentrations as low as 100 to 500 ppm. In addition to the lung and central nervous system injury mentioned, the kidneys may be damaged, with development of albuminuria and, in fatal cases, cloudy swelling and/or tubular degeneration. The liver may be enlarged. There are no characteristic blood changes.

Signs and Symptoms: The onset of symptoms following the inhalation of methyl bromide vapour is usually delayed for 4 to 6 hours, though the latent period may vary from two to forty-eight hours. In fatal poisoning, the early symptoms are headache, visual disturbances, nausea and vomiting, smarting of the eyes, irritation of the skin, listlessness, vertigo and tremor. Progress is nearly always rapid, with the development of convulsions, fever, pulmonary oedema, cyanosis, unconsciousness and death. Signs of involvement of the nervous system may be present before death. The clinical picture in non-fatal poisoning is extremely variable. Fatigue, blurred or double vision, nausea and vomiting are frequent; incoordination, tremors, convulsions, exaggeration of the patellar reflexes and a positive Babinski's sign may develop. Nearly every type of nervous disturbance has been reported. The pulmonary symptoms are comparatively slight. Recovery is frequently prolonged and there may be permanent injury, commonly characterized by sensory disturbances, weakness, disturbances of gait, irritability, and blurred vision. Locally, methyl bromide is extremely irritant to the skin and may produce severe burns.

Recommended M.A.C.: For methyl bromide, 20 ppm for repeated 8-hour exposures. For ethyl bromide, 200 ppm for repeated 8-hour exposures.

Fire Hazard: Ethyl bromide is flammable (see Properties, above).

Bromine

Formula: Br_2

Properties: A reddish brown liquid which fumes on contact with air, giving off fumes of the same colour. The fumes are irritating.

Boiling Point: 137·5°F.

Vapour Density: The fumes are 5·5 times heavier than air.

Bromine is soluble in alcohol, ether, chloroform, potassium bromide and carbon disulphide. Slightly soluble in water.

Uses and Occurrence: In the manufacture of pharmaceuticals and chemicals; as a disinfectant.

Mode of Entry into Body: Through inhalation of the vapour. Acts locally on the skin.

Physiological Action and Toxicity: The action of bromine is essentially the same as that of chlorine, being an irritant to the mucous membranes of the eyes and upper respiratory tract. Severe exposures may result in pulmonary oldema. Usually, however, the irritating qualities of the chemical force the workman to leave the exposure before serious poisoning can result. Concentrations of 40 to 60 ppm are dangerous for short exposures, and concentrations of 1000 ppm are rapidly fatal.

Signs and Symptoms: Irritation and burning of the eyes, lacrimation, cough and irritation of the nose and throat.

Recommended M.A.C.: 1 ppm for an 8-hour working day.

Fire Hazard: Has an oxidizing effect, with production of heat, and may cause fire if in contact with organic materials.

Butadiene (Vinyl Ethylene; 1,3 Butadiene)

Formula: $CH_2CHCHCH_2$ or $CH_2 = \underset{\displaystyle H}{\overset{\displaystyle |}{C}} - \underset{\displaystyle H}{\overset{\displaystyle |}{C}} = CH_2$

Properties: Gas at ordinary temperatures. Characteristic pungent odour.

Boiling Point: 23°F.

Uses: Manufacture of synthetic rubber. For this purpose it is usually kept under pressure in steel cylinders.

Mode of Entry into Body: Through inhalation of the gas or vapour.

Physiological Action and Toxicity: The vapour causes irritation of the eyes, nose, throat and lungs. At concentrations below 22,000 ppm it is mildly narcotic. There is no evidence of its having a cumulative action. It is irritant to the skin.

Signs and Symptoms: Irritation of the eyes, nose, throat and lungs; cough, fatigue and drowsiness. Its characteristic odour gives warning even in low concentrations.

Recommended M.A.C.: No maximum allowable concentration has been recommended, but it is believed that if the concentration is kept below 22,000 ppm, no permanent damage will result.

Fire Hazard: The minimum explosive limit is 2·2% by volume.

Butanone (Methyl Ethyl Ketone; M.E.K.)

Formula: $CH_3COC_2H_5$

Properties: A colourless liquid having an acetone-like odour. Inflammable.

Specific Gravity: 0·805 times as heavy as water.

Boiling Point: 176°F.

Vapour Density: 2·41 times heavier than air.

Readily volatile.

Soluble in alcohol and ether, and in most oils. Moderately soluble in water.

Uses and Occurrence: Used as a solvent, frequently mixed with acetone, in the manufacture of lacquers and varnishes, pharmaceuticals and cosmetics, artificial leather and paint removers.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Concentrations of 3,000 ppm are moderately irritating to the eyes and nose, and concentrations of 10,000 ppm are intolerable. In higher concentrations butanone is a narcotic; however, because of its irritating qualities, it is unlikely that exposure to narcotic concentrations can occur in industry, unless the workman is unable to withdraw from the exposure. It is possible that mixtures of butanone and acetone have a stronger narcotic action than either of these substances alone.

Signs and Symptoms: Lacrimation and burning of the eyes, irritation of the nose and throat, headache, possible "dopiness" and mental confusion.

Recommended M.A.C.: 200 ppm for an 8-hour working day.

Fire Hazard: Dangerous. Flash point: 30°F. Minimum explosive limit is 1·8%, maximum explosive limit 11·5%, by volume, of the vapour in air. Class 2 flammable liquid.

Butyl Acetate (Normal Butyl Acetate)

Formula: $CH_3COOC_4H_9$

Properties: A colourless liquid having a fruity odour, resembling pears.
Specific Gravity: 0·88 times as heavy as water.
Boiling Point: 260°F.
Vapour Density: 4 times heavier than air.
Volatility: Probably more than 5 or 6 times less volatile than ether.
Soluble in alcohol and ether. Slightly soluble in water.

Uses and Occurrence: Is used as a solvent in the manufacture of paints, varnishes, lacquers, leather and airplane dope. Solvent for synthetic resins and natural gums.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Butyl acetate has a stronger irritant and narcotic effect than the lower members of the acetate series, such as ethyl and methyl acetate. Concentrations of 7,000 to 10,000 ppm are markedly irritating to the eyes and nose. Because butyl acetate is seldom used in the pure state, it is rather difficult to assess reports of industrial poisoning.

Signs and Symptoms: Burning and lacrimation of the eyes, irritation of the nose and throat, headache, dizziness, drowsiness, cough and nausea have all been reported in workmen exposed to materials containing butyl acetate.

Recommended M.A.C.: 200 ppm for an 8-hour day.

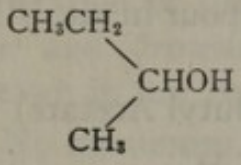
Fire Hazard: Flash point is 72°F. Minimum explosive limit is 1·7%; maximum explosive limit 15%, by volume, of the vapour in air. Class 3 flammable liquid.

Butyl Alcohol (Butanol; Butyric Alcohol)

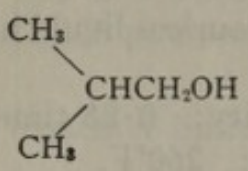
Formulae: The butyl alcohols are:

(1) Normal butyl alcohol (butanol; butyric alcohol)
$CH_3CH_2CH_2CH_2OH$

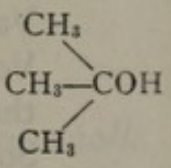
(2) Secondary butyl alcohol (butanol-2; methyl ethyl carbinol)



(3) Isobutyl alcohol



(4) Tertiary butyl alcohol (pseudobutyl alcohol)



Properties: The first three butyl alcohols mentioned are liquids; the last occurs as white crystals having a camphor-like odour.

	Boiling Point	*Vapour Density*
Normal butyl alcohol	243°F	2·55
Secondary butyl alcohol	211°F	2·55
Isobutyl alcohol	225°F	2·55
Tertiary butyl alcohol	181°F	2·55

All are soluble in water, alcohol and ether.

Uses and Occurrence: The butyl alcohols are finding increasing use in industry, particularly in the manufacture of lacquers and synthetic resins and plastics.

Mode of Entry into Body: Through inhalation of the vapour. May act locally on the skin.

Physiological Action and Toxicity: Though animal experiments have shown the butyl alcohols to possess toxic properties, they have produced few cases of poisoning in industry because of their low volatility. In 1944, the use of normal

butyl alcohol in the manufacture of raincoats was reported to have resulted in irritation of the eyes, with corneal inflammation, slight headache and dizziness, slight irritation of the nose and throat, and dermatitis about the finger-nails and along the sides of the fingers. Keratitis has also been reported by another group of investigators.

Recommended M.A.C.: Concentrations of 50 to 100 ppm are regarded as being safe for an 8-hour working day.

Fire Hazard: The butyl alcohols are all inflammable, their flash points varying from 52°F (tertiary butyl alcohol) to 82°F (normal and isobutyl alcohols). The minimum explosive limits of the latter two alcohols are about 1·7% by volume, of their vapor in air.

Cadmium and its Compounds

Symbol: Cd.

Properties: Melts at 609°F and boils at 1412°F. When heated, cadmium forms dense brown fumes of cadmium oxide.

Uses and Occurrence: In recent years, cadmium has assumed increasing importance as an ingredient of metal alloys and as an electro-plating material. Exposure occurs in the smelting of the ore, the making of alloys, electro-plating, in the making of paint pigments, and in welding, where the cadmium may occur as the plating upon the metal being welded, or as an ingredient of the welding rod.

Mode of Entry into Body: Absorption occurs through inhalation of cadmium oxide fumes when metal coated with, or containing cadmium is heated. Inhalation of powdered cadmium compounds also occurs. Domestically, ingestion of cadmium occurs when fruit juice or other acid-containing material acts upon cadmium plated utensils.

Physiological Action and Toxicity: The inhalation of fumes or dusts of cadmium primarily affects the respiratory tract; the kidneys may also be affected. Even brief exposure to high concentrations may result in pulmonary oedema and death. Usually the oedema is not massive, and little pleural effusion is present. In fatal cases, fatty degeneration of the liver, and acute inflammatory changes in the kidneys have

been noted. Ingestion of cadmium results in a gastro-intestinal type of poisoning resembling food poisoning in its symptoms.

The lethal concentration of cadmium fumes for man has been calculated to be about 5 mgm/cu. metre for an 8-hour exposure, where these fumes are generated by heating of the metal. With arc-produced fumes, the lethal concentration is estimated to be about half this value, with the same length of exposure. Concentrations lower than 5 mgm/cu. metre have caused temporary disability in man, and experimental work on animals indicates that exposures to non-lethal concentrations of fume may result in pulmonary fibrosis persisting for at least 6 months after exposure has ceased.

Signs and Symptoms: Inhalation of dust or fumes causes dryness of the throat, cough, headache, a sense of constriction in the chest, dyspnoea, and vomiting. More severe exposure results in marked lung changes, with persistent cough, pain in the chest, severe dyspnoea and prostration which may terminate fatally. X-ray changes are usually similar to those seen in broncho-pneumonia. The urine is frequently dark. These symptoms are usually delayed for some hours after exposure, and fatal concentrations may be breathed without sufficient discomfort to warn the workman to leave the exposure.

Ingestion of cadmium results in sudden nausea, salivation, vomiting and diarrhoea and abdominal pain and discomfort. Symptoms begin almost immediately after ingesting the contaminated food.

Recommended M.A.C.: The recommended maximum allowable concentration is 1 mgm per 10 cu. metres of air for an 8-hour work period.

Fire Hazard: None.

Calcium Cyanamide (Nitrolime; Lime Nitrogen)

Formula: $CaCN_2$

Properties: A white crystalline solid, decomposed by water and by moist atmosphere with the formation of

cyanamide ($CNNH_2$). The latter is a colourless solid which, on further hydrolysis, forms urea and finally ammonia.

Uses and Occurrence: Is used as a fertilizer, in the manufacture of nitrogen products, and in the hardening of steel and iron.

Mode of Entry into Body: Acts locally on the skin; the dust may be inhaled.

Physiological Action and Toxicity: Calcium cyanamide acts locally on the skin as a primary irritant, and the lesions produced vary from erythema to acute and subacute eczema. Usually the moist skin areas are attacked first, but the material is spread by scratching and parts of the body not ordinarily exposed may be affected. In severe cases ulceration may develop; the ulcers are usually covered by a black, necrotic crust. There is frequently irritation of the conjunctivae and of the mucous membranes of the nose and throat, with production of conjunctivitis, inflammed ulcers in the nose and throat, rhinitis and gingivitis. Systemically, headache, flushing of the skin of the head and neck, shortness of breath, vasodilatation with lowered blood pressure, and rapid pulse have all been described among persons who have consumed alcohol. No fatalities have been reported. Calcium cyanamide is not believed to have a cumulative action. The fatal dose, by ingestion, is probably around 20 to 30 grams for an adult.

Recommended M.A.C.: No value has been set.

Fire Hazard: Cyanamide has a flash point of 285°F.

Carbon Dioxide (Carbonic Acid; Carbonic Gas; Dry Ice)

Formula: CO_2

Properties: A colourless, odourless gas at ordinary room temperatures. May be liquefied or compressed to a white, snow-like solid (dry ice).
Boiling Point: 109°F.
Vapour Density: 1·53 times heavier than air.
Soluble in water, part of the dissolved gas combining with the water to form carbonic acid, H_2CO_3

Uses and Occurrence: Is used in the manufacture of many chemicals. Occurs wherever organic material is oxidized, as in the combustion of coal and wood, in fermentation processes, and as a product of respiration. Is used as a refrigerant.

Mode of Entry into Body: Through inhalation of the gas.

Physiological Action and Toxicity: Carbon dioxide is now generally regarded as a simple asphyxiant, symptoms resulting only when such high concentrations are reached that there is insufficient oxygen in the atmosphere to support life.

Signs and Symptoms: The signs and symptoms are those which precede asphyxia, namely headache, dizziness, shortness of breath, muscular weakness, drowsiness, and ringing in the ears. Removal from exposure results in rapid recovery. Contact of carbon dioxide snow with the skin may cause a "burn".

Recommended M.A.C.: 5,000 ppm for an 8-hour working day.

Fire Hazard: None.

Carbon Disulphide (Carbon Bisulphide)

Formula: CS_2.

Properties: When pure, carbon disulphide is a clear, colourless liquid having a slight ethereal odour. On exposure to light, the liquid turns yellowish. The commercial product has a strong, disagreeable odour. Inflammable.
Specific Gravity: 1·26.
Boiling Point: 115·4°F.
Vapour Density: 2·63.
Volatility: 1·8 times less volatile than ether; readily volatile.
Solubility: Only slightly soluble in water; is soluble in alcohol, ether, benzene and fatty oils in all proportions.

Uses and Occurrence: As a solvent for sulphur, in the cold vulcanization of rubber; more recently it is being increasingly used in the manufacture of artificial silk by the viscose process.

Mode of Entry into Body: Chiefly by inhalation of the vapour; skin absorption of toxic amounts is possible when the liquid comes in contact with the skin.

Physiological Action and Toxicity: On entering the blood, carbon disulphide is carried to all parts of the body. The chief effect is on the central nervous system, acting as a narcotic and anaesthetic in acute poisoning with death following from respiratory failure. The anaesthetic action is much more powerful than that of chloroform. In chronic poisoning, the effect on the nervous system is one of central and peripheral damage, which may be permanent if the damage has been severe. Sensory symptoms usually precede motor involvement. A secondary anaemia may be present, and the liver, kidneys and heart may be damaged.

Exposure to concentrations of 300 to 400 ppm in air for several hours will cause slight symptoms. Concentrations of 500 to 800 ppm cause headache and dizziness fairly quickly. Concentrations of 1000 to 3000 ppm are dangerous if exposure lasts for more than 30 minutes to 1 hour.

Impure carbon disulphide has a more toxic effect than the pure form. Elimination is chiefly by the lungs, about one-quarter being eliminated through the urine and skin.

Signs and Symptoms: In acute poisoning, early excitation of the central nervous system resembling alcoholic intoxication occurs, followed by depression, with stupor, restlessness, unconsciousness, and possibly death. If recovery occurs, the patient usually passes through the after-stage of narcosis, with nausea, vomiting, headache, etc. In chronic poisoning, the picture is that of involvement of the nervous system, with neuritis and disturbance of vision being the commonest early changes. Sensory changes, such as a crawling sensation in the skin, sensations of heaviness and coldness, and visually, "veiling" of objects so that they appear indistinct, are noticed first. Often there is pain in the affected parts, particularly the limbs. These symptoms are followed by motor nerve involvement; weakness of the limbs develops and there is gradually increasing loss of strength. Wasting of the muscles may occur. Mental symptoms vary from simple excitation or depression and

irritability in the mild cases to mental deterioration, Parkinsonian paralysis, and even insanity. These changes are accompanied by insomnia, loss of memory, and personality changes. Chronic fatigue is a very common complaint.

Recommended M.A.C.: The maximum allowable concentration recommended for an 8-hour exposure is generally accepted as 20 ppm. However, there is some variation in the figure, some authorities believing that a safe concentration would have to be as low as 1 ppm. In Ontario, the recommended M.A.C. is 15 ppm.

Fire Hazard: Dangerous. Flash point: −22°F. Minimum explosive limit: 1%; maximum explosive limit: 50% by volume, of the vapour in air. The vapour ignites when in contact with objects at temperatures of 212°F to 223°F (such as steam pipes and electric light bulbs). Because the vapour is heavier than air, it tends to hang low, and may travel considerable distances to a source of ignition and flash back. Class 1 flammable liquid, more dangerous than gasoline.

Carbon Monoxide

Formula: CO.

Properties: A colourless, nearly odourless gas, having a density almost the same as that of air (0·967). Inflammable.

Occurrence: Is produced by the incomplete combustion of carbon-containing materials, and is nearly always present in the gases given off from burning substances. It is present in varying amounts in exhaust gases, illuminating gas, gases from explosions, smoke, blast furnace gas, etc. Poisoning is most common from automobile exhaust gas, illuminating gas, and from faultily designed or unvented gas burners in stoves or heaters.

Mode of Entry into Body: Through inhalation of the gas.

Physiological Action and Toxicity: Carbon monoxide has an affinity for haemoglobin 210 times that of oxygen, and by combining with the haemoglobin, renders the latter incapable of carrying oxygen to the tissues. The effect on the body is therefore one of asphyxia, predominantly. In addition to this action, the presence of CO-haemoglobin in the blood

interferes with the dissociation of the remaining oxyhaemoglobin, so that the tissues are further deprived of oxygen. Thus, a person with an anaemia of 50% is better able to utilize the available oxygen in his blood than is a man with a CO saturation of 50%.

A concentration of 400 to 500 ppm in the air can be inhaled without appreciable effect for 1 hour. An hour's exposure to 600 to 700 ppm will cause barely appreciable effects, and a similar exposure to 1,000 to 1,200 ppm produces definite, but not dangerous, symptoms. An hour's exposure to a concentration of 1,500 to 2,000 ppm is dangerous, and concentrations of 4,000 ppm and over are fatal in less than an hour.

Carbon monoxide is eliminated through the lungs when air free from CO is inhaled. Over half the CO is eliminated in the first hour, where the exposure has been moderate.

Signs and Symptoms: With concentrations up to 10% of CO-haemoglobin in the blood, there rarely are any symptoms. Concentrations of 20 to 30% cause shortness of breath on moderate exertion and slight headache. Concentrations from 30 to 50% cause severe headache, mental confusion and dizziness, impairment of vision and hearing, and collapse and fainting on exertion. With concentrations of 50 to 60%, unconsciousness results, and death may follow if exposure is long. Concentrations of 80% result in almost immediate death.

Acute cases of poisoning, resulting from brief exposures to high concentrations, seldom result in any permanent disability if recovery takes place. The possibility of chronic effects as the result of repeated exposure to lower concentrations is controversial. Glycosuria does occur, and heart irregularities have been reported. Other workers have found that where the poisoning has been relatively long and severe, cerebral congestion and oedema may occur, resulting in long-lasting mental or nervous damage. Repeated exposure to low concentrations of the gas, up to 100 ppm in air, are generally believed to cause no signs of poisoning or permanent damage. Industrially, sequelae are rare, as exposure though often severe, is usually brief.

Recommended M.A.C.: The maximum allowable concentration for an 8-hour working day is generally accepted as 100 ppm.

Fire Hazard: It is a combustible gas, the minimum explosive limit being 12·5% and the maximum explosive limit being 74% of gas, by volume, in air.

Carbon Tetrachloride (Perchloromethane: Tetrachloromethane; Benzino-form)

Formula: CCl_4

Properties: A colourless liquid, having a chloroform-like odour. Non-inflammable.

Specific Gravity: 1·58 times heavier than water.

Boiling Point: 170°F.

Vapour Density: 5·3 times heavier than air.

Volatility: Is about 3 times less volatile than ether at ordinary room temperature.

Soluble in ether, alcohol, chloroform, benzol, benzine and most fixed and volatile oils. It is only very slightly soluble in water.

Commercial carbon tetrachloride contains impurities which may be toxic, especially carbon disulphide, hydrogen sulphide, hydrochloric acid, phosgene, and organic chlorine and sulphur compounds. In the presence of heat and air, carbon tetrachloride partially decomposes, forming phosgene and hydrochloric acid. This has occurred in certain instances where fire extinguishers were charged with carbon tetrachloride.

Uses and Occurrence: Carbon tetrachloride is an excellent solvent, and has the desirable quality of being non-inflammable. Its use in industry is therefore widespread.

Mode of Entry into Body: Industrially, absorption occurs chiefly through inhalation of the vapour, which tends to concentrate in the lower part of the workroom. Carbon tetrachloride can be absorbed through the skin.

Physiological Action and Toxicity: Carbon tetrachloride has a narcotic action resembling that of chloroform, though not as strong. Following exposures to high concentrations, the workman may become unconscious, and if exposure is not

terminated, death can follow from respiratory failure. Death may even occur under light anaesthesia, from ventricular fibrillation. In cases of narcosis that recover, the after-effects are more serious than those of delayed chloroform poisoning, usually taking the form of damage to the kidneys, liver and lungs. Exposure to lower concentrations, insufficient to produce unconsciousness, usually results in severe gastro-intestinal upset, and may progress to serious kidney and hepatic damage. The kidney lesion is an acute nephrosis; the liver involvement consists of an acute degeneration of the central portions of the lobules. With serious renal damage oliguria or anuria develop, and oedema of the lungs may follow. Where recovery takes place, there is usually little or no permanent disability. Marked variation in individual susceptibility to carbon tetrachloride exists, some employees appearing to be unaffected by exposures which seriously poison their fellow-workers. Alcoholism and previous liver and kidney damage seem to render the individual more susceptible. Concentrations of the order of 1,000 to 1,500 ppm are sufficient to cause symptoms if exposure continues for several hours. Repeated daily exposure to such concentrations may result in poisoning, and where carbon tetrachloride is used daily, care must be taken to keep the concentration lower than 50 ppm.

Though the common form of poisoning following industrial exposure is usually one of gastro-intestinal upset, which may be followed by renal damage, other cases have been reported in which the central nervous system has been affected, with production of polyneuritis, narrowing of the visual fields, and other neurological changes. Prolonged exposure to small amounts of carbon tetrachloride has also been reported as causing cirrhosis of the liver.

Locally, a dermatitis may be produced following long or repeated contact with the liquid. The skin oils are removed, and the skin becomes red, cracked and dry. The effect of carbon tetrachloride on the eyes either as a vapour or as a liquid, is one of irritation with lacrimation and burning.

Signs and Symptoms: Industrial poisoning is usually acute, with malaise, headache, nausea, dizziness, and confusion, which may be followed by stupor and sometimes loss of

consciousness, Symptoms of liver and kidney damage may follow later, with development of dark urine, sometimes jaundice and liver enlargement, followed by scanty urine, albuminuria and renal casts: uraemia may develop and cause death. Where the exposure has been less acute, the picture is usually one of headache, dizziness, nausea, vomiting, epigastric distress, loss of appetite, and fatigue. Visual disturbances (blind spots, spots before the eyes, a visual "haze", and restriction of the visual fields), secondary anaemia, and occasionally a slight jaundice may occur. Dermatitis may be noticed on the exposed parts.

Recommended M.A.C.: Most authorities recommend a maximum allowable concentration of 50 ppm for an 8-hour working period.

Fire Hazard: None.

Chloride of Lime (Calcium Hypochlorite; Calcium Oxychloride; Bleaching Powder; Chlorinated lime)

Formula: $CaOCl_2$

Properties: A white powder having a strong odour of chlorine. Decomposes in water and by the action of acids, liberating hypochlorous acid and chlorine.

Uses and Occurrence: Used in the bleaching of textiles, in organic synthesis, and as a deodorizer and disinfectant.

Mode of Entry into Body: Through inhalation of the dust and the liberated chlorine. Chlorinated lime, as a powder or in solution, may act locally on the skin.

Physiological Action and Toxicity: Contact of chlorinated lime with moisture or acids results in the formation of hypochlorous acid, hydrochloric acid and free chlorine. These materials are all strongly irritant to the skin and mucous membranes. Chlorine and hydrochloric acid are discussed separately.

Signs and Symptoms: The most important result of exposure to chlorinated lime is the development of dermatitis. Certain individuals appear to be more readily affected than others. Other symptoms which may develop are: irritation of the

eyes, lachrymation, cough and irritation of the upper respiratory tract. The symptoms resulting from exposure to strong concentrations of chlorine are discussed under the latter heading.

Recommended M.A.C.: No values have been recommended other than for exposures to chlorine and hydrogen chloride.

Fire Hazard: Chlorinated lime is an oxidizing agent. It is not combustible, but in contact with organic materials may cause fire.

Chlorinated Diphenyls

The chlorinated diphenyls are derivatives of diphenyl (*q.v.*), one or more hydrogen atoms being replaced by chlorine atoms. Where the chlorine content is low, these materials are oily liquids; chlorinated diphenyls having a higher chlorine content are solids. All are insoluble in water, glycerine and glycol, but are soluble in most organic solvents, thinners and oils. They are non-oxidizing, are permanently thermo-plastic, and possess low volatility. They are used chiefly as insulating materials in the manufacture of electrical equipment.

Mode of Entry into Body: Through inhalation of the vapour or fumes from the heated materials. Contact with the solid materials or their dusts produces dermatitis.

Physiological Action and Toxicity: Like the chlorinated naphthalenes, the chlorinated diphenyls have two distinct actions on the body, namely, a skin effect and a toxic action on the liver. The lesion produced in the liver is an acute yellow atrophy. This hepato-toxic action of the chlorinated diphenyls appears to be increased if there is exposure to carbon tetrachloride at the same time. The higher the chlorine content of the diphenyl compound, the more toxic is it liable to be. Oxides of chlorinated diphenyls are more toxic than the unoxidized materials.

Signs and Symptoms: The skin lesion is known as chloracne, and consists of small pimples and dark pigmentation of the exposed areas, initially. Later, comedones and pustules develop. In persons who have suffered systemic intoxi-

cation, the usual signs and symptoms are nausea, vomiting, loss of weight, jaundice, oedema and abdominal pain. Where the liver damage has been severe, the patient may pass into coma and die.

There appears to be great variation in individual susceptibility to the systemic action of the chlorinated diphenyls. Usually the workman is in exposure for some weeks or months before becoming ill. Jaundice may be the presenting sign.

Recommended M.A.C.: 10 mgm. per 10 cubic metres of air, for an 8-hour day, is the generally accepted maximum allowable concentration, though it is possible that higher concentrations, up to 50 mgm per 10 cu. metres for an 8-hour working day, may be non-injurious to health.

Fire Hazard: The chlorinated diphenyls are fire-resistant, and probably non-inflammable.

Chlorinated Hydrocarbons

A. Chlorinated Aliphatic Hydrocarbons.

In recent years, the industrial application of chlorinated hydrocarbons of the aliphatic or straight-chain series has increased. As they are excellent solvents and generally non-flammable, large quantities are used in degreasing metal, cleaning textiles, as solvents for oils, rubber, gums, tar and other organic compounds, and as thinners for lacquers.

The chlorine derivatives of the aliphatic hydrocarbons may be divided into two groups: (1) Saturated compounds, in which each carbon atom has each of its four bonds linked to a hydrogen atom, a chlorine atom, or another carbon atom. The compounds in this group may be formed by adding chlorine atoms to an unsaturated hydrocarbon of the olefine (*e.g.*, ethylene) series or of the acetylene series; or the compound may be formed by replacing one or more hydrogen atoms in a hydrocarbon of the paraffin (*e.g.*, methane) series with one or more chlorine atoms. (2) Unsaturated compounds, in which some carbon bonds remain unsaturated, and one or more double bonds still exist between carbon atoms. These compounds are formed by the addition or substitution of chlorine atoms in hydrocarbons of the olefine or acetylene series.

On the following page are listed the chlorinated derivatives of the lower aliphatic hydrocarbons, together with their more important physical properties. Those marked with an asterisk are the ones most commonly used in industry.

Saturated Chlorinated Hydrocarbons

Common Name	*Chemical Name*	*Formula*	*B.P. °F.*	*Vapour Density*	*M.A.C. in ppm.*	*Flash Point °F.*	*Minimum Expl. Limit*
*1. Methyl Chloride	Monochloromethane	CH_3Cl	−11	1·78	100	Gas	8·2%
*2. Methylene Chloride	Dichloromethane	CH_2Cl_2	104	2·93	500	Practically non-inflam.	
3. Chloroform	Trichloromethane	$CHCl_3$	142	4·13	100	Non-inflammable	
*4. Carbon Tetrachloride	Tetrachloromethane	CCl_4	170	5·3	50	Non-inflammable	
5. Ethyl Chloride	Monochloroethane	CH_3-CH_2Cl	54	2·22	1,000	−45	3·6%
*6. Ethylene Dichloride	Dichloroethane	CH_2Cl-CH_2Cl	183	3·42	75	65	6·2%
7. Ethylene Trichloride	Trichloroethane	$CHCl_2$-CH_2Cl	237	4·61	200	Non-inflammable	
*8. Acetylene Tetrachloride	Tetrachloroethane	$CHCl_2$-$CHCl_2$	297	5·73	5	Non-inflammable	
9.	Pentachloroethane	$CHCl_2$-CCl_3	325	6·99	.....	Non-inflammable	

Unsaturated Chlorinated Hydrocarbons

1. Vinyl Chloride	Chloroethylene	CH_2=$CHCl$	7	2·15	1,000	<20	4·0%
2. Acetylene Dichloride	Dichloroethylene	$CHCl$=$CHCl$	141	3·35	200	43	9·7%
*3. Acetylene Trichloride	Trichloroethylene	$CHCl$=CCl_2	188	4·53	150	Non-inflammable	
4. Perchloroethylene	Tetrachloroethylene	CCl_2=CCl_2	248	5·72	100	Non-inflammable	

Comparative Toxicity: The substitution of a chlorine (or other halogen) atom for a hydrogen greatly increases the anaesthetic action of a member of the aliphatic hydrocarbons. In addition, the chlorine derivative is usually less specific in its action and may affect other tissues of the body in addition to those of the central nervous system; in many cases the chlorine derivative is quite toxic. Thus, chloroform, in addition to its narcotic qualities, may cause liver, heart, and kidney damage, whereas methane (CH_4) is a simple asphyxiant.

As a general rule, the unsaturated chlorine derivatives are highly narcotic but less toxic than the saturated derivatives, causing degenerative changes in the liver and kidneys less frequently. In the saturated group, the narcotic effect is enhanced with an increase in the number of chlorine atoms. However, there is less relationship between the number of chlorine atoms present and the toxicity of the compound.

In dealing with the chlorinated hydrocarbons, it must be remembered that a toxic action may result from repeated exposure to concentrations which are too low to produce a narcotic effect, and which, consequently, are too low to give warning of danger. Individual susceptibility is also important when poisoning by this group of solvents is being considered. Certain workmen may be seriously affected by concentrations that seem to have no effect on fellow employees in the same exposure.

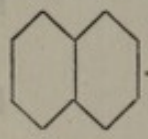
The chlorinated hydrocarbons of the aliphatic series which are most commonly used in industry are discussed elsewhere in this section. See methyl chloride, methylene chloride, carbon tetrachloride, ethylene dichloride, tetrachloroethane, tetrachloroethylene, trichloroethylene and vinyl chloride.

B. Chlorinated Aromatic Hydrocarbons

The chlorinated hydrocarbons of the aromatic series (derivatives of benzol and its homologues, toluol and xylol, of naphthalene and of anthracene) and chlorinated alicyclic compounds (possessing an irregular ring structure) are finding increasing use in industry. In most instances it is difficult to predict their toxicity and at the present time

much work remains to be done in this field. However, in the case of most aromatic chlorine compounds, their toxicity is usually no greater, and frequently is less than that of the corresponding aromatic hydrocarbon. Details of the action and toxicity of the commonly used chlorine derivatives are discussed under the specific compound (see index).

Chlorinated Naphthalenes ("Halowax")

The chlorinated naphthalenes are chlorine derivatives of naphthalene, which has the formula $C_{10}H_8$, or  .

According to the degree of chlorination, their physical state varies from a fluid to an amorphous wax. All are non-oxidizing and possess low volatility. They are insoluble in water and in most acids and alkalis, but are soluble in many organic solvents and oils. They are chiefly used as electrical insulating materials, and in the flame-proofing, moisture-proofing, acid-proofing and insect-proofing of wood and fabrics. Industrial exposure usually occurs when the chloronaphthalenes are heated, as in burning insulation off electric cables, or when the chloronaphthalene is used in solution.

Mode of Entry into Body: Through inhalation of the vapour or fumes from the heated materials. Direct contact with the skin produces dermatitis.

Physiological Action and Toxicity: The action of the chlorinated naphthalenes on the body is quite similar to that of the chlorinated diphenyls, the chief effects being the production of chloracne of the skin and, systemically, an acute yellow atrophy of the liver. The common chlorinated naphthalenes used are mixtures of tri-, tetra-, penta- and hexachloronaphthalenes. The chlorine content of these materials runs from 20 to 65%. In general, the higher the chlorine content, the greater the toxicity.

Signs and Symptoms: Exposure to fumes, or direct contact of the skin with solid chloronaphthalenes produces a form of dermatitis known as chloracne, characterized by the appearance of pimples and areas of dark pigmentation on the exposed areas, and followed by the development of pustules

and comedones. Systemic absorption results in nausea, vomiting, loss of weight, jaundice, oedema and abdominal pain. Where the liver damage has been severe, death may follow.

Recommended M.A.C.: The recommended maximum allowable concentration of trichloronaphthalene in air is 50 mgm per 10 cu. metres; where the penta- and hexachloronaphthalenes are used, the M.A.C. recommended is 5 mgm per 10 cu. metres of air, for an 8-hour working day.

Fire Hazard: Non-combustible.

Chlorine

Formula: Cl_2

Properties: A heavy, greenish-yellow gas having an irritating, pungent odour.
Vapour Density: 2·49 times as heavy as air.
Soluble in water and alkalis.
Liquefies under pressure, and is often handled in this form in steel cylinders.

Uses and Occurrence: Chlorine is used as the pure gas or liquid, or combined with calcium or sodium in the form of the hypochlorite or chlorite, as a bleaching and disinfecting agent. It is used in the pulp and textile industries, in the bleaching of flour, in the sterilization of water, and in the manufacture of many organic and inorganic products.

Mode of Entry into Body: Through inhalation of the gas or vapour.

Physiological Action and Toxicity: Chlorine is extremely irritant to the mucous membranes of the eyes and upper respiratory tract. It combines with moisture to liberate nascent oxygen and form hydrochloric acid. Both these substances, if present in quantity, cause inflammation of the tissues with which they come in contact. If the lung tissues are attacked, pulmonary oedema may result. A concentration of 3·5 ppm produces a detectable odour. 15 ppm causes immediate irritation of the throat. Concentrations of 50 ppm are dangerous for even short exposures. 1,000 ppm may be fatal, even where the exposure is brief.

Signs and Symptoms: Because of its intensely irritating properties, severe industrial exposure seldom occurs, as the workman is forced to leave exposure before he can be seriously affected. In cases where this is impossible, the initial irritation of the eyes and mucous membranes of the nose and throat is followed by cough, a feeling of suffocation, and, later, pain and a feeling of constriction in the chest. If exposure has been severe, pulmonary oedema may follow, with râles being heard over the chest.

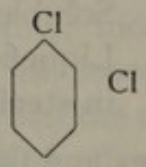
Recommended M.A.C.: The recommended maximum allowable concentration for an 8-hour working day is set by most authorities between 0·5 and 5 ppm. Where the exposure is continuous and repeated, 2 ppm should probably be taken as the M.A.C.

Fire Hazard: None.

Chlorobenzols (Chlorobenzenes)

Formulae:

Chlorobenzol
(Monochlorobenzol)
(Phenyl chloride)



Dichlorobenzol
(Ortho-dichlorobenzol)

Properties: The mono-chlorobenzol is a clear, colourless liquid having an almond-like odour. Specific gravity: 1·11. Boiling point: 270°F. Is slightly volatile at room temperature. Vapour density: 3·88 times heavier than air. The dichlorobenzols exist as three isomers; the meta compound is a colourless liquid boiling at 342°F; the ortho isomer is a colourless liquid boiling at 354°F, and the para compound is a white crystalline solid, melting at 127°F. The dichlorobenzols are only slightly volatile at room temperature. Their vapour density is 5·07 times that of air. The chlorobenzols are insoluble in water, but soluble in most organic solvents.

Uses and Occurrence: The chlorobenzols are used as solvents in the printing industry and in the manufacture of lacquers, shoe dyes, waxes and plastics. The dichlorobenzols are also used as fumigants and insecticides, and in organic synthesis.

Mode of Entry into Body: Through inhalation of the vapour. May be absorbed through the skin.

Physiological action and Toxicity: The monochlorobenzol is a fairly strong narcotic and possesses only slight irritant qualities. Concentrations of 1,200 ppm are quite narcotic for cats, and concentrations of 3,700 ppm are fatal after several hours. The dichlorobenzols are strongly narcotic, 1,000 ppm causing narcosis in guinea pigs followed by death after 20 hours exposure. Knowledge of the effects on man of repeated exposure to sub-narcotic concentrations is meagre. In general, it appears that the chlorobenzols are not as toxic as benzol. Some of the symptoms described (methaemoglobinaemia) suggest that other substances such as nitrobenzol, may have been partially responsible for the few cases of industrial illness reported. It is possible that prolonged exposure to chlorobenzol may cause kidney and liver damage.

Signs and Symptoms: Somnolence, loss of consciousness, twitchings of the extremities, cyanosis, deep, rapid respirations and a small, irregular pulse are the chief symptoms occurring in acute exposures. The urine may be burgundy red, and the red blood cells show degenerative and regenerative changes.

Recommended M.A.C.: A maximum allowable concentration of 50 ppm for repeated 8-hour exposures has been recommended for the ortho-dichlorobenzol. No limits have been set for the other chlorobenzols.

Fire Hazard: The monochlorobenzol has a flash point of 90°F. The ortho and para-dichlorobenzols flash at 150°F.

Chlorobutadiene (Neoprene; Chloroprene; 2-Chloro-1, 3-butadiene)

Formula: $CH_2CClCHCH_2$ or $CH_2 = \underset{Cl}{C} - \underset{H}{C} = CH_2$.

Properties: Colourless liquid, having a pungent odour.
Insoluble in water and in most solvents.
Polymerizes at moderately elevated temperatures to form a solid, neoprene.

Uses: Manufacture of synthetic rubber.

Mode of Entry into Body: As a vapour, through inhalation, and as a liquid, through the skin.

Physiological Action and Toxicity: Animal experiments have shown that a concentration of 250 ppm in air is toxic, and a concentration of 75 ppm may be toxic with continued exposure. Exposure to the vapour first causes irritation of the respiratory tract, followed by depression of respiration and, if exposure is continued, asphyxia. The vapour is a central nervous system depressant; in animals it causes severe degenerative changes in the vital organs, particularly the liver and kidneys. Blood pressure is lowered. Lung changes accompany exposure to the higher concentrations.

Signs and Symptoms: No human cases have been reported, but it would be expected that exposure to the vapour would result in irritation of the nose, throat and lungs, with cough a predominant early symptom. Loss of appetite and indigestion might be found. Blood pressure may be lowered.

Recommended M.A.C.: The recommended maximum allowable concentration has been set at 25 ppm for an 8-hour exposure.

Chromic Acid and the Chromates

The chromium compounds which are of interest toxicologically are chiefly: chromic acid (or chromium trioxide; chromic anhydride; CrO_3), the chromates (*e.g.*, potassium chromate: K_2CrO_4) and the bichromates (*e.g.*, potassium bichromate: $K_2Cr_2O_7$). Chromic acid and the chromates and bichromates are all yellowish or reddish crystals or powders. The water-soluble chromic acid and sodium and potassium chromates and bichromates are used in electroplating. Sodium, potassium and ammonium chromates are used as mordants in dyeing, and in the tanning of leather. The insoluble salts (*e.g.*, lead chromate: $PbCrO_4$) are used as pigments in various industries, such as paint manufacture. Other trades using chromium compounds are: the rubber industry, photography, photo-engraving, dry battery manufacture, pottery makers, etc.

Mode of Entry into Body: Through inhalation of the dust or the acid spray. Acts locally on the skin and mucous membranes.

Physiological Action and Toxicity: The effects of chromic acid and its salts are almost entirely restricted to a localized corrosive action on the skin and mucous membranes. The lesions are confined to the exposed parts, affecting chiefly the skin of the hands and forearms, and the mucous membranes of the nasal septum. The characteristic lesion is a deep, penetrating ulcer, which, for the most part, does not tend to suppurate, and which is slow in healing. An allergic reaction to chromates, resulting in asthma, dermatitis and fever has been described.

Signs and Symptoms: Small ulcers, about the size of a matchhead or end of a lead pencil, may be found, chiefly around the base of the nails, on the knuckles, dorsum of the hands and forearms. These ulcers tend to be clean, and progress slowly. They are frequently painless, even though quite deep. They heal slowly, and leave scars. On the mucous membrane of the nasal septum the ulcers are usually accompanied by purulent discharge and crusting. If exposure continues, perforation of the nasal septum may result, but produces no deformity of the nose.

Recommended M.A.C.: The recommended maximum allowable concentration in air, as chromic acid, is 1 mgm per 10 cu. metres for an 8-hour working day.

Fire Hazard: Chromic acid is an oxidizing material and when in contact with organic material, such as wood, paper or cotton, may cause ignition. Combustible material burns with great intensity when in contact with chromic acid.

Chromium

Symbol: Cr.

Chromium is a hard, steel-gray metal. The metal and its trivalent compounds are not directly toxic to the body. Chromic acid and its salts (in which the chromium is hexavalent) have a local caustic action on the mucous membranes and skin. For further details, refer to "Chromic Acid and the Chromates."

Cobalt and its Compounds

Symbol: Co.

Cobalt is being increasingly used in industry in the production of certain alloys, electromagnets, and as a bonding material in the manufacture of tungsten carbide and other metal carbide tool tips and dies. Cobalt oxide is used as a catalyst for the oxidation of ammonia and for the synthetic production of gasoline.

Mode of Entry into Body: Through inhalation and ingestion of the dust. Acts locally on the skin.

Physiological Action and Toxicity: Experimental evidence shows that the toxicity of cobalt by mouth is low. In animals, administration of cobalt salts produces as polycythaemia. In humans, a single case of poisoning showing liver and kidney damage has been attributed to cobalt. Locally, cobalt has been shown to produce dermatitis. Certain investigators have been able to demonstrate a hypersensitivity of the skin to cobalt.

Recommended M.A.C.: No values have been set.

Fire Hazard: Cobaltous nitrate is an oxidizing material, similar to copper nitrate.

Copper and its Compounds

Symbol: Cu.

Copper and its compounds are widely used in industry. Bronze is a copper alloy containing tin and sometimes zinc. Brass contains copper and zinc. Copper sulphate (blue stone or blue vitriol) is used in the textile and leather trades, in electro-plating, in the manufacture of storage batteries, and as a disinfectant and preservative. Other copper compounds which are widely used are the chloride and the oxide.

Mode of Entry into Body: Through inhalation of the dust or fume. Acts locally on the skin.

Physiological Action and Toxicity: As the sublimed oxide, copper may be responsible for one form of metal fume fever. However, in industrial practice, exposure to copper oxide fumes is nearly always associated with exposure to fumes of zinc or lead oxide. Zinc and lead both give off fumes at

temperatures much lower than does copper, and zinc oxide fumes are believed to be responsible for most cases of metal fume fever. Similarly, lead poisoning is often the true cause of illnesses attributed to copper. Other materials which are frequently associated with exposure to copper are arsenic, antimony and other metals, and "nitrous fumes".

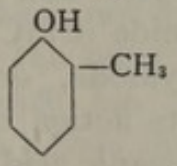
Inhalation of copper dust has caused, in animals, haemolysis of the red blood cells, deposition of haemofuscin in the liver and pancreas, and injury to the lung cells; injection of the dust has caused cirrhosis of the liver and pancreas, and a condition closely resembling haemochromatosis, or bronzed diabetes. However, there is no definite evidence that industrial exposure to copper compounds has resulted in such disease.

As regards local effect, copper chloride and sulphate have been reported as causing irritation of the skin and conjunctivae. Cuprous oxide is irritant to the eyes and upper respiratory tract. Discolouration of the skin is often seen in persons handling copper, but this does not indicate any actual injury from copper.

Recommended M.A.C.: No values have been set.

Fire Hazard: Copper nitrate is an oxidizing material and in contact with organic or other oxidizable substances may cause violent combustion on ignition.

Cresol (Cresylic Acid; Methyl Phenol; Oxytoluene)

Formula: $CH_3C_6H_4OH$ or 

Properties: There are three isomers. The ortho and para isomers are crystalline at ordinary room temperatures; the meta is a clear or yellowish liquid.
Specific Gracity: 1·042.
Melting Points: 51 to 95°F.
Boiling Points: 376 to 395°F.
Vapour Density: 3·72 times heavier than air.
Volatility: Very low at ordinary room temperature.
Soluble in alcohol, ether and chloroform; slightly soluble in water.

Uses and Occurrence: Disinfectant and bactericide; insecticide. Used also in the manufacture of synthetic rubber, resins, dyes, explosives and paint and varnish removers.

Mode of Entry into Body: Acts locally on the skin. Is absorbed through the skin, and may be absorbed through inhalation of the vapour when cresol is heated.

Physiological Action and Toxicity: Cresol is similar to phenol in its action on the body, but it is less severe in its effects. It has a corrosive action on the skin and mucous membranes. Systemic poisoning has rarely been reported, but it is possible that absorption may result in damage to the kidneys, liver and nervous system. The main hazard accompanying its use in industry lies in its action on the skin and mucous membranes, with production of severe chemical burns and dermatitis.

Recommended M.A.C.: No limits have been set.

Fire Hazard: Flash point: 178 to 187°F. No explosive limits are given. It is not a fire hazard at ordinary room temperatures.

Cyanides

The cyanide compounds most widely used in industry are:

1. Sodium cyanide (NaCN) and potassium cyanide (KCN). These salts are used on a large scale in electroplating, case hardening of steel and iron, and refining of gold and silver.
2. Calcium cyanide ($Ca(CN)_2$) is used as a fertilizer.
3. Hydrogen cyanide, or hydrocyanic acid, is used as a fumigant. Its action on the body is discussed separately, under hydrocyanic acid.
4. Cyanogen (NC-CN) and its compounds. Also discussed separately.
5. The organic cyanides, or nitriles, in which a cyanide radicle (–CN) has been introduced into an organic compound; *e.g.*, acetonitrile (CH_3CN), benzonitrile (C_6H_5CN) and acrylonitrile (CH_2CHCN), *q.v.*

Mode of Entry into Body: The non-volatile salts, such as sodium, potassium and calcium cyanides, may act locally on the skin. They are poisonous by ingestion; when heated

or acted upon by acids or moisture, hydrocyanic acid gas is given off and may be inhaled. The volatile cyanides, such as the nitriles and cyanogen compounds, may be inhaled or absorbed through the skin.

Physiological Action and Toxicity: The volatile cyanides resemble hydrocyanic acid physiologically (*q.v.*), inhibiting tissue oxidation and causing death through asphyxia. Cyanogen is probably as toxic as hydrocyanic acid; the nitriles are generally considered somewhat less toxic, probably because of their lower volatility. The non-volatile cyanide salts appear to be relatively non-toxic systemically, so long as they are not ingested and care is taken to prevent the formation of hydrocyanic acid. Workers, such as electroplaters and picklers, who are daily exposed to cyanide solutions, may develop a "cyanide" rash on the exposed parts, characterized by itching, and by macular, papular and vesicular eruptions. Frequently there is secondary infection. Exposure to small amounts of cyanide compounds over long periods of time is reported to cause loss of appetite, headache, weakness, nausea, dizziness, and symptoms of irritation of the upper respiratory tract and eyes.

Recommended M.A.C.: For hydrocyanic acid and acrylonitrile, the maximum allowable concentration is 20 ppm for repeated 8-hour exposures. The allowable concentration of cyanide dust, mist and fume has been recommended as 50 mgm (as CN) per 10 cubic metres of air.

Fire Hazard: The cyanide salts are not flammable; the volatile cyanides are flammable for the most part, and their gases form explosive mixtures with air. Since the minimum explosive concentrations are higher than the concentrations which are dangerous to health, the risk of poisoning is therefore greater than the fire hazard.

Cyanogen and its Compounds

Formula: NC-CN.

Properties: A colourless gas, having a pungent, penetrating odour. Inflammable.

Vapour Density: 1·8 times heavier than air.

Soluble in water, alcohol and ether.

Uses and Occurrence: Cyanogen and its halogen derivatives (cyanogen chloride, CNCl, cyanogen bromide CNBr) are used in organic synthesis and as war gases.

Mode of Entry into Body: Through inhalation of the gas or vapour.

Physiological Action and Toxicity: Cyanogen and its compounds resemble hydrocyanic acid in their action on the body, inhibiting cellular oxidation and causing asphyxia by rendering the oxygen in the blood unavailable to the tissues. The chloride and bromide are, in addition, strongly irritant to the eyes and respiratory tract, causing lacrimation, conjunctivitis, sneezing, coughing and difficulty in breathing. Cyanogen and its compounds may also cause dermatitis of an itchy, papular or vesicular nature.

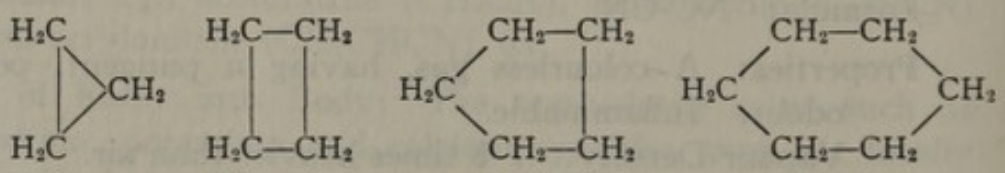
Signs and Symptoms: High concentrations of cyanogen are rapidly fatal; in weaker concentrations there is headache, dizziness, staggering gait, a feeling of suffocation, and nausea. In the case of cyanogen chloride and bromide, these symptoms are usually preceded by signs of irritation of the nose, throat and lungs.

Recommended M.A.C.: No value has been set, but the maximum allowable concentration should probably not be much greater than 20 ppm for an 8-hour working day (the value set for hydrocyanic acid).

Fire Hazard: Cyanogen is flammable and forms explosive mixtures with air. The minimum explosive limit is not listed, but is probably around 3 to 5%, by volume, of the gas in air.

Cycloparaffins

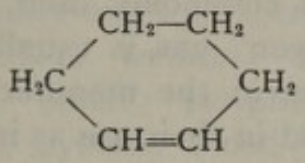
Members of the saturated cycloparaffins, or cycloalkane series of hydrocarbons are characterized by the arrangement of the carbon atoms in a ring, with no unsaturated or double bonds present. Representative members are:



Cyclopropane Cyclobutane Cyclopentane Cyclohexane

By substitution of an alkyl group for one or more hydrogen atoms, alkyl derivatives may be formed, *e.g.*, methyl cyclopropane. Saturated cycloparaffins containing 3 or 4 carbons are gases at ordinary temperatures; those containing 5 or more carbons are colourless liquids having an odour resembling coal oil. The latter are used chiefly as degreasing agents and as solvents.

Where a double bond exists in the carbon ring, the cycloparaffin is unsaturated, and is a member of the cycloalkene series. Thus, cyclohexene has the formula:



Physiological Action and Toxicity: Both the saturated and unsaturated members of the cycloparaffin series are narcotic, and may cause death through respiratory paralysis. For most of the members there appears to be little range between the concentrations causing deep narcosis and those causing death. There is very little information in the literature regarding the chronic effects resulting from exposure of humans to the cycloparaffins. Experimental work with rabbits indicate that barely demonstrable changes in the liver and kidneys may result from exposure to 786 ppm of cyclohexane for 6 hours daily, repeated for 50 days.

Recommended M.A.C.'s: Recommended maximum allowable concentrations have been suggested for certain of the cycloparaffins, as follows: Cyclohexane: 400 ppm for an 8-hour working day. Cyclohexene: 400 ppm for similar exposures. Methyl cyclohexane: 500 ppm for similar exposures.

Fire Hazards: All members of the cycloparaffin series are flammable. Cyclohexane flashes at 1°F, and has a minimum explosive limit of 1·3% and a maximum explosive limit of 8·35%, by volume, of the vapour in air. Methyl cyclohexane flashes at 25°F, and has a similar minimum explosive limit.

D.D.T. (Dichloro-Diphenyl-Trichloroethane)

Properties: D.D.T. is a white, crystalline powder. When pure it is tasteless and nearly odourless. It is nearly insoluble in water, but is quite soluble in oils and in certain organic solvents such as cyclohexanone, benzol, toluol, xylol and acetone.

Uses and Occurrence: D.D.T. is used as an insecticide in the form of solutions, dusting powders, or as aerosol sprays. The D.D.T. content of the various commercial preparations varies from 1% to 25%, with 3%, 5% and 10% preparations being the most commonly used. In the aerosol "bomb", compressed "freon" gas is usually used as the propellant. Exposure occurs in the manufacture and handling of the preparations and in their use as insecticides.

Mode of Entry into Body: Through inhalation of the dust or spray, through ingestion of contaminated foods, and through absorption of D.D.T. in non-aqueous solutions through the skin.

Physiological Action and Toxicity: Experimental work in animals indicates that D.D.T. may cause systemic poisoning and even death when administered under certain conditions. Fatal poisoning results in focal necrosis of the liver and degenerative changes in the kidneys and in the cells of the anterior horns of the spinal cord and the cerebral motor nuclei. Solutions of the material in oil or organic solvents are much more toxic than the dry powder or aqueous suspensions. The use of 10% D.D.T. powders on humans as a delousing agent has produced no systemic effects or evidence of irritation of the skin. No cases of human poisoning have resulted from the use of 5% or 10% D.D.T. aerosol sprays and solutions when these materials were employed as insecticides according to the directions given for their use.

The risk of poisoning following ingestion of contaminated food is very small. One man ingested 770 mgm of D.D.T. in olive oil without apparent ill effect. The few cases of poisoning reported in the literature were the result of absorption of D.D.T. in oily or acetone solution through the skin, or of ingestion. D.D.T. is excreted slowly in the urine and in milk, and repeated exposure may result in a cumulative action.

Signs and Symptoms: Following ingestion of oily solutions of D.D.T., nausea, vomiting, stiffness in the jaws and mental anxiety may result. In a child of 19 months who drank 1 ounce of a 5% kerosene solution, coma and convulsions developed after 90 minutes, and within four hours the child was dead. Autopsy revealed oedema of the brain and lungs and enlargement of the spleen. Experiments with monkeys indicated that the D.D.T. in kerosene was responsible, kerosene alone having no such effect. In 3 reported cases of illness, absorption of D.D.T. through the skin resulted in symptoms of lassitude, heaviness and aching in the limbs, irritability and apprehension, pains in the joints, involuntary muscular tremors, diminished reflexes and auditory acuity, patchy peripheral anaesthesia, yellow vision, decreased systolic blood pressure and decreased red and white cell counts. In two of the cases, there was no evidence of illness after 33 days. In the third case, recovery was slow and the patient lost 10 weeks from work. At the end of a year, recovery was stated as not quite complete.

Contact of the skin with kerosene solutions of D.D.T. has caused dermatitis which in most instances has been regarded as due to the action of the kerosene.

Recommended M.A.C.: No values have been suggested. The greatest risk of poisoning occurs in persons handling the concentrated solutions. Precautions should be taken in such cases to avoid skin contact.

Dichloroethyl Ether

Formula: CH_2ClCH_2-O-CH_2CH_2Cl.

Properties: A colourless liquid having a pungent odour. Specific gravity: 1·22. Boiling point: 352°F. Vapour density: 4·93 times heavier than air. Insoluble in water but soluble in most organic solvents.

Uses and Occurrence: Is used as a solvent in the textile industry and in the extraction of waxes from lubricating oils. Used in the manufacture of organic chemicals.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: The vapour is irritant to the mucous membranes of the eyes and nose. It affects the kidneys and liver in varying degrees, and is a mild narcotic. Guinea pigs cannot be killed immediately by exposure to concentrations which can be attained at ordinary room temperature, but exposure to 1,000 ppm for 30 to 60 minutes may produce death after several days. Autopsy shows congestion of the lungs and upper respiratory tract, pulmonary oedema, and congestion of the liver, brain and kidneys. The pulmonary oedema apparently develops after a latent period of several hours, similar to the action of "nitrous fumes". In humans, exposure to 500 to 1,000 ppm causes severe irritation of the eyes and nose after brief exposure, and deep inhalation is nauseating and intolerable. A concentration of 100 ppm produces slight nausea and irritation; concentrations of 35 ppm are practically free from irritation, though the odour is easily detectable. No cases of industrial poisoning have been reported.

Signs and Symptoms: Irritation of the eyes and nose, nausea, dizziness, retching, slow respiration which gradually becomes shallow and rapid, and unconsciousness. Pulmonary oedema may develop after a latent period of some hours.

Recommended M.A.C.: A limit of 15 ppm has been suggested for repeated 8-hour exposures.

Fire Hazard: Flash point: 131°F.

2,4-D (2,4-Dichlorophenoxy Acetic Acid)

2,4-D is a selective weed killer. In certain concentrations it is toxic to broad leaved plants but has little or no effect upon narrow leaved grasses, grains and sugar cane. It is only slightly soluble in water and oils, and is usually used in the form of water-soluble sodium or ammonium salts, either in solution, or as the dry dusting powder, or as an emulsion of the oil-soluble esters. It is absorbed through the epidermal cells of the leaves, from which it is transported to various parts of the plant.

Workers using 2,4-D preparations are exposed to the dust of the sodium or ammonium salts and to the mist from

tributyl phosphate-diesel oil emulsions. Poisonous impurities may be present in commercial preparations.

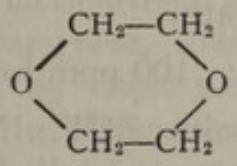
Mode of Entry into Body: Through inhalation and ingestion. Soluble preparations may be absorbed through the skin. Ingestion of 2,4-D may occur following the eating of treated plants.

Physiological Action and Toxicity: Experimental work on ingestion has shown that the amounts of 2,4-D required to kill 50% of experimental animals are: For mice, 375 mgm per kilogram of weight; for rats, 660 mgm per kg; for rabbits, 800 mgm per kg; and for guinea pigs, 1,000 mgm per kg. No toxic effects on humans have been reported from its use as a weed-killer, and no ill effects have been discovered in animals eating pasture grass liberally sprayed with 2,4-D. Experimental work indicates that mist or dust of the sodium salt is relatively non-toxic. The ingestion of 500 mgm daily by a man for 21 days had no apparent ill effect.

Recommended M.A.C.: No values have been suggested.

Diethylene Dioxide (Dioxan)

Formula:



Properties: A colourless liquid having a faint ether-like odour.
Specific Gravity: 1·035 times heavier than water.
Boiling Point: 214°F.
Vapour Density: 3·03 times heavier than air.
Volatility: 4·3 times less volatile than ether.
Soluble in water, most organic solvents and in fatty oils.

Uses and Occurrence: Solvent for cellulose acetate, nitrocellulose, oils, fats, resins and pigments. Used in the manufacture of lacquers, paints and varnishes, paint and varnish removers, stains, printing inks, cements and glues, polishes and pastes, and cleaning preparations. Is also employed in the defatting of wool and in the textile industry.

Mode of Entry into Body: Through inhalation of the vapour, May be absorbed through the skin.

Physiological Action and Toxicity: Exposure of animals to concentrations of 0·1 to 3% of dioxan vapour causes irritation of the eyes and nose, followed by narcosis and/or pulmonary oedema and death. The irritative effects probably provide sufficient warning, in acute exposures, to enable the workman to leave exposure before he is seriously affected. On the other hand, repeated exposure to low concentrations has resulted in human fatalities, the organs chiefly affected being the liver and kidneys. Death resulted from acute haemorrhagic nephritis. The hepatic lesion consists of an acute central necrosis of the lobules. The brain and lungs may show acute oedema.

Signs and Symptoms: In acute exposures, the signs and symptoms consist of irritation of the eyes and naso-pharynx, which may later subside, to be followed by headache, drowsiness, dizziness, and occasionally nausea and vomiting. In chronic exposures, there may be loss of appetite, nausea and vomiting, pain and tenderness in the abdomen and lumbar region, malaise, and enlargement of the liver without jaundice. There may be changes in the blood picture. Further exposure may result in suppression of urine, followed by uraemia and death.

Recommended M.A.C.: 100 ppm for an 8-hour working day.

Fire Hazard: Flash point: 65°F. Minimum explosive limit is 1·9%. Maximum explosive limit 22·2%, by volume, of the vapour in air. Class 2 flammable liquid.

Dimethyl Sulphate (Methyl Sulphate)

Formula: $(CH_3)_2SO_4$.

Properties: A colourless, oily liquid having a faint, mint-like odour.

Specific Gravity: 1·33 times heavier than water.

Boiling Point: 370°F. At 122°F, grayish vapour is given off the liquid.

Is slightly volatile at ordinary room temperatures.

Vapour Density: 4·35 times heavier than air.

Practically insoluble in water, but easily soluble in organic solvents.

Uses and Occurrence: Used in organic synthesis and in the manufacture of dyes, colours and war gas.

Mode of Entry into Body: Through inhalation of the vapour. Acts locally on the skin.

Physiological Action and Toxicity: Contact of the skin and mucous membranes with the liquid or vapour, even for short periods, results in intense irritation of these tissues after a period of several hours. There is no odour or initial irritation to give warning of exposure. On brief, mild exposures, conjunctivitis, catarrhal inflammation of the mucous membranes of the nose, throat, larynx and trachea and possibly some reddening of the skin develop after the latent period. With longer, heavier exposures, the cornea shows clouding, the irritative changes of the naso-pharynx are more marked, and after 6 to 8 hours pulmonary oedema may develop. Death may occur in 3 or 4 days. The liver and kidneys are frequently damaged. Spilling of the liquid on the skin can cause ulceration and local necrosis. The fatal concentration for cats and monkeys is in the range of 25 to 200 ppm. of the vapour in air.

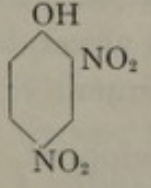
Signs and Symptoms: After a latent period of several hours, there is severe lacrimation, conjunctivitis, photophobia, coughing and hoarseness, followed, in the case of more severe exposures, by chest pain, dyspnoea, cyanosis and possibly death. In patients surviving severe exposures, there may be serious injury to the liver and kidneys, with suppression of urine, jaundice, albuminuria and haematuria appearing. Death may be delayed for several weeks, and results from the kidney or liver damage.

Recommended M.A.C.: 1 ppm for an 8-hour working day.

Fire Hazard: Flash point: 182°F.

Dinitrophenol

Formulae: There are 6 isomers of dinitrophenol; the most important is the 1,2,4-dinitrophenol, which has the formula



. All 6 isomers occur as crystalline solids, the

1,2,4- isomer having a melting point of 239°F. All are soluble in ether. Their solubility in water and alcohol vary.

Uses: Military explosive. Used in the manufacture of dyes and picric acid.

Mode of Entry into Body: Chiefly by absorption through the skin; also by ingestion and inhalation of the dust or fumes.

Physiological Action and Toxicity: Dinitrophenol and dinitrocresol are powerful metabolic stimulants. According to French investigators, the mechanism of intoxication consists of an enormous increase (up to 1200%) in the intracellular oxidative processes, with consequent increase in oxygen metabolism and a rise of body temperature. The basal metabolic rate may be raised to more than 400. There is rapid pulse and respiration, profuse sweating, severe thirst, shortness of breath and progressive loss of weight. Symptoms of tinnitus, vertigo, flickering in front of the eyes, restlessness, irritability and fright may be present. The face is livid and often cyanotic and the skin is frequently yellow. Death may be preceded by unconsciousness. There are no characteristic pathological changes, though the liver and kidneys may show evidence of injury. In less severe poisoning, the symptoms are mainly referable to the gastro-intestinal tract. Dinitrophenol and dinistrocresol are not believed to cause methaemoglobinaemia.

Signs and Symptoms: In subacute poisoning, the symptoms most commonly encountered are anorexia, white furred tongue, nausea and vomiting. Some patients have diarrhoea and colicky abdominal pain. The liver may be sensitive to pressure and some jaundice may be present. There is general weakness, fatigue, dizziness, headache and profuse sweating. Excretion products of the nitro compound can usually be found in the urine. In acute cases, the onset is sudden and the symptoms include those described in the paragraph above. Dinitrophenol may cause contact dermatitis. Its therapeutic use as a reducing agent has been followed by the development of cataract, frequently bilateral.

Recommended M.A.C.: No limits have been adopted.

Diphenyl

Formula: $C_6H_5–C_6H_5$ or 

Properties: Occurs as white scales having a pleasant odour.
Specific Gravity: 0·984 times as heavy as water.
Melting Point: 158°F.
Boiling Point: 491°F.
Vapour Density: 5·3 times heavier than air.
Soluble in alcohol and ether; insoluble in water.

Uses and Occurrence: Diphenyl and its amino and nitro derivatives are used as constituents of various plastics and resins.

Mode of Entry into Body: Through inhalation of the dust, or, when heated, of the vapour. Can probably be absorbed through the skin, to some extent.

Physiological Action and Toxicity: Though no trouble has been reported from the use of diphenyl or its amino and nitro derivatives in industry, animal experiments indicate that these substances, when absorbed through the lungs or skin, may produce severe broncho-pulmonary lesions and minor toxic changes in the kidneys and liver. Repeated application of the materials to the skin did not produce any signs of irritation. The maximum safe concentration of dusts of diphenyl or its amino or nitro derivatives in air, for rats, mice and rabbits is between 3 and 300 mgm per cubic metre of air, the ortho-nitro diphenyl appearing to be the most toxic.

Recommended M.A.C.: No value has yet been set.

Fire Hazard: Flash point: 235°F.

Ethyl Acetate (Acetic Ether; Vinegar Naphtha)

Formula: $CH_3COOC_2H_5$

Properties: A colourless liquid having an aromatic odour slightly like that of vinegar.
Specific Gravity: 0·899 times as heavy as water.
Boiling Point: 171°F.
Vapour Density: 3·04 times heavier than air.
Volatility: about 3 times less volatile than ethyl ether.

Soluble in chloroform, alcohol and ether. Slightly soluble in water.

Uses and Occurrence: Is an excellent solvent, and is widely used in industry, particularly in the manufacture of paints, varnishes, and lacquers, plastics, drugs, liquid cements, etc.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Is less irritating to the mucous membranes than are the other acetate esters. It has a narcotic action somewhat stronger than that of methyl acetate. A concentration of 12,000 ppm is narcotic for cats.

Signs and Symptoms: Inflammation of the gums has been reported. Apparently, irritation of the conjunctivae is rare. Workers exposed to ethyl acetate may develop exzematous skin conditions.

Recommended M.A.C.: 400 ppm for an 8-hour working day.

Fire Hazard: Dangerous. Flash point is 24°F. Minimum explosive limit is 2·2%, maximum explosive limit 11·5% by volume, of the vapour in air. Class 1 flammable liquid.

Ethyl Alcohol (Ethanol: Cologne Spirits; Grain Alcohol; Ethyl Hydroxide)

Formula: CH_3CH_2OH

Properties: A clear colourless liquid having a characteristic odour. Inflammable.
Specific Gravity: 0·79 times as heavy as water.
Boiling Point: 173°F.
Vapour Density: 1·59 times heavier than air.
Volatility: 8·3 times less volatile than ether.
Soluble in water, methyl alcohol and ether.

Uses and Occurrence: Denatured ethyl alcohol is very widely used in industry as a solvent. The pure alcohol is employed as the starting point in the manufacture of many compounds.

Mode of Entry into Body: Through inhalation of the vapour. Ethyl alcohol can be absorbed through the skin, but this route is of little importance in industrial poisoning.

Physiological Action and Toxicity: The systemic effect of ethyl alcohol differs from that of methyl alcohol. Ethyl alcohol is rapidly oxidized in the body to carbon dioxide and water, and in contrast to methyl alcohol, no cumulative effect occurs. Though ethyl alcohol possesses narcotic properties, concentrations sufficient to produce this effect are not reached in industry. Exposure to concentrations of 5,000 to 10,000 ppm result in irritation of the eyes and mucous membranes of the upper respiratory tract. If continued for an hour, stupor and drowsiness may result. Concentrations below 1,000 ppm usually produce no signs of intoxication. There is no concrete evidence that repeated exposure to ethyl alcohol vapour results in cirrhosis of the liver. The main effect of ethyl alcohol is due to its irritant action on the mucous membranes of the eyes and upper respiratory tract.

Signs and Symptoms: Exposure to concentrations of over 1,000 ppm may cause headache, irritation of the eyes, nose and throat, and, if long continued, drowsiness and lassitude, loss of appetite and inability to concentrate.

Recommended M.A.C.: 1,000 ppm for an 8-hour working day.

Fire Hazard: Dangerous; flash point, 55°F. Minimum explosive limit is 3·2%. Maximum explosive limit 19%, by volume, of the vapour in air. Is a class 2 flammable liquid. Where alcohol-water mixtures are used, the fire hazard is lessened, since the flash point is raised to 72°F to 144°F.

Ethyl Benzene (Ethyl Benzol; Phenyl Ethane)

Formula: $C_6H_5C_2H_5$ or 

Properties: A colourless, clear liquid, having a specific gravity of 0·867.

Boiling Point: 277°F.

Vapour Density: 3·66 times heavier than air.

Soluble in alcohol, ether, benzol and carbon tetrachloride; is practically insoluble in water.

Uses and Occurrence: Is used as a solvent and lacquer thinner; may be blended with gasoline as an "anti-knock" agent.

Mode of Entry into Body: Through inhalation of the vapour; acts locally on the skin, and is absorbed through it.

Physiological Action and Toxicity: The liquid is irritant to the skin and mucous membranes. A concentration of 0·1% of the vapour in air is irritant to the eyes of humans, and a concentration of 0·2% is extremely irritant at first, then causes dizziness, irritation of the nose and throat and a sense of constriction of the chest. Exposure of guinea pigs to 1% concentrations has been reported as causing ataxia, loss of consciousness, tremor of the extremities and finally death through respiratory failure. The pathological findings were congestion of the brain and lungs, with oedema. No data are available regarding the effect of chronic exposure.

Signs and Symptoms: Erythema and inflammation of the skin may result from contact of the skin with the liquid. Exposure to the vapour causes lacrimation and irritation of the nose and throat, dizziness, and a sense of constriction of the chest. The irritant properties are sufficient to cause workers to leave an atmosphere containing 0·5% of the vapour.

Recommended M.A.C.: 200 ppm for an 8-hour working day.

Fire Hazard: Flash point: 59°F. No explosive limits are given. Class 2 flammable liquid.

Ethyl Silicate (Tetraethyl-ortho-silicate)

Formula: $(CH_3CH_2)_4\ Si\ O_4$

Properties: A clear, colourless liquid having a faint, ethereal odour.

Specific gravity: 0·9356.

Boiling point: 335°F.

Vapour density: 7·2 times heavier than air. Not readily volatile at ordinary room temperatures. Hydrolyzed by water to form silicic acid. Soluble in alcohol.

Uses and Occurrence: Employed as a water-proofing agent for stone and concrete, as a solvent and vehicle in heat and chemical resistant paints and lacquers, and as a bonding agent.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Ethyl silicate is irritant to the eyes and upper respiratory tract. Concentrations of 2,500 ppm produce narcosis in guinea pigs after 1½ hours exposure. This is the maximum concentration obtainable at ordinary room temperatures by evaporation. The maximum concentration which these animals can inhale for 60 minutes without the production of serious disturbances is 2,000 ppm. For several hours exposure, concentrations of 500 ppm or more are required to cause serious injury. Animals dying after acute exposures show pulmonary oedema, sometimes with secondary pneumonia, acute nephritis and evidence of injury to the liver. For humans, concentrations of 85 ppm produce a detectable odour, and 700 ppm cause stinging of the eyes and nose. No cases of human poisoning have been reported nor have experiments dealing with the effects of prolonged exposure been reported.

Signs and Symptoms: Irritation of the eyes and nose, lachrymation, tremors, respiratory difficulty or irregularity, anaemia, leucocytosis, and narcosis have been reported in animal experiments. Brief exposures of humans to 3,000 ppm cause only extreme irritation of the eyes and nose.

Recommended M.A.C.: On the basis of animal experiments, it has been suggested that the maximum allowable concentration for repeated 8-hour exposures be 100 ppm.

Fire Hazard: Flash point: 125°F. Because of its low volatility, it is doubtful if explosive concentrations can be formed at room temperature.

Ethylene Chlorohydrin (2-Chloroethyl Alcohol; Glycol Chlorohydrin)

Formula: CH_2ClCH_2OH.

Properties: A clear, oily fluid having an odour resembling alcohol.

Specific Gravity: 1·21 times heavier than water.

Boiling Point: 264°F.

Vapour Density: 2·78 times heavier than air.

Soluble in water, alcohol, gasoline and most organic liquids.

When heated to 212°F with water, it decomposes into glycol and hydrochloric acid. When heated alone to 363°F, it decomposes into ethylene chloride and acetaldehyde.

Uses and Occurrence: Solvent for cellulose acetate; is used in the synthesis of drugs and dyes, in the manufacture of lacquers and varnish, and in the oilcloth industry.

Mode of Entry into Body: Through inhalation of the vapour and by absorption through the skin.

Physiological Action and Toxicity: Ethylene chlorohydrin is a narcotic poison affecting the nervous system and the liver, spleen and lungs. Exposure to the vapour may result in irritation of the mucous membranes, followed by sleepiness, drowsiness and giddiness, nausea and vomiting. The initial symptoms may be slight. After a latent period of several hours dyspnoea, severe headache, stupor, cyanosis and pain over the heart may develop. Autopsy shows pulmonary oedema, ulceration of the mucous membranes of the larger bronchi, and acute liver and kidney lesions. Fatal amounts of ethylene chlorohydrin may be absorbed through the skin.

Recommended M.A.C.: 5 ppm. for an 8-hour working day.

Fire Hazard: Ethylene chlorohydrin has a flash point of 140°F. Class 3 flammable liquid.

Ethylene Dichloride (1, 2-Dichloroethane; Dutch Liquid; Dutch Oil; Elayl Chloride; sometimes called Ethylene Chloride)

Formula: $CH_2Cl\text{-}CH_2Cl$

Properties: A colourless, oily liquid having a chloroform-like odour. Inflammable. Specific Gravity: 1·257. Boils at 182°F. Is four times less volatile than ether. The vapour is 3·3 times heavier than air. Solubility: Is miscible with alcohol, ether, and most organic solvents, but is practically insoluble in water.

Uses and Occurrence: Is an excellent solvent for fats, oils and waxes, rubber, resins, gums, etc. Used chiefly in dry-cleaning of textiles, as a thinner in lacquers, in the production of photographic film, and in the purification of oils.

Mode of Entry into Body: Through inhalation of the vapour; possibly by skin absorption.

Physiological Effect and Toxicity: Ethylene dichloride has a distinctive odour and strong local irritating effects, which give warning of its presence in relatively safe concentrations. There is irritation of the eyes and upper respiratory passages. Ethylene dichloride has a specific effect on the cornea. Exposure to the vapour, or, in animals, injection under the skin, produces a clouding which may progress to endothelial necrosis and infiltration of the cornea by lymphocytes and connective tissue cells. The narcotic action of the compound is strong, probably of the same order as chloroform. Its toxic effects upon the liver and kidneys are less than that of carbon tetrachloride, but animal experiments indicate that these organs may show congestion and fatty degeneration. Oedema of the lungs has also been reported in animals. Dermatitis in man has been observed.

Signs and Symptoms: In short exposures to high concentrations, the picture is one of irritation of the eyes, nose and throat, followed by dizziness, nausea, vomiting, increasing stupor, cyanosis, rapid pulse, and loss of consciousness.

Chronic poisoning, where the exposure has occurred over a period of several months, may cause loss of appetite, nausea and vomiting, epigastric distress, tremors, nystagmus, leucocytosis, low blood sugar levels, and possibly dermatitis if there has been skin contact.

Recommended M.A.C.: 75 to 100 ppm for an 8-hour working period.

Fire Hazard: Ethylene dichloride has a flash point of 65°F; its minimum explosive limit is 6·2%, its maximum explosive limit is 15·9%. Class 2 flammable liquid.

Ethylene Glycol Monoethyl Ether ("Cellosolve")

Formula: $\begin{matrix} CH_2OC_2H_5 \\ | \\ CH_2OH \end{matrix}$

Properties: A colourless, nearly odourless liquid.
Specific Gravity: 0·93 times as heavy as water.
Boiling Point: 275°F.
Vapour Density: 3·1 times heavier than air.

Volatility: 43 times less volatile than ether, at ordinary room temperatures.
Soluble in water, alcohol, aromatic hydrocarbons and fatty oils.

Uses and Occurrence: Used as a solvent for nitrocellulose, natural and synthetic resins. Is used in the manufacture of lacquers and lacquer thinners, varnish removers, and cleaning solutions. Exposure may occur in spray-painting.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Animal experiments indicate that exposure to air saturated with cellosolve vapour (0·6%) for periods of 18 to 24 hours may produce congestion and oedema of the lungs and congestion of the kidneys. Exposure of humans to the same concentration for a few seconds resulted in irritation of the eyes. No cases of poisoning have so far been reported in industry.

Recommended M.A.C.: 200 ppm for an 8-hour working day. For "Cellosolve" acetate a figure of 100 ppm has been recommended.

Fire Hazard: Flash point: 104°F. Minimum explosive limit is 2·6%; maximum explosive limit 15·7%, by volume, of the vapour in air.

Ethylene Glycol Monomethyl Ether ("Methyl Cellosolve").

Formula: $\begin{matrix} CH_2OCH_3 \\ | \\ CH_2OH \end{matrix}$

Properties: A colourless liquid having a faint alcoholic odour.
Specific Gravity: 0·966 times as heavy as water.
Boiling Point: 255°F.
Vapour Density: 2·62 times heavier than air.
Volatility: About 35 times less volatile than ether.
Soluble in water, alcohol and the aromatic hydrocarbons.

Uses and Occurrence: Solvent for nitrocellulose and cellulose acetate, natural and synthetic resins. Is used in the manufacture of lacquers, enamels, varnishes, stains, and transparent wrapping materials. Used as a solvent for the stiffening material with which "fused" or "trubenized" shirt collars are impregnated.

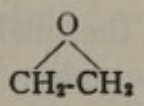
Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: When used under conditions which do not require the application of heat, methyl cellosolve probably presents little hazard to health. However, in the manufacture of fused collars, which require pressing with a hot iron, cases have been reported showing disturbance of haemopoietic system with or without neurological signs and symptoms. The blood picture may resemble that produced by exposure to benzol. Two cases reported had severe aplastic anaemia with tremors and marked mental dullness. One case had multiple neuritis and four others had abnormal reflexes. The commonest change in the blood picture was the finding of immature neutrophils (shift to the left); in other cases there were a reduction in number of the blood platelets and a macrocytic anaemia. The persons affected had been exposed to vapours of methyl cellosolve (76 ppm), ethyl and methyl alcohol, ethyl acetate and petroleum naphtha.

Signs and Symptoms: The first signs of poisoning are probably abnormalities found in the blood picture, as mentioned above. Reflexes may be exaggerated or abnormal in character, and may be accompanied by complaints of drowsiness and fatigue. Tremors may be present. Severe damage probably takes the form of an aplastic anaemia.

Recommended M.A.C.: 25 ppm for an 8-hour working day.
Fire Hazard: Flash point: 107°F.

Ethylene Oxide

Formula: 

Properties: A colourless gas at ordinary room temperature, liquefying at 51°F. Inflammable. Vapour density: 1·52 times heavier than air. Soluble in most organic solvents and miscible with water in all proportions.

Uses and Occurrence: Used as a fumigant and in organic synthesis.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Ethylene oxide is irritant to the eyes and to the mucous membranes of the upper respiratory tract. It possesses mild narcotic action. Exposure of guinea pigs to concentrations of 250 ppm for 8 hours produced no symptoms or appreciable harmful effect. In dogs and cats, higher concentrations produced symptoms of irritation of the respiratory tract, narcosis and death. Haemorrhages were found in the brain, and blood pigment in the spleen and liver. Pulmonary oedema has also been reported.

Signs and Symptoms: Headache, nausea and vomiting, irritation of the eyes, lachrymation, dizziness, cough, dyspnoea, irritation of the nose, and on exposure to high concentrations, signs of pulmonary oedema.

Recommended M.A.C.: 100 ppm for repeated 8-hour exposures.

Fire Hazard: The minimum explosive limit is 3%, the maximum explosive limit 80% by volume, of the vapour in air. Flash point is below 20°F.

Fluorine and the Fluorides

Formula: F_2

Properties: A colourless, corrosive gas. Vapour density: 1·14 times as heavy as air. Insoluble in water.

Uses and Occurrence: Fluorine is little used in industry, though small amounts are employed in organic synthesis. Hydrofluoric acid and certain other fluorides are fairly widely used. (Refer to "Hydrofluoric Acid".)

Exposure to fluorides occurs in the smelting of steel and in the recovery of aluminum from cryolite (a natural fluoride of sodium and aluminum). Recently there has been evidence that the fumes produced by the electrolysis of molten fluorides containing beryllium fluoride or oxyfluoride are extremely toxic, the effect being chiefly on the lungs. This question is discussed under "Beryllium and its Compounds".

Mode of Entry into Body: Through inhalation of the dust or smoke.

Physiological Action and Toxicity: Acute effects resulting from exposure to fluorine compounds are due to hydrogen fluoride, and are discussed under "Hydrofluoric Acid". Chronic fluorine poisoning, or "fluorosis" occurs in miners of cryolite, and consists of a sclerosis of the bones, caused by fixation of the calcium by the fluorine. There may also be some calcification of the ligaments. The teeth are mottled, and there is osteosclerosis and osteomalacia. The bony and ligamentous changes are demonstrable of X-ray.

Signs and Symptoms: Loss of weight, anorexia, anaemia, wasting and cachexia, and dental defects are among the common findings in chronic fluorine poisoning. There may be an eosinophilia, and impairment of growth in young workers.

Recommended M.A.C.: There is little information in the literature regarding toxic limits for the fluorides. In the United States, Massachusetts uses the figure 1 mgm per cubic metre of air as a guide; Oregon has set a limit of 2 mgm/cu. metre of air as the maximum concentration of fluoride dust or smoke which is regarded as safe.

Formaldehyde: (Adorin; Aldoform; Festoform; Formalin; Formalith; Formic Aldehyde; Formitrol; Formol; Formolyptol; Methanal; Methyl Aldehyde; Oxymethylene; Preservaline)

Formula: HCHO.

Properties: Colourless gas, having a pungent, suffocating odour. The vapour density of the gas is 0·815.
Boiling Point: −6°F.

Soluble in water and alcohol. Fairly soluble in ether. Polymerizes to form paraformaldehyde (polyoxymethylene, or formic paraldehyde (HCHO)x), at temperatures above 68°F.

Formaldehyde is widely used in industry as the aqueous solution, the usual strength being 37% formaldehyde by weight (approx. 40% by vol.). Such solutions may contain up to 15% of methyl alcohol (to prevent polymerization) plus traces of acetic and formic acids. The commercial 37% solution has a specific gravity of 1·075 and boils between

200 and 215°F, depending upon its content of methyl alcohol. The density of the vapour from the commercial solution is 1·03.

Mode of Entry into Body: As a vapour, through inhalation. Acts locally on the skin.

Physiological Action and Toxicity: Formaldehyde acts upon proteins to form irreversible compounds; its action is therefore chiefly local. Some formaldehyde, in contact with living tissue, is thought to be changed into formic acid and methyl alcohol. However, the symptoms which follow inhalation are due to the intense local irritation, the decomposition products playing a negligible role in the case of acute poisoning.

Exposure to the vapour causes intense irritation of the mucous membranes of the eyes and respiratory tract, and of the skin. Because of its irritant action on the upper respiratory tract, exposure in humans is seldom long enough to produce irritation of the lungs. Heavy exposures, however, cause irritation of the lower respiratory tract. Fatal formaldehyde poisonings have almost always been the result of ingestion of the liquid (ingestion of 60 cc of the commercial solution has been followed by recovery; ingestion of 90 cc has resulted in death), such cases exhibiting gastro-intestinal, cerebral, renal, and/or pulmonary damage. An occasional death from massive exposure to the gas has been reported as due to spasm of the larynx. Ordinarily, there is complete recovery from brief exposures to concentrations below 500 ppm.

Signs and Symptoms: Exposure to low concentrations of the vapour causes weeping and burning of the eyes, irritation of the upper air passages. Heavier exposures cause cough, tightness of the chest, a sense of tightness in the head, loss of appetite, sleeplessness, general weakness and palpitations. As used industrially, the commonest effect of formaldehyde is on the skin, causing a moist eczema. Certain individuals may acquire a marked hypersensitivity. Chronic conjunctivitis, rhinitis, and bronchitis may be aggravated by exposure to formaldehyde. Its use can also result in the fingernails becoming soft, brown and fibrous.

Recommended M.A.C.: The maximum allowable concentration for an 8-hour exposure is considered to be 20 ppm to 10 ppm, the latter figure being now favoured by most authorities. These concentrations usually are sufficient to cause noticeable irritation in most persons, and 50 ppm is said to be unendurable to breathe.

Fire Hazard: The 37% formaldehyde solution has a flash point of 187° to 215°F, depending on methyl alcohol content, and at ordinary temperatures does not constitute a fire hazard.

Formic Acid

Formula: HCOOH

Properties: A colourless, fuming liquid having a pungent, penetrating odour. Specific gravity: 1·2178 times heavier than water. Solidifies below 46°F., and boils at 213°F. Soluble in water, alcohol and ether.

Uses and Occurrence: Is used in the manufacture of organic esters, insecticides, fumigants, refrigerants and other products. Is employed in the dyeing and finishing of textiles, electroplating, silvering of glass, coagulation of rubber and other processes.

Mode of Entry into Body: Through inhalation of the vapour or spray. The liquid acts locally on the skin.

Physiological Action and Toxicity: The vapour is intensely irritant to the eyes and upper respiratory tract, causing lachrymation, coughing, soreness of the throat and difficulty in breathing. The warning properties are effective and workers can seldom remain in harmful concentrations. Contact of the liquid or spray with the skin may cause dermatitis and chemical burns.

Recommended M.A.C.: No values have been adopted.

Freon

The term "Freon" is a trade name which is applied to methane and ethane derivatives which contain both chlorine and fluorine and which are used as refrigerants in air-conditioning and refrigerating systems. There are at least six products on the market at present. The table below lists their chemical composition, physical properties, and toxicity.

The "Freons"

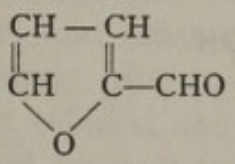
Name	*Formula*	*Ordinary State*	*Boiling Point °F.*	*Vapour Density*	*Relative Toxicity*
1. Monofluoro-trichloro-methane (Freon-11; F-11)	CCl_3F	Colourless Liquid.	74·7	4·7	3+
2. Dichloro-difluoro-methane (Freon-12; F-12)	CCl_2F_2	Gas	−20·6	4·2	+
3. Dichloro-monofluoro-methane (Freon-21; F-21)	$CHCl_2F$	Gas	48·	3·5	4+
4. Difluoro-monochloro-methane (Freon-22; F-22)	$CHClF_2$	Gas	−41·8	3·0	2+
5. Trichloro-trifluoro-ethane (Freon-113; F-113)	$C_2Cl_3F_3$	Liquid	118·	6·7	4+
6. Dichloro-tetrafluoro-ethane (Freon-114; F-114)	$C_2Cl_2F_4$	Gas	39·1	5·9	+

Toxicity: (Based upon reports of the Underwriters' Laboratories, Chicago, Ill.)
+ 21%, by volume, of vapour in air kills or seriously injures animals in 2 hours.
3+ 10%, by volume, of vapour in air kills or seriously injures animals in 2 hours.
4+ 10%, by volume, of vapour in air kills or seriously injures animals in 30 minutes.

Physiological Action and Toxicity: Animal experimentation indicates that the toxicity of the chlorine-fluorine derivatives of methane and ethane is very low. The U.S. Bureau of Mines reports that concentrations of 20% dichloro-difluoro-methane and of 14% dichloro-tetrafluoro-ethane do not have any serious effects on dogs and guinea pigs after repeated 8-hour exposures. As indicated above, some of the compounds are rather more toxic. Cases of industrial poisoning have not been reported, but it would be possible for these refrigerants to cause death through asphyxiation. On contact with flames or hot surfaces, "Freon" may be decomposed to carbon monoxide, chlorine, phosgene, hydrochloric acid and hydrofluoric acid.

Fire Hazard: None. The "Freon" compounds are non-flammable.

Furfural (Furfurol; Furfuranecarboxylic Aldehyde; Pyromucic Aldehyde; Furfuraldehyde)

Formula: C_4H_3OCHO or 

Properties: Colourless liquid when pure, changing to a reddish brown when exposed to light and air. Penetrating odour. Flammable. Boils at 322°F, and has a vapour density of 3·31. The liquid is very slightly volatile at ordinary temperatures. Furfural is about 8% soluble in water, and readily soluble in alcohol, ether, benzene, and other organic solvents.

Uses: As a solvent for cellulose derivatives, synthetic resins, and oils; as an accelerator in rubber; as a fungicide; and in the manufacture of various condensation products.

Mode of Entry into Body: As a vapour, through inhalation.

Physiological Action and Toxicity: Irritant to the mucous membranes and eyes; heavy exposure can cause excitation of the central nervous system, cramps and paralysis. It is about one-third as toxic as formaldehyde, when administered to animals other than as a vapour. Actually, its practical toxicity is much less, due to its low volatility.

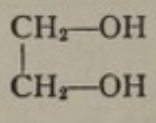
Signs and Symptoms: Redness and weeping of the eyes, irritation of the nose and throat, headache. Furfural is said to cause anaesthesia of the cornea.

Recommended M.A.C.: No maximum allowable concentration has been recommended, since the low volatility of the substance usually keeps concentrations below the injurious level.

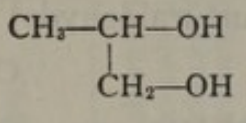
Fire Hazard: Slight; flash point: 150°F; minimum explosive limit: 2·1%. It is a class 3 flammable liquid, having about the same hazard as kerosene.

Glycols

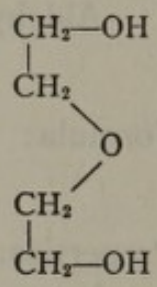
The glycols are aliphatic hydrocarbons which contain two hydroxy (OH) groups. The simplest is ethylene glycol. The other common glycols used in industry are propylene glycol and diethylene glycol.



Ethylene Glycol



Propylene Glycol



Diethylene Glycol

When one of the hydroxy groups in a glycol is replaced with a chlorine atom, a chlorohydrin is formed, *e.g.*, ethylene chlorohydrin. When the hydrogen atom of the hydroxy group is replaced by an alkyl group, such as the methyl, $-CH_3$, an ether is formed, *e.g.*, ethylene glycol monomethyl ether (methyl cellosolve). The two examples mentioned are discussed elsewhere in this book; in addition, there are many other glycols and glycol derivatives which are finding increasing use in industry as solvents for plastics, inks and coatings, and as anti-freeze compounds.

Properties: The glycols are rather viscous, colourless liquids which are soluble in water and alcohol in all proportions, but are almost completely insoluble in fatty oils. Their volatility is very low at ordinary room temperatures.

	Specific Gravity	*Boiling Point °F.*	*Vapour Density*	*Flash Point °F.*
Ethylene Glycol.....	1·11	387	2·14	232
Propylene Glycol.....	1·04	370	2·52	210
Diethylene Glycol.....	1·12	472	3·66	255

Mode of Entry into Body: Through accidental or intentional ingestion; to a lesser extent, through inhalation of the vapour when the glycols are heated.

Physiological Action and Toxicity: Though ethylene glycol and diethylene glycol are poisonous when ingested, there has been little definite evidence that inhalation of the vapour, under industrial conditions, has produced organic injury. At ordinary room temperatures the glycols are practically non-volatile; when heated, the vapour given off has been reported to have produced symptoms pointing to an action on the central nervous system and bone marrow. When ingested, diethylene glycol and ethylene glycol have both produced severe renal damage, due to deposition of oxalic acid crystals in the kidney tubules, and fatalities have been reported. Propylene glycol appears to be much less toxic.

Recommended M.A.C.: No values have been set, due to the low toxicity of the glycols under most conditions of use.

Fire Hazard: Flash points are given above.

Hexamethylene Tetramine ("Hex"; Hexamine; Urotropine)

Formula: $(CH_2)_6N_4$.

Properties: A white, crystalline powder, soluble in water and alcohol and insoluble in ether.

Uses and Occurrence: In addition to its use as a urinary antiseptic in medicine, hexamine is an excellent rubber accelerator. However, because of its irritant effect on the skin, it is gradually being replaced by less troublesome materials.

Physiological Action and Toxicity: The use of hexamethylene tetramine in industry has caused numerous cases of dermatitis. The persons affected are usually those handling the warm rubber stock, subsequent to the mixing operation. Few cases arise in individuals handling the dry powder.

Apparently the perspiration associated with the handling of the warm stock causes a decomposition of the hexamine which is then oxidized to formic acid; the latter is thought to be the real cause of the dermatitis. No systemic effects have been reported.

Signs and Symptoms: The rash occurs first on the wrists, then extends up the forearms, and may involve the face and neck or even extend over the whole body. It resembles the rash of poison ivy, and is inclined to be very itchy, particularly at night. It usually disappears within a few days after removal from exposure.

Recommended M.A.C.: No value has been set.

Hydrochloric Acid (Hydrogen Chloride; Muriatic Acid)

Formula: HCl.

Properties: Hydrogen chloride is a colourless gas having a pungent, irritating odour.
Vapour Density: 1·27 times as heavy as air.
Soluble in water, alcohol and ether.
On exposure to air, the atmospheric moisture is condensed, with the formation of minute droplets of hydrochloric acid solution.

Uses and Occurrence: The anhydrous hydrogen chloride is seldom encountered in industry. Hydrochloric acid solution, on the other hand, is widely used in organic synthesis, in picking of metals, in the etching and engraving trades, in electroplating, and many other industries.

Mode of Entry into Body: Through inhalation of the vapour or mist. Acts locally on the skin.

Physiological Action and Toxicity: Hydrogen chloride is an irritant to the mucous membranes of the eyes and respiratory tract, and a concentration of 35 ppm causes irritation of the throat after short exposures. Concentrations of 50 to 100 ppm are tolerable for 1 hour. More severe exposures result in pulmonary oedema, and often laryngeal spasm. Concentrations of 1,000 to 2,000 ppm are dangerous, even for brief exposures. Mists of hydrochloric acid are considered less

harmful than the anhydrous hydrogen chloride, since the droplets have no dehydrating action. In general, hydrochloric acid causes little trouble in industry, other than from accidental splashes and burns.

Signs and Symptoms: Irritation of the eyes, nose and throat, with lacrimation and burning, cough, and, in severe exposures, a sense of constriction of the chest and chest pain. Râles may be present in cases of severe gassing.

Recommended M.A.C.: 10 ppm for an 8-hour working day.

Hydrocyanic Acid (Hydrogen Cyanide; Formonitrile; Prussic Acid; Cyanhydric Acid)

Formula: HCN.

Properties: A colourless, volatile liquid having an odour of bitter almonds.
Boiling Point: 79°F.
Vapour Density: 0·935 times as heavy as air.

Uses and Occurrence: Used as a disinfectant and fumigant, and in the manufacture of dye intermediates. The gas is liberated by the action of acids on cyanides. Exposure may occur in the extraction of gold from its ore.

Mode of Entry into Body: Through inhalation of the gas; by absorption through the skin.

Physiological Action and Toxicity: Hydrocyanic acid and the cyanides are true protoplasmic poisons, combining in the tissues with the enzymes associated with cellular oxidation. They thereby render the oxygen unavailable to the tissues, and cause death through asphyxia. The suspension of tissue oxidation lasts only while the cyanide is present; upon its removal, normal function is restored provided death has not already occurred. Hydrocyanic acid combines with difficulty with haemoglobin, but readily with methaemoglobin to form cyanmethaemoglobin. This fact is utilized in the treatment of cyanide poisoning, when an attempt is made to induce methaemoglobin formation. The presence of cherry-red venous blood in cases of cyanide poisoning is due to the inability of the tissues to remove the oxygen from the blood. Exposure to concentrations of 100 to 200 ppm for periods of 30 to 60 minutes is dangerous to life.

Signs and Symptoms: In cases of acute cyanide poisoning, death is extremely rapid; frequently the person exposed takes only a few breaths, then falls; death may be immediate, though sometimes breathing may continue for a few minutes. In less acute cases, there is headache, dizziness, unsteadiness of gait, a feeling of suffocation, and nausea. Where the patient recovers, there is rarely any disability or sequelae.

Recommended M.A.C.: 20 ppm for an 8-hour working day.

Fire Hazard: Liquid hydrocyanic acid is flammable, burning much like alcohol. The flash point is 0°F; the minimum explosive limit is 5·6%, the maximum explosive limit 40%, by volume, of the gas in air. Hydrocyanic acid is not explosive in the concentrations ordinarily used for fumigation.

Hydrofluoric Acid (Hydrogen Fluoride)

Formula: HF.

Properties: A colourless, fuming liquid. Is extremely corrosive to the skin.
Boiling Point: 67°F.
Vapour Density: 0·71 times as heavy as air.
Soluble in water.

Uses and Occurrence: Is used for pickling metals, etching glass and treating ceramics. Exposure is encountered in the manufacture of frosted electric light bulbs and fluorescent tubes, in the brewing industry where it is used as an antiseptic, and in the production of beryllium and aluminum. Is used as a catalyst in the petroleum industry.

Mode of Entry into Body: Through inhalation of the vapour. Acts locally on the skin.

Physiological Action and Toxicity: Hydrogen fluoride is extremely irritant and corrosive to the skin and mucous membranes. Inhalation of the vapour may cause ulcers of the upper respiratory tract. Concentrations of 50 to 250 ppm are dangerous, even for brief exposures. On the skin, hydrofluoric acid produces severe chemical burns which are slow in healing. The subcutaneous tissues may be affected, becoming blanched and bloodless. Gangrene of the affected areas may follow.

Signs and Symptoms: Conjunctivitis, rhinitis, dermatitis, and ulceration of the mucous membrane of the nose are the common signs following inhalation of the gas. The effect on the skin is that of a chemical burn, as described above.

Recommended M.A.C. The maximum allowable concentration recommended is 3 ppm for an 8-hour working day.

Hydrogen Sulphide (Sulphuretted Hydrogen)

Formula: H_2S.

Properties: A colourless gas, having an offensive, rotten-egg odour. Inflammable.
Specific Gravity: Slightly heavier than air (1·189).
Soluble in water and alcohol.

Occurrence: Is not used in industry, but occurs as a by-product in certain industrial processes. It is formed by the decomposition of animal or vegetable matter in the presence of insufficient oxygen. It is a hazard in sewers, mines, wells and tunnels. It is a by-product in the manufacture of viscose rayon; it occurs in the manufacture of sulphides, sulphates and certain dyes, and in the refining of oils, particularly those produced in Texas and Mexico.

Mode of Entry into Body: Through inhalation of the gas. Not absorbed through the skin in harmful amounts.

Physiological Action and Toxicity: Hydrogen sulphide is both an irritant and an asphyxiant. Low concentrations of from 20 to 150 ppm cause irritation of the eyes; slightly higher concentrations may cause irritation of the upper respiratory tract, and if exposure is prolonged, pulmonary oedema may result. The irritant action has been explained on the basis that H_2S combines with the alkali present in moist surface tissues to form sodium sulphide, a caustic.

With higher concentrations the action of the gas on the nervous system becomes more prominent, and a 30-minute exposure to 500 ppm results in headache, dizziness, excitement, staggering gait, diarrhoea and dysuria, followed sometimes by bronchitis or bronchopneumonia. The action on the nervous system is, with small amounts, one of depression; in larger amounts it stimulates, and with very high amounts

the respiratory centre is paralyzed. Exposures of 800 to 1000 pm may be fatal in 30 minutes, and higher concentrations are instantly fatal. Fatal hydrogen sulphide poisoning may occur even more rapidly than that following exposure to a similar concentration of hydrogen cyanide. H_2S does not combine with the haemoglobin of the blood; its asphyxiant action is due to paralysis of the respiratory centre.

Signs and Symptoms: With repeated exposures to low concentrations conjunctivitis, photophobia, corneal bullae, tearing, pain and blurred vision are the commonest findings. Higher concentrations may cause rhinitis, bronchitis and occasionally pulmonary oedema. Exposure to very high concentrations results in immediate death. Chronic poisoning results in headache, inflammation of the conjunctivae and eyelids, digestive disturbances, loss of weight and general debility.

Recommended M.A.C.: 20 ppm for an 8-hour work period.

Fire Hazard: The minimum explosive limit is 4·3%; the maximum explosive limit is 46% by volume, of the gas in air. The gas is flammable, with an ignition temperature of 500° F.

Iodine

Formula: I_2.

Properties: Purplish-black, flat crystals, slightly volatile at room temperature. As the temperature is increased, purple fumes are given off.
Melting Point: 237°F.; Boiling Point: 363°F.
Vapour Density: 8·7 times heavier than air.
Soluble in alcohol, carbon disulphide, chloroform, ether, glycerol and alkaline iodide solutions; insoluble in water.

Uses and Occurrence: Used in the manufacture of pharmaceuticals, dyes, and organic compounds. Is used also as an analytical reagent.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: The effect of iodine vapour upon the body is similar to that of chlorine and bromine,

but it is more irritating to the lungs. Serious exposures are seldom encountered in industry, due to the low volatility of the solid at ordinary room temperatures.

Signs and Symptoms: Irritation and burning of the eyes, lachrymation, cough, irritation of the nose and throat.

Recommended M.A.C.: 1 mgm per 10 cubic metres of air.

Iron Carbonyl

Formulae: There are two carbonyls of iron, the pentacarbonyl $Fe(CO)_5$, and the tetracarbonyl $Fe(CO)_4$.

Properties: The pentacarbonyl is a thick, oily, reddish-brown or yellow liquid. Boils at 217°F. and solidifies into yellowish crystals at 68°F. It is soluble in nickel carbonyl and in most organic solvents; insoluble in water. The tetracarbonyl occurs as dark green lustrous crystals which decompose to iron and carbon monoxide on heating to 284°F. Insoluble in water, but soluble in most organic solvents.

Uses and Occurrence: Used in organic synthesis and in military explosives. The pentacarbonyl has been used as an anti-knock agent in gasoline.

Mode of entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: The iron carbonyls are considered to resemble nickel carbonyl in their action on the body, though their toxicity may be somewhat less. No cases of industrial poisoning have been reported.

Recommended M.A.C.: No values have been set, but the maximum allowable concentration should probably be about the same as that of nickel carbonyl; *i.e.*, 1 ppm.

Fire Hazard: The pentacarbonyl ignites at 95°F.

Isoprene

Formula: $CH_2=C(CH_3)-CH=CH_2$

Properties: A colourless liquid, having a specific gravity of 0·681.

Boiling Point: 93°F.

Insoluble in water, but soluble in alcohol and ether.

Uses and Occurrence: Used in the manufacture of synthetic rubber.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Isoprene is irritant to the mucous membranes of the eyes, nose and respiratory tract. Concentrations of 2 per cent of isoprene in air are not narcotic to mice, but do produce irritative changes in the bronchi. Concentrations of 3·5 per cent to 4·5 per cent are narcotic to mice, and concentrations of 5 per cent are fatal.

Recommended M.A.C. No value has been set.

Ketones

The commonest ketone used in industry is acetone. Recently, others have been introduced, such as butanone, pentanone, methyl butyl ketone (hexanone) and methyl isobutyl ketone (hexone). The latter two are of relatively little importance; refer to "Acetone," "Butanone" and "Pentanone" for further details.

In general, the ketones are narcotics, and irritant to the mucous membranes of the eyes and upper respiratory tract. Starting with acetone, as one ascends the series, solubility and volatility decrease, the boiling point increases and the irritant effect becomes greater.

Lead and its Compounds

Symbol: Pb.

Lead poisoning is one of the commonest of occupational diseases. The presence of lead-bearing materials or lead compounds in an industrial plant does not necessarily result in exposure on the part of the workman. The lead must be in such form, and so distributed, as to gain entrance into the body or tissues of the workman in measurable quantity, otherwise no exposure can be said to exist.

The lead compounds commonly occurring in industry are:

1. Suboxide of lead, (Pb_2O) a velvety black powder which forms on metallic lead and upon molten lead. It is dislodged from molten lead when the latter is stirred or poured.

2. Lead monoxide, (PbO). This compound exists in several allotropic forms. Thus, if moderately heated, the oxide is yellow in colour—"massicot". If greater heat is applied, the oxide is red—"litharge"; the latter is used in the manufacture of storage batteries, compounding rubber, etc.
3. Red oxide of lead, or minium, (Pb_3O_4) used in the manufacture of batteries, pigments, paints, glazes, enamels and glass.
4. Lead peroxide or dioxide, (PbO_2)
5. Lead sesquioxide, (Pb_2O_3).
6. White lead, or basic carbonate of lead, ($PbCO_3$ combined with lead hydroxide $Pb(OH)_2$), used in making white paint, as a glaze, and in making rubber compounds.
7. Lead sulphate, ($PbSO_4$); used in white paints and in the rubber industry; occurs also as a constituent in the fumes and dusts in smelting of ore.
8. Lead chromate, ($PbCrO_4$) or "chrome yellow"; used as a pigment in paints and dyes. It is mixed with prussian blue to form "chrome green". Basic lead chromate is the pigment "chrome orange".
9. Lead sulphide, (PbS) is the lead ore "galena".
10. Lead acetate, ($Pb(CH_3COO)_2$) is "sugar of lead". Used chiefly in the manufacture of other lead compounds.
11. Lead arsenate, ($Pb_3(AsO_4)_2$) is used as an insecticide.
12. Lead tetraethyl, ($Pb(C_2H_5)_4$) is used as an anti-knock agent in many gasolines. It constitutes a hazard to men manufacturing it, and blending it with gasoline, but it is not a hazard following blending, as the concentration of the tetraethyl lead is around 1:1300 in the blended gasoline.
13. Lead naphthallate; is becoming more commonly used as a drier in paints.

Mode of Entry into Body:

1. By inhalation of the dusts, fumes, mists or vapours.
2. By ingestion of lead compounds trapped in the upper respiratory tract or introduced into the mouth on food, tobacco, fingers, or other objects.

3. Through the skin; this route is of especial importance in the case of the organic compounds of lead, as lead tetraethyl. In the case of the inorganic forms of lead, this route is of no practical importance.

Physiological Action and Toxicity: When lead is ingested, much of it passes through the body unabsorbed, and is eliminated in the faeces. The greater portion of the lead that is absorbed is caught by the liver and excreted, in part, in the bile. For this reason, larger amounts of lead are necessary to cause poisoning if absorption is by this route, and a longer period of exposure is usually necessary to produce symptoms. On the other hand, when lead is inhaled, absorption takes place easily from the respiratory tract and symptoms tend to develop more quickly. From the point of view of industrial poisoning, inhalation of lead is much more important than is ingestion.

Lead is a cumulative poison. A part of the small daily doses, which individually are unimportant, is not eliminated; eventually a point is reached where symptoms and disability occur.

The fate of lead in the body is at present a controversial issue. It was formerly believed that lead entered the circulation following absorption in the lungs and was transported as the diphosphate to the bones where it was deposited as the triphosphate. The metabolism of lead followed closely that of calcium; administration of calcium drove the lead into the bones, and withdrawal of calcium permitted its release, or "de-leading". Recently, the storing effect of lead has been questioned. However, it is still generally thought that symptoms of lead poisoning occur only in the presence of circulating lead.

Lead produces a brittleness of the red blood cells so that they haemolyse with but slight trauma; the haemoglobin is not affected. Due to their increased fragility, the red cells are destroyed more rapidly in the body than normally, producing an anaemia. The loss of circulating red cells stimulates the production of new young cells which on entering the blood stream, are acted upon by the circulating lead, with resultant coagulation of their basophilic material.

These cells, after suitable staining, are recognized as "stippled cells". As regards the effect of lead on the white blood cells, there is no uniformity of opinion. In addition to its effect on the red cells of the blood, lead produces a damaging effect on the organs or tissues with which it comes in contact. No specific or characteristic lesion is produced. Autopsies of deaths attributed to lead poisoning and experimental work on animals, have shown pathological lesion of the kidneys, liver, male gonads, nervous system, blood vessels and other tissues. None of these changes, however, have been found consistently.

In cases of lead poisoning, the amount of lead found in the blood is frequently in excess of 0·07 mgm per 100 cc of whole blood. The urinary lead excretion generally exceeds 0·1 mgm per litre of urine.

The toxicity of the various lead compounds appears to depend upon several factors. (1) The solubility of the compound in the body fluids. (2) The fineness of the particles of the compound; solubility is greater, of course, in proportion to the fineness of the particles. (3) Conditions under which the compound is being used; where a lead compound is used as a powder, contamination of the atmosphere will be much less where the powder is kept damp. Of the various lead compounds, the carbonate, the monoxide and the sulphate are considered to be more toxic than metallic lead or other lead compounds. Lead arsenate is very toxic, due to the presence of the arsenic radicle. The toxicity of lead chromate, or "chrome yellow" is less than would be expected, due to its low solubility.

Signs and Symptoms: Industrial lead poisoning commonly occurs following prolonged exposure to lead or its compounds. The common clinical types of lead poisoning may be classified according to their clinical picture as (a) alimentary; (b) neuromotor; and (c) encephalic. Some cases may show a combination of clinical types. The alimentary type occurs most frequently, and is characterized by abdominal discomfort or pain. Severe cases may present actual colic. Other complaints are: constipation and/or diarrhoea, loss of appetite, metallic taste, nausea and vomiting, lassitude, insomnia, weakness, joint and muscle

pains, irritability, headache and dizziness. Pallor, lead line on the gums, pyorrhoea, loss of weight, abdominal tenderness, basophilic stippling, anaemia, slight albuminuria increased urinary excretion, and an increase in the lead content of the whole blood, are signs which may accompany the above symptoms.

In the neuro-muscular type, the chief complaint is weakness, frequently of the extensor muscles of the wrist and hand, unilateral or bilateral. Other muscle groups which are subject to constant use may be affected. Gastroenteric symptoms are usually present, but are not as severe as in the alimentary type of poisoning. Joint and muscle pains are likely to be more severe. Headache, dizziness and insomnia are frequently prominent. True paralysis is uncommon, and is usually the result of prolonged exposure.

Lead encephalopathy is the most severe but the rarest manifestation of lead poisoning. In the industrial worker it follows rapid and heavy lead absorption. Organic lead compounds, such as tetraethyl lead, are absorbed rapidly through the skin as well as through the lungs, and are selectively absorbed by the central nervous system. The clinical picture in these cases is usually an encephalopathy. With inorganic lead compounds, comparable concentrations in the central nervous system are reached only when the workplace is heavily contaminated with vapour, fume and dust. Encephalopathy begins abruptly, and is characterized by signs of cerebral and meningeal involvement. There is usually stupor, progressing to coma, with or without convulsion, and often terminating in death. Excitation, confusion and mania are less common. In milder cases of shorter duration, there may be symptoms of headache, dizziness, somnolence and insomnia. The cerebrospinal pressure may be increased.

Recommended M.A.C.: 1·5 mgm per 10 cu. metres of air, for an 8-hour working day.

Magnesium and its Compounds

Symbol: Mg.

Magnesium is a light, silver metal, having a melting point of 1209° F. It is used chiefly in the manufacture of light

alloys, pyrotechnic and photographic flash powders, as a substitute for zinc in dry cell batteries, and in organic synthesis.

Mode of Entry into Body: Through inhalation of the dust or fume. Particles penetrating the skin may produce local effects.

Physiological Action and Toxicity: The inhalation of fumes of freshly sublimed magnesium oxide may cause metal fume fever. (See "Metal Fume Fever.") There is no evidence that magnesium produces true systemic poisoning. Occupational health hazards may exist in magnesium foundries from the presence of atmospheric contaminants such as fluorides, sulphur dioxide, carbon tetrachloride and chromium compounds.

Particles of metallic magnesium or magnesium alloy which perforate the skin or gain entry through cuts and scratches may produce a severe local lesion characterized by the evolution of gas and acute inflammatory reaction, frequently with necrosis. The condition has been called a "chemical gas gangrene". Gaseous blebs may develop within 24 hours of the injury. The inflammatory response is marked at the site of injury and there may be signs of lymphangitis. The lesion is very slow to heal.

The most serious hazard presented by magnesium is the danger from burns. Finely divided magnesium powder is readily combustible and may be ignited by a spark when dispersed in the air. The molten metal, on splashing, frequently catches fire and burns with intense heat. The injuries sustained by workmen through contact with the burning metal do not differ in any way from ordinary thermal burns.

Recommended M.A.C.: 150 mgm per 10 cubic metres of air is recommended as the maximum allowable concentration for magnesium oxide fume.

Fire Hazard: Ingots or blocks of magnesium are difficult to ignite. However, metals and alloys in the form of powder, shavings, strips or sheets which contain a high percentage of magnesium, are readily flammable.

Manganese and its Compounds

Symbol: Mn.

Manganese is a hard, brittle, grey metal closely resembling iron. It is used chiefly in the manufacture of manganese steel alloys and alloys of copper, aluminum and other metals. Exposure to manganese dioxide (MnO_2) occurs in the crushing and loading of the pyrolusite ore. The oxides are used as oxidising and bleaching agents for many purposes; the chloride is used in dyeing and the persulphate, manganate and permanganate are also used as oxidizing and bleaching agents. Other uses of manganese include the manufacture of dry cell batteries, matches and fireworks. Exposure to manganese fumes may occur during the welding of manganese alloys.

Mode of Entry into Body: Through inhalation of the dusts and fumes.

Physiological Action and Toxicity: Chronic manganese poisoning is a rare but clearly characterized disease which results from the inhalation of fumes or dusts of manganese. Exposure to heavy concentrations of dusts or fumes for three months may produce the condition, but usually cases develop after one to three years of exposure. The central nervous system is the chief site of damage, the characteristic lesion being a destruction of the cells of the basal ganglia, followed by gross scarring and shrinking. Sometimes the globus pallidus and other parts of the nervous system are involved. The symptomatology is described below. The lowest average concentration at which chronic manganese poisoning is reported to have occurred is 30 mgm per cubic metre of air. If cases are removed from exposure shortly after the appearance of symptoms, some improvement in the patient's condition frequently occurs, though there may be some residual disturbances in gait and speech. When well established, however, the disease results in permanent disability.

Individuals exposed to dusts and fumes of manganese have been reported by several investigators to suffer from a much higher incidence of upper respiratory infections and pneumonia than does the general population. It has not yet

been possible to prove that a definite pneumonitis results in humans from exposure to manganese dusts or fumes under industrial conditions. However, experiments with mice have produced definite and striking lung pathology which varied in intensity with the length of exposure to the dust. Microscopically, there was mononuclear infiltration, which, in the animals longest exposed, was quite intense; the alveolar cells and cells lining the bronchi were swollen and hydropic, and the alveoli and interstitial spaces were packed with large mononuclear cells. Areas of necrosis and haemorrhage were not uncommon. Few lymphocytes and practically no polymorphonuclear cells were seen.

Signs and Symptoms: Chronic manganese poisoning begins usually with complaints of langour and sleepiness. This is followed by weakness in the legs and the development of a stolid, mask-like facies, and the patient speaks with a slow monotonous voice. Then muscular twitchings appear. varying from a fine tremor of the hands to coarse, rhythmical movements of the arms, legs and trunk. Nocturnal cramps of the legs appear about the same time. There is a slight increase in tendon reflexes, ankle and patellar clonus, and a typical Parkinsonian slapping gait. The handwriting may be quite minute. There are no sensory disturbances, and no eye, gastro-intestinal or genito-urinary complaints. The urine and spinal fluid are normal, and the blood shows no abnormality or only a slight leucopaenia. The symptoms may simulate progressive bulbar paralysis, post-encephalitic Parkinsonism, multiple sclerosis, amyotrophic lateral sclerosis and progressive lenticular degeneration (Wilson's disease). Often, a history of exposure is the only aid in establishing the diagnosis.

Recommended M.A.C.: 60 mgm per 10 cubic metres of air.

Fire Hazard: Potassium permanganate and other oxidizing compounds in contact with organic materials will cause violent combustion on ignition.

Mercury and its Compounds (Quicksilver; Hydrargyrum)

Symbol: Hg.

Properties: A silvery liquid metal. Specific Gravity: 13·6. Solidifies at −38°F, and boils at 675°F. The vapour is 6·9 times heavier than air. At room temperatures sufficient mercury evaporates from the liquid state to constitute a hazard to health. Mercury is soluble in acids, insoluble in water, alcohol and ether. It forms amalgams with most of the common metals except iron and platinum.

Uses and Occurrences: Exposure to mercury is not common in Canada, but is important when it does occur. Mercury is used in the manufacture of scientific instruments, (thermometers, barometers, high vacuum pumps, etc.) electrical contacts and mercury vapour lamps, and mercury compounds are employed in many diverse industries. Mercury fulminate ($Hg(CNO)_2$) is a constituent of percussion caps and detonators. Mercuric nitrate ($Hg(NO_3)_2$) and mercuric chloride ($HgCl_2$) are other mercury compounds used commonly. Recently there has been increased use of organic mercury compounds, such as the ethyl mercury phosphate or chloride, in the seed treating industry and as fungicides. Dimethyl mercury compounds have also been used as fungicides.

Mode of Entry into Body: Occurs in industry chiefly through inhalation of the vapour or dust. Ingestion may be a route where soiled hands are not washed before eating.

Physiological Action and Toxicity: Mercury is a general protoplasmic poison; after absorption it circulates in the blood, possibly as the albuminate or the oxy-chloro-albuminate, and is stored in the liver, kidneys, spleen and bone. It is eliminated in the urine, faeces, sweat, saliva and milk. In industrial poisoning, the chief effect is upon the central nervous system and upon the mouth and gums. Colitis has been reported not infrequently; rarely, a nephritis is reported. The organic mercury compounds, like the organic lead compounds, appear to have an affinity for the lipoid-containing organs, resulting in disturbances of the central nervous system resembling those of tetraethyl lead.

Fulminate of mercury rarely produces symptoms of systemic poisoning, but frequently causes a dermatitis. Organic mercury compouds may act as vesicants on the skin.

Signs and Symptoms: The cardinal symptoms of industrial mercury poisoning are stomatitis, tremors, and psychic disturbances. Usually the first complaints are of excessive salivation and pain on chewing; in severe cases there may be gingivitis, with loosening of the teeth, and a dark line on the gum margins resembling the "lead line". In slow poisoning the salivation may be absent, and the only complaint that of dryness of the throat and mouth. In addition to the stomatitis, the patient may have colicky abdominal pain and diarrhoea. Tremor and psychic disturbances are commonly seen in the slow, chronic form of the poisoning; the tremor is of the intention type, and may be seen when the patient spreads the outstretched fingers or protrudes the tongue, or attempts to perform specified movements. Muscles of the face, hands and arms are chiefly affected. In more severe cases there may also be convulsive or shaking movements; writing is frequently illegible. Hyperactive knee-jerks and scanning speech may be present in advanced cases. The psyhic disturbance (so-called "erethism") includes such changes as loss of memory, insomnia, lack of confidence, irritability, vague fears and depression.

With poisoning by organic mercury compounds, the nervous system alone is usually affected. Tremor, ataxia, difficulty in speaking, and gross constriction of the visual fields are reported, but there is no loss of memory or intelligence. The peripheral nerves and dorsal spinal roots may be affected, and the patient's condition resemble a dorsal root lesion.

The dermatitis produced by fulminate of mercury takes the form of small, discrete ulcers on the exposed parts, and is usually accompanied by conjunctivitis and inflammation of the mucous membranes of the nose and throat.

Recommended M.A.C.: 1 mgm per 10 cu. metres of air, for an 8-hour working period.

Fire Hazard: Fulminate of mercury is a primary explosive. The other mercury compounds present no particular fire hazard.

Metal Fume Fever

Metal fume fever is an acute febrile reaction which results from the inhalation of fumes of the metallic oxides. The condition occurs most frequently following exposure to freshly sublimed zinc oxide, in occupations where zinc or zinc-containing alloys such as brass and bronze are heated to a temperature near their boiling point. Fumes of copper oxide and magnesium oxide have been shown to cause typical episodes of chills and fever. Other metallic oxides, such as those of lead, manganese, arsenic and antimony have been reported to cause similar illness.

Symptoms usually begin several hours after the patient has left exposure. A feeling of dryness in the throat is noticed first, followed by a dry cough and some tightness in the chest. The patient then develops chills which may last for an hour or two, with general aching and malaise. Nausea and vomiting may occur. Then the fever rises, often to 102°F. or higher, and is followed by profuse sweating and return of the temperature to normal. There is usually a leucocytosis. The symptoms generally pass off in 12 to 24 hours, and the patient feels well, or may complain of some weakness and lassitude for a day or so. It is characteristic of metal fume fever that the onset is acute, the duration short, and recovery complete. Following the fever the patient is relatively immune to further attacks for a few days, but this immunity is rapidly lost. Thus, cases occur most frequently on Mondays, after holidays, and amongst new employees.

Due to the transitory nature of the illness and the fact that cases are seldom investigated, there is little information in the literature regarding the pathology of the disease. No fatalities have been reported. It has been suggested that the fever is a reaction of the body to proteids liberated by the destructive action of the fume on the epithelial cells of the respiratory tract. Cases coming under observation may be confused with attacks of influenza and pneumonia. Even though the association of the illness with exposure to metallic fumes be recognized, care should be taken not to overlook the possibility of cadmium fume poisoning which may cause acute pulmonary oedema, or poisoning from lead or other heavy metals.

Methyl Acetate

Formula: CH_3COOCH_3

Properties: A colourless, volatile fluid. Inflammable. Has a pleasant, fruity odour.
Specific Gravity: 0·92 times as heavy as water.
Boiling Point: 135° F.
Vapour Density: 2·6 times heavier than air.
Volatility: Is 2·2 times less volatile than ethyl ether.
Soluble in water up to 25%; is miscible with the common hydrocarbon solvents.

Uses and Occurrences: Is an excellent solvent, and is used in the manufacture of paints, varnishes, lacquers, plastics, artificial leather and perfume.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Methyl acetate is narcotic, but is less so than the higher members of the acetate series. It has an irritating effect upon the mucuous membranes of the eyes and upper respiratory tract, and in this respect its action is stronger than that of the higher members of the series. The irritant concentration is about 10,000 ppm.

Signs and Symptoms: Irritation and burning of the eyes, lacrimination, dyspnoea, palpitation of the heart, and complaints of depression or of feeling intoxicated or dizzy.

Recommended M.A.C.: 200 ppm. for an 8-hour working day.

Fire Hazard: Dangerous; flash point is 15° F. Minimum explosive limit is 4%; maximum explosive limit, 14% by volume of the vapour in air. Class 1 flammable liquid.

Methyl Alcohol (Methanol; Methyl Hydrate; Methyl Hydroxide; Wood Alcohol; Colonial Spirits; Columbian Spirits)

Formula: CH_3OH

Properties: A clear, colourless liquid having a faint odour similar to that of ethyl alcohol. Inflammable.
Specific Gravity: 0·79 times as heavy as water.
Boiling Point: 147° F.
Vapour Density: 1·11 (about the same as that of air).
Volatility: 6·3 times less volatile than ether. Fairly readily volatile. Soluble in water, alcohol and ether.

Uses and Occurrence: Used as a solvent in the manufacture of lacquers, varnishes, shellac, and cleaning and polishing materials. Is used as a denaturant for ethyl alcohol. Is also used in organic synthesis, and to some extent as a fuel.

Mode of Entry into Body: Through inhalation of the vapour. Methyl Alcohol can be absorbed through the skin, though it appears that this route is of relatively little importance in industrial poisoning.

Physiological Action and Toxicity: Methyl alcohol possesses distinct narcotic properties. It is also slightly irritant to the mucous membranes. Its main toxic effect is exerted upon the nervous system, particularly the optic nerves and possibly the retinae. The effect upon the eyes has been attributed to optic neuritis, which subsides but is followed by atrophy of the optic nerve. Other workers ascribe the effect to a degeneration of the ganglion cells of the retinae. Once absorbed, methyl alcohol is only very slowly eliminated. Coma resulting from massive exposures may last as long as two to four days. In the body the products formed by its oxidation are formaldehyde and formic acid, both of which are toxic; this oxidation process takes place slowly, and is probably of little importance, since both products are neutralized in the alkaline media of the body. The toxicity of methyl alcohol is thought to be due to the action of the unoxidized alcohol itself. Because of the slowness with which it is eliminated, methyl alcohol should be regarded as a cumulative poison. Though single exposures may cause no harmful effect, daily exposure may result in the accumulation of sufficient methyl alcohol in the body to cause illness.

The concentration required to produce narcosis in most animals is quite high, certainly above 25,000 ppm even for fairly long exposures. Where the exposure is repeated daily, the toxic concentration is much lower. Slight symptoms of anemia and conjunctivitis have been reported in workers exposed daily to 200 ppm. On the other hand, dogs exposed to 500 ppm for 8 hours a day for 379 days have shown no significant effect.

Signs and Symptoms: Severe exposures may cause dizziness, unconsciousness, sighing respiration, cardiac depression, and eventually death. Where the exposure is less severe, the first symptoms may be blurring of vision, photophobia and conjunctivitis, followed by the development of definite eye lesions. There may be headache, gastro-intestinal disturbances, dizziness and a feeling of intoxication. The visual symptoms may clear temporarily, only to recur later and progress to actual blindness. Irritation of the mucous membranes of the throat and respiratory tract, peripheral neuritis, and occasionally, symptoms referable to other lesions of the nervous system have been reported. The skin may become dry and cracked due to the solvent action of methyl alcohol.

Recommended M.A.C.: 200 ppm for an 8-hour working day.

Fire Hazard: Dangerous. Flash point: 54° F. Minimum explosive limit is 6%, maximum explosive limit 36·5%, by volume, of the vapour in air. Class 2 flammable liquid.

Methyl Chloride (Chloromethane; Monochloromethane)

Formula: CH_3Cl

Properties: A colourless gas, having a sweet, ether-like odour. Inflammable, forming phosgene on burning in open flame. Liquefies at −10·7° F. The gas is 1·7 times as heavy as air. Soluble in water, alcohol, ether and chloroform.

Uses: Chiefly as a refrigerant.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Methyl chloride has very slight irritant properties and may be breathed without noticeable discomfort. It has some narcotic action, but this effect is several times weaker than that of chloroform. In the body, the absorbed methyl chloride is broken down into methyl alcohol and hydrochloric acid, the latter combining with the sodium in the blood and being excreted as sodium chloride. Acute poisoning, characterized by the narcotic effect, is rare in industry. Repeated exposure to low concentrations causes damage to the central nervous

system, and, less frequently, to the liver, kidneys, bone marrow and cardio-vascular system. Haemorrhages into the lungs, intestinal tract and dura have been reported. Sprayed on the skin, methyl chloride produces anaesthesia through freezing of the tissue as it evaporates.

Signs and Symptoms: In exposures to high concentrations, dizziness, drowsiness, inco-ordination, confusion, nausea and vomiting, abdominal pains, hiccoughs, diplopia and dimness of vision are followed by delirium, convulsions and coma. Death may be immediate, but if the exposure is not fatal, recovery is usually slow, and degenerative changes in the central nervous system are not uncommon. The liver, kidneys, and bone marrow may be affected, with resulting acute nephritis and anaemia. Death may occur several days after exposure, resulting from degenerative changes in the heart, liver, and especially the kidneys. In repeated exposures to lower concentrations there is usually fatigue, loss of appetite, muscular weakness, drowsiness, and dimness of vision. After-effects are commonly the result of damage to the central nervous system, with visual changes and attacks of depression and other psychic disturbances being reported.

Recommended M.A.C.: 100 ppm for an 8-hour working period.

Fire Hazard: The minimum explosive limit is 8·2%, the maximum 19·7% by volume in air. Methyl chloride is classed as a combustible gas.

Methyl Formate

Formula: CH_3COOH.

Properties: A colourless, inflammable liquid having an agreeable, penetrating odour. Specific gravity: 0·950 times as heavy as water. Boiling point: 89°F. Quite volatile at ordinary room temperatures. Vapour density: 2·07 times heavier than air. Soluble in water, alcohol and ether.

Uses and Occurrences: Used in organic synthesis and in the manufacture of fumigants, larvicides and war gases.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Methyl formate and the higher formate esters are irritant to the eyes and mucous membranes of the upper respiratory tract. They exert a narcotic effect in high concentrations. No symptoms occur in humans exposed for one minute to concentrations of 0·15% methyl formate, but concentrations of 1·5% to 2·5% are dangerous for even short exposures. Exposure of cats to concentrations of 0·15% methyl formate for one hour causes irritation of the mucous membranes, giddiness and occasionally death from pulmonary oedema. Higher concentrations of about 1% produce more marked irritant effects, inco-ordination, retching, narcosis and death. In addition to the pulmonary oedema, a hyperaemia of the liver, kidneys, brain and adrenals is present in animals dying from acute exposures. The odour of the formate is easily detectable and its irritant effect is greater than that of the acetate.

Signs and Symptoms: Irritation of the eyes, lacrymation, irritation of the upper respiratory tract, coughing, difficulty in breathing, and, in exposure to high concentrations, dizziness, incoordination and possible pulmonary oedema.

Recommended M.A.C.: Suggested values range from 100 ppm to 500 ppm for repeated 8-hour exposures.

Fire Hazard: Flash point: −2° F. Minimum explosive limit: 5%; maximum explosive limit: 22·7% by volume, of the vapour in air. Class 1 flammable liquid.

Methylene Chloride (Dichloromethane; Methyl Dichloride)

Formula: CH_2Cl_2.

Properties: A colourless liquid having an odour like that of chloroform. Specific gravity: 1·336. Boiling point: 104°F.; is readily volatile at room temperature (1·8 times less volatile than ether). Vapour density: 2·93 times heavier than air. Soluble in alcohol, ether and other organic solvents; very slightly soluble in water.

Uses: Used as a solvent, as a degreasing and cleaning fluid, and in the manufacture of paint and lacquer removers.

Mode of Entry into Body: Through inhalation of the vapour. May be absorbed through the skin.

Physiological Action and Toxicity: The narcotic effect of methylene chloride is less than that of chloroform; its local irritant effect is somewhat stronger. The range between narcotic and fatal concentrations is less than that of chloroform. Exposure of cats to concentrations of 2·3% causes deep narcosis in 11 minutes; recovery following exposure to narcotic concentrations is usually complete, with no residual injury. With repeated daily exposure to lower concentrations, fatty infiltration of the liver and kidneys may occur though methylene chloride is generally regarded as being less active than chloroform and carbon tetrachloride in this respect. Methylene chloride decomposes in the presence of heat and moisture to form phosgene and hydrochloric acid.

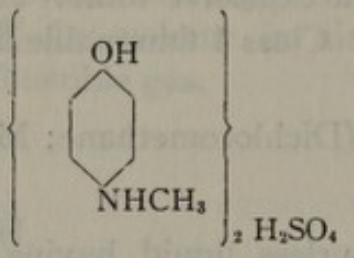
Signs and Symptoms: Headache, dizziness, nausea and vomiting, difficulty in breathing, stupor, slight irritation of the eyes, and loss of consciousness.

Recommended M.A.C.: 500 ppm for repeated 8-hour exposures.

Fire Hazard: None. Methylene chloride is practically non-inflammable. However, in the presence of heat and moisture it may decompose to form phosgene and hydrochloric acid.

Metol (Methyl-para-aminophenol Sulphate)

Formula:



Properties: A colourless, crystalline solid, soluble in water, but insoluble in alcohol and ether.

Uses: Is used as a photographic developer.

Mode of Entry into Body: Acts locally on the skin.

Physiological Action and Toxicity: Users of metol are commonly affected by eczematous lesions on the hands and arms. Occasionally there are ulcers on the skin and eyelids. The irritating substance may be an impurity, since metol is said to be non-irritating when chemically pure.

Recommended M.A.C.: No value has been set, since the substance has only a local action.

Fire Hazard: Metol has not been listed as a fire hazard.

Naphtha

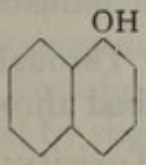
The term naphtha has been used to designate distillation products of both petroleum and coal-tar. Petroleum naphtha or V.M. & P. naphtha consists chiefly of straight-chain hydrocarbons of the paraffin series, and its effect on the body is similar to that of benzine or gasoline (see "Petroleum Hydrocarbons"). Stoddard solvent and mineral spirits may also be known by the name "Safety Solvent Naphtha," since the flash point of these materials is 100 to 110°F, as compared with the flash point of 20 to 45°F for V.M. & P. naphtha and benzine.

Solvent naphtha and high flash naphtha are coal-tar derivatives, and are composed chiefly of benzol and its homologues, toluol and xylol, or their alkyl derivatives. Solvent naphtha has a flash point of 78°F, high flash naphtha flashes above 100°F. Because of their possible benzol content, these naphthas may be potentially serious hazards to health. See "Benzol," "Toluol," "Xylol" and "Ethyl Benzene."

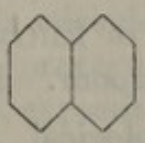
Recommended M.A.C. The recommended maximum allowable concentration of coal tar naptha for daily 8-hour exposures is 200 ppm.

Naphthols

Formulae:



Alpha-naphthol



Beta-naphthol

Properties: The naphthols are white, crystalline solids. The alpha-naphthol melts at 202°F and boils at 532°F; the beta compound melts at 251°F and boils at 545°F. Vapour density: 4·9 times heavier than air. Both are soluble in alcohol and ether, and are slightly soluble in water.

Uses and Occurrence: Used in organic synthesis and in the manufacture of dyes, pharmaceuticals and rubber.

Mode of Entry into Body: Through inhalation of the dust and vapour. May be absorbed through the skin.

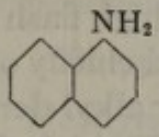
Physiological Action and Toxicity: The naphthols are thought to be more toxic than naphthalene, the beta-naphthol being slightly less toxic than the alpha derivative. They are irritant to the skin and mucous membranes and may cause dermatitis. It is reported that sufficient naphthol may be absorbed through the skin to cause irritation of the kidneys and injury to the cornea and lens of the eye.

Recommended M.A.C.: No values have been adopted.

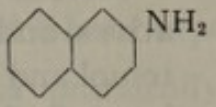
Fire Hazard: Beta-naphthol has a flash point of 307°F.

Naphthylamines (Aminonaphthalenes)

Formulae:



Alpha-naphthylamine



Beta-naphthylamine

Properties: In the pure state, both the naphthylamines are white crystals. The alpha compound melts at 122°F and boils at 574°F. Beta-naphthylamine melts at 234°F and boils at 561°F. They are slightly soluble in water but are readily soluble in alcohol and ether.

Uses and Occurrence: Occur in the manufacture of aniline dyes; used in organic synthesis, and to a small extent as an accelerator in the rubber industry.

Mode of Entry into Body: Through inhalation of the dust or vapour. May be absorbed through the skin.

Physiological Action and Toxicity: Though certain investigators have reported that the naphthylamines can cause symptoms similar to those produced by aniline, they are generally considered to be much less toxic in their systemic action than the benzol derivatives. Of the two naphthylamines, the alpha is reported to be less toxic than the beta compound. For over 50 years it has been known that aniline dye workers suffer from a higher incidence of benign

and malignant tumours of the bladder than do unexposed workers. Several aromatic amino compounds have been considered to be the causative agents, including aniline, para-toluidine, benzidine and the naphthylamines. It has been recently reported that neoplastic lesions of the bladder have been successfully reproduced in animals by the use of beta-naphthylamine, ortho-toluidine and benzidine. Recent clinical evidence also suggests that the responsible agents are beta-naphthylamine, benzidine and alpha-naphthylamine containing about 5% of the beta compound. Attempts to produce the lesion by the use of aniline have been unsuccessful.

Signs and Symptoms: The development of bladder tumour in workers exposed to aromatic amino compounds usually follows exposure of from 3 to 20 years or more. The first complaint is nearly always one of haematuria. There may be frequency of micturition and difficulty in voiding. Cystoscopic examination reveals a tumour which may be found to be a simple papilloma, an adenocarcinoma, or occasionally a sarcoma.

Recommended M.A.C.: No values have been suggested.

Fire Hazard: Alpha-naphthylamine has a flash point of 315°F.

Nickel and its Compounds

Symbol: Ni.

Nickel is a hard, silvery, inert metal which is used extensively in the manufacture of alloys of iron, chromium and tungsten. Its salts (sulphates, oxides, chlorides and nitrates) are used in the hydrogenation of oils, in nickel-plating, in chemistry and in certain other organic processes. In the production of nickel by the Mond process, exposure to carbon monoxide and to nickel carbonyl may occur. See "Nickel Carbonyl".

Mode of Entry into the Body: Through the inhalation of the dusts and fumes of nickel oxide and nickel salts. The salts act locally on the skin.

Physiological Action and Toxicity: Nickel and most salts of nickel are not generally considered to cause systemic poison-

ing. Ingestion of large doses of nickel (1 to 3 mgm per kg. of weight) has been shown to cause intestinal disorders, convulsions and asphyxia in dogs. Nickel has been found in the hair of persons exposed to nickel oxide dust, but no systemic effects which could be attributed to nickel alone have been reported. In 1938, the British described 10 cases of lung cancer and numerous cases of cancer of the nose and nasopharynx occurring in workers in a nickel refinery, The exact cause of the malignancies was never completely explained but arsenic was incriminated. The most common effect resulting from exposure to nickel compounds is the development of "nickel itch". This form of dermatitis occurs chiefly in persons doing nickel-plating. There is marked variation in individual susceptibility to the dermatitis. It occurs more frequently under conditions of high temperature and humidity when the skin is moist, and chiefly affects the hands and arms.

Signs and Symptoms: The dermatitis begins as a sensation of burning and itching, and is followed by the development of erythematous patches and papules which may vesiculate. Itching is frequently severe, and is often worse at night. The condition sometimes extends to involve the skin of the face, shoulders, chest and back. As a rule, recovery takes place within a week or two after removing the patient from exposure, but may be delayed for several months in certain cases. An obstinate eczema is reported to occur in susceptible employees.

Recommended M.A.C.: No limits have been suggested for nickel and its compounds, other than nickel carbonyl.

Fire Hazard. Nickel nitrate is an oxidizing material. In contact with organic materials it will cause violent combustion on ignition.

Nickel Carbonyl

Formula: $Ni(CO)_4$.

Properties: Nickel carbonyl is a colourless or pale, straw-coloured, liquid, having an odour resembling that of soot. It solidifies in the form of needle-like crystals at 73°F. It is volatile at ordinary room temperatures. At 140°F the

vapour decomposes with explosion. Soluble in alcohol, benzol and chloroform. Is decomposed by nitric acid.

Uses and Occurrence: Nickel carbonyl is formed by the passing of carbon monoxide over finely powdered nickel. This reaction is utilized in the purification of nickel by the Mond process. The process is enclosed, and exposure occurs when breaks occur in the pipes.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Nickel carbonyl is thought to decompose upon contact with the respiratory tissues with liberation of carbon monoxide and the formation of a fine deposit of a nickel compound over the surfaces of the alveoli. This material is irritant to the lungs, and causes pulmonary congestion and oedema. The material is absorbed into the blood and may cause pathological changes in other parts of the body. On autopsy, in addition to the pulmonary oedema, there are usually haemorrhagic foci in the lungs, in the white matter and especially the corpus callosum of the brain, in the medulla and cervical and upper dorsal regions of the spinal cord, and in the adrenals. Fatty degeneration of the liver, kidneys, heart and cerebral vessels is frequently present. Nickel carbonyl is said to be 5 to 10 times as toxic as carbon monoxide, and the lesions produced are believed to be due to the action of the nickel radicle rather than to the carbon monoxide. Exposure of cats and dogs for 1 hour to a concentration of 180 ppm is reported to cause death in 12 to 14 hours.

Signs and Symptoms: The inhalation of high concentrations of nickel carbonyl causes almost immediate symptoms of dizziness, headache, nausea and vomiting, difficulty in breathing and fever. On removal of the patient to open air the symptoms abate or disappear. Twelve to 36 hours later dyspnoea, cyanosis and epigastric and chest pain may develop. The patient may cough up blood-streaked sputum, and there may be signs in the chest of pulmonary oedema. Death, preceded by delirium, occurs usually between the 4th and 12th days.

Recommended M.A.C.: 1 ppm for repeated 8-hour exposures.

Fire hazard: The vapour is inflammable and explodes when heated to 140°F.

Nicotine and Tobacco

Properties: The alkaloid nicotine is a thick, colourless oily liquid, turning brown on exposure to air. It boils at 476°F, and is soluble in water, alcohol, ether and oils. Nicotine sulphate occurs as white crystals.

Uses and Occurrence: Nicotine sulphate is used as an insecticide for horticultural purposes, being sold usually in 40% concentration. Exposure occurs chiefly in the preparation of the insecticide and to some extent in the use of the material.

Mode of Entry into Body: Nicotine and the aqueous solutions of its salts are absorbed mainly through the skin. Tobacco dust may be inhaled.

Physiological Action and Toxicity: The most serious and most acute cases of poisoning resulting from exposure to tobacco or its alkaloid, nicotine, are those which follow the spilling of nicotine or concentrated aqueous solutions of nicotine sulphate on the skin. The symptoms which develop are due to the action of the alkaloid on the central and sympathetic nervous systems. At first there is excessive salivation and lacrymation, followed by headache, dizziness, numbness, sweating, disturbance of vision, contraction of the pupils, rapid respiration and pulse. A half-hour later the patient may develop nausea and vomiting, faintness, coldness of the extremities with rigors and increasing dyspnoea, and eventually collapse. Recovery usually takes place within 48 hours. Contact of the skin with the diluted solutions of nicotine insecticides, as prepared ready for spraying, appears to cause little trouble.

Exposure to tobacco dust, at one time thought to predispose to tuberculosis and to cause nervous disorders, including dimness of vision and toxic amblyopia, is generally considered to be relatively innocuous. It may cause irritation of the upper respiratory tract, but there is no definite proof of predisposition to tuberculosis. In the first cleaning of the tobacco leaves there may be some exposure to silica, but it is doubtful if cases of silicosis have ever developed in workmen employed at this job. New employees who are exposed to tobacco dust frequently complain of dizziness,

nausea and vomiting, an effect essentially the same as that produced by smoking in unaccustomed individuals. These complaints tend to disappear with further exposure.

Recommended M.A.C.: No values have been set for allowable concentrations of tobacco dust in air.

Nitric Acid (Azotic Acid; Hydrogen Nitrate)

Formula: HNO_3

Properties: A transparent, colourless or yellowish liquid, having a suffocating, pungent odour. "Fumes" on exposure to air. Specific Gravity: 1·530. Boiling Point: 186·8°F. An aqueous solution containing 68% hydrogen nitrate boils at 251°F., and is the "concentrated nitric acid" of commerce. Hydrogen nitrate is soluble in water and alcohol. It is strongly corrosive to most metals, and reacts readily with most chemicals.

Uses and Occurrence: Is used in organic synthesis, in the manufacture of dyes, drugs, explosives, celluloid, nitrates, in metallurgy and in photo-engraving and etching of metal.

Mode of Entry into Body: Through inhalation of the vapour. Nitric acid acts locally on the skin and mucous membranes.

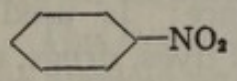
Physiological Action and Toxicity: The exact composition of the "fumes" or vapour produced by nitric acid depends upon such factors as temperature, humidity, and whether or not the acid comes in contact with other materials such as heavy metals or organic compounds. Depending upon these factors, the vapour will consist of a mixture of the various oxides of nitrogen and of nitric acid vapour. The effects produced by the oxides are discussed under the heading "Nitrous Fumes". Nitric acid vapour is irritant to the mucous membranes of the eyes and respiratory tract and to the skin. It is corrosive to the teeth. Because of its irritant properties, exposure to dangerous concentrations of the acid vapour seldom occur. It must be remembered however, that in the absence of symptoms of irritation, exposure to the more dangerous nitrogen oxides may occur. Where exposure to the vapour or "fumes" produced by nitric acid is suspected, the patient should be treated as a potential case of "nitrous fume" poisoning.

Signs and Symptoms: Nitric acid vapour causes irritation of the eyes and upper respiratory tract, with lachrymation, cough, choking, and difficulty in breathing. The teeth may show evidence of corrosion. Contact of the liquid with the eyes or skin may produce chemical burns. (See also "Nitrous Fumes").

Recommended M.A.C.: No values have been set for exposures to nitric acid vapour. The generally recommended M.A.C. for oxides of nitrogen is 10 to 25 ppm for repeated 8-hour exposures.

Fire Hazard: Strong nitric acid may cause spontaneous ignition when in contact with organic materials, such as sawdust, excelsior, paper and cotton waste.

Nitrobenzol (Essence of Mirbane; Oil of Mirbane; Nitrobenzene; Nitrobenzide; Mono-Nitrobenzol)

Formula: $C_6H_5NO_2$, or 

Properties: A yellowish, oily liquid having characteristic, pleasant odour.
Specific Gravity: 1·2 times heavier than water.
Boiling Point: 412°F.
Vapour Density: 4·25 times heavier than air.
Very slightly soluble in water; soluble in alcohol and ether.

Uses and Occurrence: Used in the manufacture of aniline and its derivatives, dyes, dust preventives and floor polishes, shoe polishes and lacquers.

Mode of Entry into Body: In industrial exposures, the most important route of entry is by absorption through the skin; cases of poisoning have been reported following inhalation of the vapour.

Physiological Action and Toxicity: In the nitration of benzol, exposure to nitrous fumes frequently occurs. The effect of mono-nitrobenzol on the body is similar to that of aniline, but nitrobenzol is usually regarded as being more toxic, especially in regard to its effect upon the nervous system. Exposure, particularly from spilling the liquid on the skin, is usually followed by a latent period of one to four hours

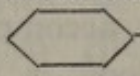
before the onset of symptoms. The patient then develops headache, followed by progressive depression of the central nervous system. The patient may become comatose and finally die of respiratory failure. A reduction in the oxygen-carrying power of the blood, with cyanosis, accompanies the effect on the nervous system. The blood is chocolate coloured; though it is generally believed that nitrobenzol causes the formation of methaemoglobin, many investigators feel that the decreased oxygen-carrying power of the blood is the result of other pigments formed in the blood by nitrobenzol. Where the exposure is less acute but prolonged, there is usually a reduction in the number of red blood cells and the haemoglobin; there may be signs of regeneration, with production of stippled cells, polychromasia, and nucleated red cells. There is no specific change in the white cell count or morphology. The excretion of nitrobenzol and its oxidation products, and of blood pigments, results in dark coloured urine. Animal experiments and human autopsy findings indicate that nitrobenzol may cause toxic hepatitis and nephrosis. Repeated exposure to the material may cause dermatitis of an eczematous type.

Signs and Symptoms: In cases of acute exposure, the picture is one of cyanosis and progressive depression of the central nervous system, starting with headache and sometimes followed by twitchings and tremors. There is frequently a burning sensation in the nose and throat, nausea and vomiting, gastro-intestinal upset, and a lowering of the body temperature. As the cyanosis becomes more severe there are complaints of dizziness, the pulse becomes irregular and feeble, and the patient lapses into coma. In cases of chronic exposure the commonest findings are anaemia, pallor, headache, weakness and fatigue and sometimes vomiting, soreness in the chest, tenderness of the tongue and palate, and irritation of the respiratory tract.

Recommended M.A.C.: 1 ppm for an 8-hour working day.

Fire Hazard: Flash point is 190°F.

Nitro Compounds of Benzol, Toluol and Xylol

When one of the hydrogen atoms in the benzol ring is replaced with a nitro ($-NO_2$) group, the compound formed is nitro-benzol (also called nitrobenzene and oil of mirbane),  , a yellowish liquid. Replacement by two nitro groups results in the formation of dinitrobenzol, which exists as three isomers, all of which are crystalline solids. If three nitro groups are introduced into the benzol ring, trinitrobenzol is formed. This compound has three crystalline isomers.

Mononitrobenzol is discussed under the heading "Nitrobenzol." The di- and trinitrobenzols, like the mononitrobenzol, are absorbed chiefly through the skin and through inhalation of the dust or vapour when these materials are heated. The dinitrobenzols are believed to be somewhat more toxic than the mononitrobenzol, and more toxic than aniline. The effect of di- and trinitrobenzol on the body is similar to that of aniline and mononitrobenzol, with reduction of the oxygen-carrying power of the blood and depression of the nervous system being responsible for most of the symptoms following acute exposure. Poisoning with the solid nitro compounds is usually slower and less severe, than is the case with the liquid nitro and amino benzols since absorption is less rapid. Thus, chronic poisoning occurs more frequently than acute, the picture in the chronic form being one of anaemia, moderate cyanosis, fatigue, slight dizziness, headache, insomnia and loss of weight. The urine is frequently dark in colour; the skin on the exposed parts is often yellowish-brown, and the hair yellowish-red. There may be irritation of the nose and throat, nausea and vomiting, sclerotic icterus, and complaints related to the nervous system. Prolonged chronic exposure may result in damage to the liver and kidneys, with production of acute yellow atrophy, toxic hepatitis, and fatty degeneration of the kidneys.

The introduction of one or more chlorine atoms into the nitrobenzol ring results in the formation of chloro-nitrobenzol compounds or nitrochlors. The chloro-mononitrobenzols have essentially the same toxic effect as nitrobenzol. The

chlorine derivatives of dinitrobenzol, on the other hand, while resembling dinitrobenzol in their systemic effect, are much more irritating to the skin. They act as direct irritants, and in addition may cause sensitivity.

From the industrial aspect the most important of the nitro derivatives of toluol and xylol is trinitrotoluol (T.N.T., trinitrotoluene) which is discussed separately.

Nitroglycerine (Trinitroglycerol)

Formula: CH_2NO_3 | $CHNO_3$ | CH_2NO_3

Properties: A pale yellow, oily liquid. Specific gravity: 1·6 times heavier than water. Vapour density: 7·84 times heavier than air. Soluble in alcohol and ether, slightly soluble in water.

Uses and Occurrence: Is used in the manufacture of dynamite and other explosives, and in medicine.

Mode of Entry into Body: Through inhalation of the vapour. May be absorbed through the skin.

Physiological Action and Toxicity: Absorption of nitroglycerine causes vasodilatation of the arterioles, with dramatic fall in blood pressure. The symptoms produced are due to this effect and disappear when the blood pressure returns to normal. In severe cases the patient may collapse, and several deaths have been attributed to nitrite effect. It has been suggested that repeated daily exposure may produce pathological alterations in the circulatory system, but the evidence supporting this is inconclusive.

Signs and Symptoms: The chief symptoms are headache of a severe, throbbing nature, flushing of the face, palpitation of the heart, tachycardia, nausea and vomiting, and decreased blood pressure. The symptoms usually disappear within several hours of leaving the exposure but may return on resuming work. There is fairly definite evidence that men working with nitrogylcerine establish a tolerance, but this disappears after a few days absence from work. Return

to exposure on Monday mornings results in the recurrence of symptoms in many workers. The nitrite effect is apparently enhanced by the drinking of alcohol.

Recommended M.A.C.: 0·5 ppm for repeated 8-hour exposures.

Fire Hazard: Dangerous. Explodes at a temperature of 500°F.

"Nitrous Fumes"

The term "nitrous fumes" is applied to a mixture of oxides of nitrogen. Strictly speaking, the term is a misnomer, since "nitrous fumes" are neither "nitrous" nor fumes.

There are six oxides of nitrogen: Nitrous oxide, or "laughing gas" (N_2O); nitric oxide (NO); nitrogen dioxide (NO_2); nitrogen trioxide (N_2O_3); nitrogen tetroxide (N_2O_4) and nitrogen pentoxide (N_2O_5). Nitrous oxide is a colourless, odourless, non-irritating, inert gas possessing weak narcotic properties when mixed with oxygen; when inhaled alone it is a simple asphyxiant. Nitric oxide does not exist in air, for in the presence of oxygen and moisture it is oxidized to the dioxide. Nitrogen dioxide is a dark brown gas which, when cooled, combines with itself, two molecules of NO_2 uniting to form one molecule of N_2O_4. The latter occurs as a colourless or pale yellow solid having a boiling point of 70·5°F. Nitrous fumes are chiefly a mixture of the dioxide and the tetroxide, its colour and the proportions of the constituents present depending upon the temperature. At 100°F the proportions are about 30% dioxide and 70% tetroxide. It is in this proportion that the gases (the reddish-brown nitrous fumes) act on the respiratory system.

Occurrence: Exposure to nitrous fumes may occur whenever nitric acid acts upon organic material, such as wood, sawdust and refuse; it occurs when nitric acid is heated, and when organic nitro compounds are burned, as for example celluloid, nitrocellulose (guncotton) and dynamite. The action of nitric acid upon metals, as in metal etching and pickling, liberates the fumes. In high-temperature welding, as with the oxyacetylene or electric torch, the nitrogen and oxygen of the air unite to form oxides of nitrogen. Exposure may also occur in many manufacturing processes where nitric acid is made or used.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: The oxides of nitrogen are fairly soluble in water, reacting with it in the presence of oxygen to form nitric and nitrous acids. This action takes place deep in the respiratory system. The acids formed are irritant, causing congestion of the throat and bronchi, and oedema of the lungs. The acids are neutralized by the alkalis present in the tissues, with the formation of nitrates and nitrites. The latter may cause some arterial dilatation, fall in blood pressure, headache and dizziness, and there may be some formation of methaemoglobin. However, the nitrite effect is of secondary importance.

Signs and Symptoms: Because of their relatively low solubility in water, the nitrogen oxides are only slightly irritant to the mucous membranes of the upper respiratory tract. Their warning power is therefore low, and dangerous amounts of the fumes may be breathed before the workman notices any real discomfort. Higher concentrations (60 to 150 ppm) cause immediate irritation of the nose and throat, with coughing and burning in the throat and chest. These symptoms often clear up on breathing fresh air, and the workman may feel well for several hours. Some 6 to 24 hours after exposure, he develops a sensation of tightness and burning in the chest, shortness of breath, sleeplessness and restlessness. Dyspnoea and air hunger may increase rapidly, with development of cyanosis and loss of conciousness, followed by death. In cases which recover from the pulmonary oedema, there is usually no permanent disability, but pneumonia may develop later. Concentrations of 100 to 150 ppm are dangerous for short exposures of 30 to 60 minutes. Concentrations of 200 to 700 ppm may be fatal after even very short exposures.

Continued exposure to low concentrations of the fumes, insufficient to cause pulmonary oedema, is said to result in chronic irritation of the respiratory tract, with cough, headache, loss of appetite, dyspepsia, corrosion of the teeth and gradual loss of strength.

Exposure to nitrous fumes is always potentially serious, and persons so exposed should be kept under close observation for at least 48 hours.

Recommended M.A.C.: 10 to 25 ppm for repeated 8-hour exposures.

Oxalic Acid

Formula: $\begin{matrix} COOH \\ | \\ COOH \end{matrix}$

Properties: Colourless, transparent crystals. Melting point: 368°F. Soluble in water, alcohol and ether.

Uses and Occurrence: Used as an oxidizing and bleaching agent for straw and leather, and as a cleaner for brick, stone and metal. It is used also by dry cleaners, dye and ink makers, engravers and lithographers, glycerine refiners and metal polish workers.

Mode of Entry into Body: Through inhalation of the dust or vapour and ingestion of the dust. May act locally on the skin.

Physiological Action and Toxicity: Acute oxalic poisoning results from ingestion of a solution of the acid. There is marked corrosion of the mouth, oesophagus and stomach, with symptoms of vomiting, burning abdominal pain, collapse and sometimes convulsions. Death may follow quickly. The systemic effects are attributed to the removal by the oxalic acid of the calcium in the blood. The renal tubules become obstructed by the insoluble calcium oxalate, and there is profound kidney disturbance. The inhalation of the dust or vapour may cause symptoms of irritation of the upper respiratory tract, gastro-intestinal disturbances, albuminuria, gradual loss of weight, increasing weakness and nervous system complaints. Oxalic acid has a caustic action on the skin and may cause dermatitis; a case of early gangrene of the fingers resembling that caused by phenol has been described.

Signs and Symptoms: The chief effects of inhalation of the dusts or vapour are irritation of the eyes and upper respiratory tract, ulceration of the mucous membrane of the nose and throat, epistaxis, headache, irritability and nervousness. More severe cases may show albuminuria, chronic cough, vomiting, pain in the back and gradual

emaciation and weakness. The skin lesions are characterized by cracking and fissuring of the skin and the development of slow-healing ulcers. The skin may be bluish in colour, and the nails brittle and yellow.

Recommended M.A.C.: No values have been adopted.

Ozone

Formula: O_3

Properties: A colourless or bluish gas having a characteristic odour. Vapour density: 1·658 times heavier than air. Soluble in water, oil of turpentine, and certain other organic materials.

Uses and Occurrence: Is produced by passing oxygen through the blue zone of high tension electrical discharges. It is used as an oxidizing and bleaching agent.

Mode of Entry into Body: Through inhalation of the gas.

Physiological Action and Toxicity: Ozone has a strong irritant action on the upper respiratory system. Concentrations of 0·015 parts of ozone per million parts of air produce a barely detectable odour. Concentrations of 1 ppm produce a disagreeable sulphur-like odour and may cause headache and irritation of the upper respiratory tract which disappear after leaving the exposure. Exposure of guinea pigs to higher concentrations may cause death from lung congestion and oedema. No systemic effects have been reported following industrial exposures.

Recommended M.A.C.: Ranges from 0·15 ppm to 1 ppm for repeated 8-hour exposures.

Fire Hazard: No fire hazard is mentioned in the literature. Though ozone is an oxidizing agent, it is probable that the concentrations reached in most exposures are so low that they do not constitute a fire hazard.

Paraldehyde: (Para Acetaldehyde; Paracetaldehyde; 2, 4, 6-Trimethyl-1, 3, 5, Trioxane)

Formula: $(CH_3CHO)_3$

Properties: Colourless liquid with a characteristic odour, is a polymerization product of acetaldehyde. Flammable. In the presence of acids, paraldehyde decomposes to form

acetaldehyde. Specific Gravity: 0·99. Boils at 255°F, and has a vapour density four and a half times that of air. Is about 12% soluble in water at 68°F, and is miscible with alcohol, chloroform, ether and most volatile oils.

Uses: Is used as a rubber accelerator and anti-oxidant. Is used in the manufacture of dyes and chemicals, paints and varnishes. Is a solvent for fats, oils, waxes, gums, resins, and cellulose derivatives.

Mode of Entry into Body: Through ingestion of the liquid.

Physiological Action and Toxicity: Hypnotic and analgesic. Has virtually no irritating properties. There is a wide range between the hypnotic dose and the toxic dose; 2 to 5 grams produces a soporific effect; recovery has been observed following the ingestion of 50 grams. Continued use of the drug has been known to result in habituation. There have been no cases reported of industrial poisoning.

Signs and Symptoms: Incoordination and drowsiness, followed by sleep. With larger doses, the pupils dilate, reflexes are lost, and patient lapses into coma. Pulse becomes weak, respirations shallow, followed by cyanosis. Death results from respiratory paralysis.

Recommended M.A.C.: No figure has been set since absorption of sufficient paraldehyde to cause acute poisoning can only occur through ingestion.

Fire Hazard: Flash point: 63°F. Minimum explosive limit is 1·3%, by volume, in air. It is a class 2 flammable liquid presenting the same hazard as methyl alcohol.

Pentanone (Methyl Propyl Ketone; Methyl Propanone)

Formula: $CH_3CO(CH_2)_2CH_3$

Properties: A colourless liquid having an aromatic odour. Inflammable.
Specific Gracity: 0·812 times as heavy as water.
Boiling Point: 216°F.
Vapour Density: 2·96 times heavier than air.
Is fairly volatile.
Only slightly soluble in water.

Uses and Occurrence: Is used as a solvent in the manufacture of lacquers, varnishes, paint removers, and artificial leather.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action: The irritant action of pentanone in relation to its narcotic action, is more marked than in the case of the lower ketones, acetone and butanone. Concentrations of 1,500 ppm of pentanone are irritant to the eyes and nose after several hours exposure. (*Cf.*, acetone, butanone.)

Signs and Symptoms: Lacrimation and burning of the eyes, irritation of the nose and throat.

Recommended M.A.C.: 200 ppm for an 8-hour working day.

Fire Hazard: Dangerous. Flash point: 45 to 60°F. Minimum explosive limit is 1·5%; maximum explosive limit 8%, by volume, of the vapour in air. Class 2 flammable liquid.

Petroleum Hydrocarbons

Petroleum is a thick, heavy, inflammable oily liquid, varying in colour from yellowish to dark reddish-brown or black. It usually shows a distinct greenish fluorescence.

The composition of the oils varies according to their place of origin. For the most part, they are mixtures of straight-chain hydrocarbons of the methane or paraffin series, but they also contain olefines, cycloparaffins and, in some oils, a variable amount of aromatic hydrocarbons such as benzol. Many oils also contain appreciable quantities of sulphur.

Separation of the constituents is effected by distillation. "Straight-run" distillates are produced by ordinary distillation, "cracked" distillates by a process involving the use of very high temperatures, under which the higher hydrocarbons are split into lower-boiling members. "Straight-run" distillates consist chiefly of saturated hydrocarbons of the paraffin series between pentane and octane. Distillates produced by "cracking" may contain up to 50% of unsaturated hydrocarbons (*e.g.*, olefines) and cycloparaffins. The fractions produced by the distillation of Texan, Californian and Mexican oils are rich in cycloparaffins, while those produced from Rumanian and Borneo oils may contain between 25 and 40% aromatic hydrocarbons.

The following table briefly summarizes the distillation fractions of petroleum:

Distillation Fractions of Petroleum

Name of Fraction	*Boiling Point °F*	*Main Constituents*	*Vapour Density*	*Flash Point °F*	*Explosive Limits % by vol.*	
					min.	max.
Petroleum Ether	70–140	Butane, Pentane, Hexane	2·5	–50	1·4	5·9
Petroleum Benzine	100–190	Pentane, Hexane, Heptane	.....	.....	....	.....
Naphtha, V.M. & P	212–320	Octane, Nonane	.....	20–45	1·2	6·0
Gasoline	100–400	Pentane, Hexane, Heptane, Octane, Nonane, Decane and some higher members	3–4	–50	1·3	6·0
Stoddard Solvent, Mineral Spirits	300–400	Nonane, Decane and some higher members	.....	100–110	1·1	6·0
Kerosene	300–400	Nonane, Decane and mainly higher members	.....	100–165	....	.....
Lubricating Oils, Paraffin Oil	above 570	Higher members	.....	above 300	....	.....

The term benzine has been applied to many distillation products of petroleum. For the most part, the name has been rather loosely used, with little regard to the exact chemical composition of the material so named. The application of the name has frequently extended to distillation fractions which might alternatively be designated as: petroleum ether, petroleum spirit, gasoline, V.M. & P. naphtha and benzoline.

Gasoline products also vary widely in composition, and may consist of butane, pentane, hexane and heptane in the case of "high test" gasolines, or higher members of the paraffin series may be present resulting in a "low test" product. In addition, modern gasolines are usually blended with other hydrocarbons and light hydrocarbons derived from coal. The coal derivatives are often quite toxic. "Anti-knock" agents such as tetraethyl lead, aniline and nitro-benzene are also present in most modern gasolines.

Uses and Occurence: In addition to their use as motor fuels, the distillation products of petroleum are widely used as solvents in many industries, such as the manufacture of paints, varnishes and lacquers, cements and rubber; in dry cleaning; in the extraction of fats; in degreasing operations and in colour printing.

Mode of Entry into Body: Through inhalation of the vapour. The liquid may be absorbed through the skin.

Physiological Action and Toxicity: The members of the paraffin series above butane are liquids. Their vapours and those of the olefine series, exert a narcotic action on the central nervous system. This effect is stronger in the higher members of both series, if comparison is made on a basis of weight. However, the boiling points increase and the volatilities decrease as one ascends either series, and homologues of the paraffin series higher than decane are not sufficiently volatile at ordinary temperatures to produce concentrations which are physiologically active. In addition to their narcotic action, the members between pentane and decane have an irritant effect on the nervous system, resulting in a state of mental excitement and muscular jactitation preceding and following the period of anaesthesia. There is only a narrow margin between the concentration which

produces anaesthesia and that which produces death. A concentration of 5000 ppm of pentane in air produces no symptoms when breathed for 10 minutes. A similar concentration of hexane causes dizziness after 10 minutes, and a comparable concentration of heptane produces marked dizziness, muscular incoordination and emotional disturbances after only 4 minutes inhalation. Concentrations of 1,250 to 2,500 ppm of gasoline produce slight symptoms after several hours, and 20,000 to 30,000 ppm may be fatal after even short exposures.

Following recovery from the acute effects, irritation of the kidneys and pneumonia may develop, and serious neurological sequelae (multiple sclerosis, retro-bulbar neuritis, encephalitis, epilepsy, vertigo and nystagmus) have been reported.

Repeated exposure to non-narcotic concentrations of benzine, gasoline and kerosene have been stated by some authorities to cause chronic poisoning, characterized by nervous and gastro-intestinal complaints and possibly blood changes. Other investigators feel that chronic poisoning is very rare, and only occurs in employees who are exposed to fairly high concentrations. Where complaints exist, they are frequently relieved by removal of the patient from exposure. Locally, benzine, gasoline and kerosene are irritant to the skin, and the vapours are irritant to the eyes and mucous membranes of the upper respiratory tract.

Signs and Symptoms: The signs and symptoms vary according to the duration and concentration of the exposure. Where the exposure has been severe, the worker becomes dizzy, then unconscious, and may develop convulsions. There may be muscular twitchings of the face and extremities, the pulse is rapid and thready, respirations slow, the mucous membranes cyanotic, and the skin cold and clammy as in collapse. If the patient recovers he may develop albuminuria and other signs of kidney involvement. Depending upon the duration of the cerebral depression, serious neurological after-effects may develop. In less severe exposures, the face is flushed and the picture may simulate inebriation (*e.g.*, naphtha jag) with hysterical laughter, staggering gait,

nausea and vomiting, headache, blurred vision and smarting of the eyes. Frequently the symptoms develop or become worse on removing the patient to the open air.

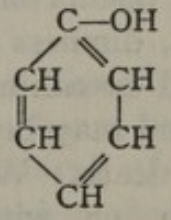
The symptoms of chronic poisoning described by some investigators include headache, dizziness, loss of appetite, insomnia, nervousness, weakness, pains in the legs, back and heart region, and shortness of breath; there may be pallor, decreased haemoglobin and red cell values, loss of weight and muscular twitchings. Workers handling the petroleum solvents may develop dermatitis.

Recommended M.A.C.: There is much variation in the literature for the concentrations deemed safe for repeated daily exposures. The more recent values suggested are listed in the table below.

Fire Hazard: Available data regarding the liquid members of the paraffin series are listed in the following table. For data regarding the distillation fractions of petroleum, see above.

Hydrocarbon	*Recommended M.A.C. ppm*	*Flash Point°F*	*Minimum Exp. Limit*	*Maximum Exp. Limit*
Pentane	1,000	−40	1·4%	8·0%
Hexane	500	−7	1·25%	7·0%
Heptane	500	25	1·0%	6·0%
Octane	500	56	0·84%	3·2%
Nonane	.....	88	0·74%	2·9%
Decane	.....	115	0·67%	2·6%
Naphtha (Petroleum)	500	.....	.....	.....
Gasoline	500	−50	1·3%	6·0%
Stoddard Solvent	500	100-110	1·1%	6·0%

Phenol (Carbolic Acid; Phenic Acid; Phenylic Acid; Phenyl Hydrate; Hydroxybenzene; Monohydroxybenzene)

Formula: C_6H_5OH or 

Properties: A white, crystalline solid, turning pink or red if impure or under the influence of light. Absorbs moisture

from the air and liquefies. Has a distinctive odour. Specific Gravity: 1·07. Melting Point: 108°F. Boiling Point: 358° F. Vapour Density: 3·24. Soluble in water, alcohol, ether, chloroform, glycerol, alkalis and oils.

Uses and Occurrence: Phenol is derived from coal-tar, either directly or by manufacture from benzol. It is used as an antiseptic and disinfectant, in the manufacture of varnish and paint removers, and in the manufacture of drugs, and of synthetic resins and plastics.

Mode of Entry into Body: Phenol is absorbed chiefly through the skin. Absorption may also occur through the lungs, either as a vapour, if the phenol has been heated, or in droplet or mist form.

Physiological Action and Toxicity: While phenol is not a serious hazard to health in industry, provided it is handled properly, improper handling may result in serious poisoning. Phenol has a corrosive action on skin and mucous membranes with which it comes in contact. In acute poisoning, however, the main effect is on the central nervous system. Absorption from spilling phenolic solutions on the skin may be very rapid, and death results from collapse within 30 minutes to several hours. Death has resulted from absorption of phenol through a skin area of 64 square inches. Where death is delayed, damage to the kidneys, liver, pancreas, spleen, and oedema of the lungs may result. Absorbed phenol is partly excreted by the kidneys, partly oxidized. Part of the excreted portion is combined with sulphuric and glycuronic acids, the remainder is excreted unchanged.

Signs and Symptoms: In acute poisoning the signs and symptoms develop rapidly, frequently within 15 to 20 minutes following spilling of phenol on the skin. Headache, dizziness, muscular weakness, dimness of vision, ringing in the ears, irregular and rapid breathing, weak pulse, and dyspnoea may all develop, and may be followed by loss of consciousness, collapse and death. When taken internally, there is also nausea, with or without vomiting, and severe abdominal pain. On the skin, the affected area is white wrinkled and softened, and there is usually no immediate

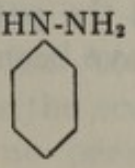
complaint of pain; later intense burning is felt, followed by local anaesthesia, and still later, by gangrene.

Chronic poisoning, following prolonged exposures to low concentrations of the vapour or mist, results in digestive disturbances (vomiting, difficulty in swallowing, excessive salivation, diarrhoea, loss of appetite), nervous disorders (headache, fainting, dizziness, mental disturbances) and skin eruptions. Chronic poisoning may terminate fatally in cases where there has been extensive damage to the kidneys or liver. Dermatitis resulting from contact with phenol or phenol-containing products is fairly common in industry.

Recommended M.A.C.: No maximum allowable limit has been set for phenol.

Fire Hazard: Phenol flashes at 185°F; no explosive limits have been set. Due to its high flash point, however, the danger of fire is ordinarily very small.

Phenyl Hydrazine

Formula: 

Properties: A light yellow oil having a specific gravity of 1·097. Solidifies as pale yellow crystals at 67°F. Boiling point: 470°F. Vapour density: 3·8 times heavier than air. Slightly soluble in hot water, freely soluble in alcohol and ether.

Uses and Occurrence: Used in the manufacture of dyes, in organic synthesis, and as an analytical reagent.

Mode of Entry into Body: Is absorbed chiefly through the skin.

Physiological Action and Toxicity: The ingestion or subcutaneous injection of phenyl hydrazine has been repeatedly shown to cause haemolysis of the red blood cells, an effect which has been utilized in the treatment of polycythaemia. The erythrocytes frequently contain Heinz bodies. Part of the haemoglobin is converted to methaemoglobin. Patho-

logical changes seen in animals include congestion of the spleen with hyperplasia of the reticulo-endothelial system, degeneration and necrosis of the liver cells with extensive pigmentation, early damage to the tubules of the kidneys with fatty changes in the cortical portion, and hyperplasia of the bone marrow. The most common effect of occupational exposure is the development of dermatitis which, in sensitized persons, may be quite severe. Systemic effects include anaemia and general weakness, gastro-intestinal disturbances and injury to the kidneys.

Signs and Symptoms: The systemic symptoms reported are fatigue, headache, dizziness, anorexia, gastritis, diarrhoea, weakness, pallor and dyspnoea. On examination anaemia, methaemoglobinaemia, and albumen, blood cells, casts and blood pigments in the urine may be found. Direct contact of the liquid with the skin may cause erythema, marked itching especially between the fingers, and papular and vesicular eruptions. In more severe cases there may be swelling and oedema of the hands, arms and eyelids in addition to the skin eruption. In sensitized individuals, it is frequently necessary to change the patient's occupation.

Recommended M.A.C.: No values have been set.

Phosgene (Carbonyl Chloride)

Formula: $COCl_2$

Properties: A colourless, non-irritating gas. Liquefies at 46·3°F.

Vapour Density: 3·4 times heavier than air.

Uses and Occurrence: Used in the manufacture of dyes and chlorine derivatives of organic compounds. Has been employed as a war gas. Phosgene may be formed by the decomposition of chlorinated hydrocarbons on contact with flames or with heated metal surfaces, as, for example, when carbon tetrachloride is used in fire extinguishers.

Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: In the presence of moisture, phosgene decomposes to form hydrochloric acid and carbon dioxide. This action takes place within the body, when the

gas reaches the bronchioles and the alveoli of the lungs. There is little irritant effect upon the upper respiratory tract, and the warning properties of the gas are therefore very slight. The liberation of hydrochloric acid in the lung tissues results in the development of pulmonary oedema, which may be followed by broncho-pneumonia, and occasionally lung abscess. There may be formation of haematin in the blood. Degenerative changes in the nerves have been reported as later sequelae. Concentrations of 3 to 5 ppm of phosgene in air cause irritation of the eyes and throat, with coughing. 25 ppm is dangerous for exposures lasting 30 to 60 minutes, and 50 ppm is rapidly fatal after even short exposures.

Signs and Symptoms: There may be no immediate warning that dangerous concentrations of the gas are being breathed. After a latent period of 2 to 24 hours the patient complains of burning in the throat and chest, shortness of breath and increasing dyspnoea. There may be moist râles in the chest. Where the exposure has been severe, the development of pulmonary oedema may be so rapid that the patient dies within 36 hours of exposure. In cases where the exposure has been less, pneumonia may develop several days after the occurrence of the accident. In patients who recover, no permanent residual disability is thought to occur.

Recommended M.A.C.: 1 ppm for an 8-hour working day.

Phosphine (Phosphoretted Hydrogen; Hydrogen Phosphide)

Formula: PH_3

Properties: A colourless gas having a characteristic garlic-like odour. Inflammable.
Vapour Density: 1·185 times heavier than air.
Soluble in alcohol and ether; slightly soluble in cold water.

Occurrence: Phosphine is liberated by the action of water and acids upon certain phosphides, such as calcium phosphide and aluminium phosphide. In the manufacture of calcium carbide and ferro-silicon, phosphides may be present as impurities, and cases of poisoning have resulted from

contact of water with these impurities. The acetylene generated by the action of water on impure calcium carbide may contain phosphine, and acetylene welders may be exposed.

Mode of Entry into Body: Through inhalation of the gas.

Physiological Action and Toxicity: Phosphine is a very toxic gas, but its action on the body has not been fully worked out. It appears to cause, chiefly, a depression of the central nervous system and irritation of the lungs; autopsy findings in human cases may be entirely negative, or there may be pulmonary oedema, dilation of the heart and hyperaemia of the visceral organs.

Signs and Symptoms: Inhalation of phosphine causes restlessness, followed by tremors, fatigue, slight drowsiness, nausea, vomiting, and, frequently, severe gastric pain and diarrhoea. There is often headache, thirst, dizziness, oppression in the chest and burning substernal pain; later the patient may become dyspnoeic and develop cough and sputum. Coma or convulsions may precede death. The symptoms frequently are confused with those of food poisoning. Mild cases recover without after-effects.

Chronic poisoning, characterized by anaemia, bronchitis, gastro-intestinal disturbances and visual, speech and motor disturbances, are said to result from continued exposure to very low concentrations. Exposure to 400 to 600 ppm for 30 to 60 minutes is dangerous; slight symptoms result from exposures to 7 ppm for periods of several hours.

Recommended M.A.C.: The allowable concentrations recommended by various authorities vary from 1 ppm to 0·05 ppm where the exposure is for periods of 8 hours.

Fire Hazard: Dangerous. Explodes spontaneously in contact with oxidizing agents.

Phosphorus and its Compounds

Symbol: P.

Properties: Phosphorus is a non-metallic element occurring in the pure state in three allotropic forms: (a) White or yellow phosphorus; this is a light yellow, waxlike, semi-transparent

solid, luminous in the dark, and having a distinctive, disagreeable odour. Melting Point: 112°F. Soluble in carbon disulphide, benzol and chloroform; slightly soluble in alcohol, ether and fixed oils. Insoluble in water. (b) Red phosphorus, a bright reddish-brown, odourless, amorphous powder which volatilizes on heating to 1338°F; soluble in absolute alcohol but insoluble in carbon disulphide. (c) Black phosphorus, an allotropic form which is of scientific interest only.

Uses and Occurrence: Yellow phosphorus is used in the manufacture of chemical smoke screens, incendiary bombs, in metallurgy and as a constituent of rat poison. Its use in the manufacture of matches and certain fireworks was discontinued some years ago because of the serious cases of poisoning which occurred amongst the employees. Red phosphorus is now used in place of the yellow in the manufacture of safety matches, and phosphorus sesquisulphide (P_4S_3) is used in the manufacture of "strike-anywhere" matches.

Other compounds of phosphorus encountered in industry are phosphine (PH_3), which is discussed separately; phosphoric anhydride (P_2O_5), used as a dehydrating agent in drying the gases used for filling electric light bulbs; and phosphorus oxychloride, trichloride and pentachloride, all of which are used as chlorinating agents.

Mode of Entry into Body: On exposure to air, yellow phosphorus ignites spontaneously to form oxides and acids of phosphorus, which may be inhaled. Poisoning can occur from ingestion of pastes and other materials containing yellow phosphorus, but most cases of industrial poisoning are believed to have been due to inhalation of the fumes.

Physiological Action and Toxicity: Acute phosphorus poisoning usually follows ingestion of yellow phosphorus. The average fatal dose is considered to be of the order of 100 mgm., though 15 mgm may be severely toxic. In fatal cases, death commonly occurs in 4 to 5 days and the cause of death is in most instances due to fatty degeneration of the liver. Focal necrosis of the brain and damage to the kidneys have also been reported. Ingestion of acutely toxic doses in industry is very unlikely to occur.

Chronic poisoning following exposure to the fumes of yellow phosphorus is characterized by the involvement of the osseous system. Most typical of this involvement is the necrosis of the mandible and/or maxilla ("phossy jaw"). However, other bones may be involved. There is first a generalized reaction of the periosteum, producing a hyperostosis of the entire skeleton; this is followed by a resorption of the calcium salts, leading eventually to bone atrophy. Fractures of the weight-bearing bones may occur. The exact nature of the changes which take place in the production of phossy jaw is not clear. The presence of carious teeth and diseased gums seems to predispose to the condition, but it has developed in edentulous persons and in individuals having healthy teeth and gums. The mandible is more commonly involved than the maxilla. Usually, there are several years of exposure before the development of symptoms, but cases have been reported in workers who have had only 2 or 3 months' exposure. The condition frequently results in gross deformity of the face, due to the formation of sequestra and the need for surgical removal of large portions of the mandible or maxilla. Death may occur from secondary infection. Other systemic effects of chronic poisoning include anaemia and leucopaenia. No definite disease of the liver has been described.

Red phosphorus is considered to be relatively non-toxic. The occasional case of injury reported following exposure to this material is thought to be due to the presence of yellow phosphorus as an impurity in the red variety.

Phosphoric anhydride has not been reported as causing any ill effects. The oxychloride, trichloride and pentachloride of phosphorus are irritant to the eyes and upper respiratory tract, and it has been suggested that they may cause pulmonary oedema. The sesquisulphide may cause dermatitis, with development of erythema and vesicles and pustules. Yellow phosphorus may cause thermal burns of the skin and conjunctivae.

Signs and Symptoms: In acute poisoning the patient commonly develops nausea and vomiting and severe abdominal pain within a few hours of ingesting the yellow phosphorus. The vomitus may have a garlic-like odour, and be luminous

in the dark. Symptoms may disappear for 24 to 36 hours, then reappear. Diarrhoea and abdominal tenderness develop, and jaundice may occur. In non-fatal cases the condition progresses for 5 to 15 days, after which recovery occurs.

The first symptoms occurring in chronic poisoning are pain in the jaw, toothache, swelling and ulceration of the gums with a fetid discharge, and pain in the joints. The teeth may loosen. There may be loss of appetite, indefinite digestive complaints and loss of weight.

Recommended M.A.C.: The generally recommended M.A.C.'s for phosphorus and its compounds are: Yellow phosphorus, 1 mgm per 10 cubic metres of air. Phosphorus pentachloride, 10 mgm per 10 cubic metres of air. Phosphorus pentasulphide, 10 mgm per 10 cubic metres of air. Phosphorus trichloride, 0·5 ppm.

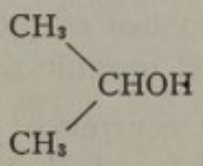
Fire Hazard: The yellow phosphorus ignites spontaneously on contact with air. Red phosphorus ignites at a temperature of 392°F. The sesquisulphide is ignited by friction and is highly flammable.

Propyl Alcohol (Propanol)

Formulae: The propyl alcohols are:

(1) Normal propyl alcohol (propanol−1) $CH_3CH_2CH_2OH$.

(2) Isopropyl alcohol (isopropanol; secondary propyl alcohol; dimethyl carbinol)



Properties: Both propyl alcohols are clear liquids. Inflammable. Soluble in water, alcohol and ether.

	Boiling Point	*Vapour Density*
Normal Propyl Alcohol.....	207°F	2·07
Isopropyl Alcohol.....	181°F	2·07

Uses and Occurrence: Propyl alcohol is not as widely used in industry as are the lower alcohols. It is employed as a solvent for dopes, waxes, oils, resins and plastics, lacquers and solvent mixtures.

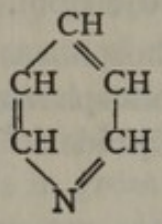
Mode of Entry into Body: Through inhalation of the vapour.

Physiological Action and Toxicity: Though possessing narcotic and local irritant properties, propyl alcohol causes little trouble in industrial practice, since it is not readily volatile. No cases of industrial poisoning have been reported.

Recommended M.A.C.: A concentration of 400 ppm is believed to be safe for an 8-hour working exposure.

Fire Hazard: Dangerous. Flash points are 59°F for normal propyl alcohol and 53°F for isopropyl alcohol. The minimum explosive limit for both isomers is 2·5%, by volume, of the vapour in air. Class 2 flammable liquids.

Pyridine

Formula: C_5H_5N or 

Properties: A colourless or slightly yellow volatile liquid, having a bitter taste. Specific gravity: 0·9893. Boiling point: 240°F. Vapour density: 2·73 times heavier than air. The commercial product has a boiling range of from 201°F to 320°F, and contains impurities, chiefly higher homologues of pyridine. Pyridine is soluble in water and in most organic solvents and fatty oils.

Uses and Occurrence: Used in the dyeing industry, as a denaturant for alcohol, and in the manufacture of chemicals and explosives, lacquers, polishes and rubber.

Mode of Entry into Body: Through inhalation of the vapour. The liquid may be irritant to the skin.

Physiological Action and Toxicity: Pyridine is irritant to the mucous membranes of the eyes and upper respiratory tract. It is narcotic in high concentrations and may cause partial loss of consciousness. Recovery from such exposures is usually complete, with no residual injury. The narcotic dose has not been determined. Repeated exposure to low concentrations has been reported to cause disturbances of

the central nervous system and gastro-intestinal complaints, but these effects may be due to impurities present in the commercial product.

Signs and Symptoms: Irritation of the eyes, lacrimation, cough, dyspnoea, headache, dizziness, gastro-intestinal complaints and dermatitis.

Recommended: M.A.C.: No values have been suggested.

Fire Hazard: Flash point: 68°F. Minimum explosive limit: 1·8%, maximum explosive limit: 12·4%, by volume, of the vapour in air.

Selenium and its Compounds

Symbol: Se.

Selenium occurs as a steel-gray, crystalline solid or as dark red crystals or powder. Chemically, it resembles sulphur in many ways. It melts at 423°F. It is used in the manufacture of red glass, in the vulcanizing of rubber, in electrical rectifiers and photo-electric cells, as a stabilizer in lubricating oils, in making alloys of steel and brass, as a pigment in paints and inks, in the manufacture of plastics and in the chemical industry. Selenium oxychloride ($SeOCl_2$) is an oxidizing and chlorinating agent. Exposure to selenium may occur in the refining of copper, lead and zinc, in the roasting of pyrites and in making lime and cement.

Mode of Entry into Body: Through inhalation or ingestion of the dust; through inhalation of the gas, hydrogen selenide (SeH_2); by absorption through the skin.

Physiological Action and Toxicity: The physiological action of selenium closely resembles that of tellurium. Part of the absorbed selenium combines with organic radicles, possibly the methyl group. The resultant compound is excreted in the breath and in the sweat, imparting a "garlic-like" odour to both. The sulphide (SeS) is highly toxic, a concentration of 5 mgm per cubic metre producing intolerable eye and nasal irritation. Experimental evidence suggests that the sulphide may cause a chemical pneumonitis which may persist in a sub-acute form, and fatty changes in the liver which may improve later. A human case, attributed to

hydrogen selenide poisoning, demonstrated "garlic" breath, symptoms of acute irritation of the eyes and upper respiratory tract, a purple rash on both cheeks, dyspnoea and abundant râles over both lungs. Thrombophlebitis and a definite cardiac lesion developed some weeks later.

Signs and Symptoms: The usual signs and symptoms occurring in exposed workers are pallor, irritation of the eyes, lachrymation, sore throat, bronchial irritation, cough, garlicky odour of the breath and sweat (the secretion of which is not inhibited as in tellurium poisoning), metallic taste, nervousness, occasionally pain in the lumbar region, and night sweats. Selenium may be detected in the urine. Certain selenium compounds, such as selenious acid and some of its salts, may cause dermatitis. The oxychloride, when in contact with the skin, causes acute painful burns which may be slow to heal.

Recommended M.A.C.: 1 mgm per 10 cubic metres of air.

Fire Hazard: Selenium is flammable, forming selenium dioxide.

Silver and its Compounds

Symbol: Ag.

Uses and Occurrence: Silver is used in the manufacture of electrical contacts and relays, aviation bearings, and as a lining for special tanks, pipes and containers. Silver nitrate, a white, crystalline solid, is used as a laboratory reagent, and in the silvering of mirrors, in silver plating and in photography.

Mode of Entry into Body: Through inhalation and ingestion of particles of silver or its salts; through ingestion of solutions of silver salts; by the introduction of solid particles through breaks in the skin.

Physiological Action and Toxicity: The absorption of silver compounds into the circulation and the subsequent deposition of the reduced silver in various tissues of the body may result in the production of a generalized greyish pigmentation of the skin and mucous membranes—a condition known as argyria. The introduction of fine particles of silver through breaks in the skin produces a local pigmentation at the site of the injury.

Generalized argyria, rarely seen at the present time, was not infrequent in the past. The condition developed slowly, usually after some 2 to 25 years of exposure. Pigmentation was noticeable first in conjunctivae, and later in the mucous membranes of the mouth and gums and in the skin. There were no constitutional symptoms, and no physical disability. Persons exhibiting the condition, and who subsequently died from unrelated disease, showed, on autopsy, a deposition of silver in the blood vessel walls, kidneys, testes, pituitary, choroid plexus, and mucous membrane of the nose, maxillary antra, trachea and bronchi. Once deposited, there is no known method by which the silver can be eliminated; the pigmentation is permanent.

Recommended M.A.C.: No limits have been set.

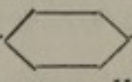
Fire Hazard: Silver nitrate is an oxidizing material. Silver picrate and fulminate are explosives.

Solder

Ordinary solder is an alloy of equal parts of lead and tin. Other solders contain these metals in different proportions; occasionally cadmium, copper, silver and bismuth may be added. Certain solders are formed so as to contain the flux as a waxy core. In other cases, a grease-like flux is used and is applied to those parts of the metal which are to be united.

The health hazards associated with soldering operations are lead fumes, cadmium fumes, hydrogen chloride gas (which is evolved from the acid flux) and zinc chloride burns. Lead, cadmium and hydrogen chloride are discussed separately. Zinc chloride is a caustic; it is used in soldering in the form of a "butter" or grease. The spattering of droplets of this material on the skin of the face or hands or in the eyes results in painful burns.

Styrene (Monomeric Styrene; Phenyl Ethylene; Styrol; Styrolene; Cinnamene; Cinnamol; Cinnamenol; Vinyl Benzene)

Formula: $C_6H_5CHCH_2$ or 

Properties: A colourless or yellowish, oily liquid, having a pungent, aromatic odour. Boils at 293°F. Is insoluble in water, but soluble in alcohol, ether, and most organic

solvents. Polymerizes easily at high temperatures to form a glassy product.

Uses: In manufacture of synthetic rubber and resins; plasticizer for nitrocellulose.

Mode of Entry into Body: As a vapour, through inhalation.

Physiological Action and Toxicity: The vapour, in concentrations of 200 ppm, possesses a disagreeable, gassy odour. In concentrations of around 500 ppm, the vapour causes irritation of the eyes, nose and throat, lassitude and fatigue. Slightly higher concentrations cause drowsiness and light narcosis. Concentrations of 2,000 to 10,000 ppm are fatal to animals for short exposures, causing death through action on the central nervous system. Lower concentrations may cause death through lung irritation and pneumonia. The action of styrene on the skin resembles that of toluol and benzol, consisting essentially of a de-fatting action. Excreted chiefly as benzoic acid, a small percentage as organic sulphate.

Signs and Symptoms: Lacrimation and irritation of the eyes, irritation of the nose and throat; occasionally there is mild conjunctivitis, coughing. Lassitude and fatigue are common complaints. With higher concentrations, drowsiness and incoordination are seen. The skin may appear dry, rough and cracked.

Recommended M.A.C.: The recommended maximum allowable concentration for an 8-hour exposure has been set at 400 ppm. A concentration of 200 ppm produces definite warning, in the form of a disagreeable, gassy odour; some authorities recently have favoured this latter concentration as a desirable maximum allowable concentration.

Fire Hazard: Flash point: 88°F. Minimum explosive limit: 1·1%. Maximum explosive limit: 6·1%, by volume. Class 3 flammable liquid.

Sulphur Chloride (Sulphur Monochloride, Sulphur Subchloride)

Formula: S_2Cl_2

Properties: An amber or reddish-yellow fuming liquid having an irritating, penetrating, odour. Specific gravity of the liquid is 1·687. Vapour density is 3·31 times that of air.

Boiling point is 276°F. Soluble in alcohol, ether, benzol, carbon disulphide and amyl acetate. Decomposes on contact with water, to form fumes of sulphur dioxide, sulphur, and hydrochloric acid. The sulphur dioxide in turn forms sulphurous and sulphuric acids.

Uses and Occurrence: Used as a solvent, fumigant, and in the manufacture of chemicals.

Mode of Entry into Body: Through inhalation of the fumes formed on contact with water.

Physiological Action and Toxicity: Under ordinary conditions, decomposition is considered to be nearly complete before inhalation takes place. Sulphur chloride thus has an irritant action on the eyes and mucous membranes of the upper respiratory tract. In atmospheres of low humidity, it is possible that undecomposed vapours may be inhaled, and would act on the deeper parts of the respiratory tract, thereby increasing the toxicity of the compound.

Signs and Symptoms: Smarting, burning and weeping of the eyes, cough and irritation of the nose and throat.

Recommended M.A.C.: 1 ppm for an 8-hour working period.

Fire Hazard: Flash point is 245°F. No explosive limits are given in the literature, but under ordinary conditions and temperatures sulphur chloride would not constitute a fire hazard.

Sulphur Dioxide (Sulphurous Acid Anhydride)

Formula: SO_2.

Properties: A colourless gas at ordinary temperatures. Below 14°F it is a clear liquid. Odour is quite pungent and suffocating.
Vapour Density: 2·23 times as heavy as air.
Boiling Point: 14°F.
Soluble in water. On contact with water part of the SO_2 is oxidized, and sulphurous and sulphuric acids are formed.

Uses and Occurrence: Is used and occurs widely in industry.

Mode of Entry into Body: As a vapour, through inhalation. Acts locally on the skin.

Physiological Action and Toxicity: A concentration of 3 to 5 ppm produces a detectable odour. Higher concentrations (of 20 ppm) produce immediate irritation of the eyes and coughing. Concentrations above this level cannot be tolerated and continued exposure may result in oedema of the larynx and lungs, and broncho-pneumonia. Repeated exposure to the gas results in nasopharyngitis, bronchitis, shortness of breath on exertion, increased fatigue and alteration of the sense of taste and smell. Brief exposures, lasting up to $\frac{1}{2}$ hour, to concentrations up to 100 ppm do not produce disability, but concentrations of 400 to 500 ppm are dangerous, even for short exposures.

Signs and Symptoms: Irritation and burning of the eyes, with weeping and redness, cough and irritation of the nose and throat. If exposure is continued, dyspnoea and respiratory distress may develop.

Recommended M.A.C.: 10 ppm for an 8-hour work period.

Sulphur Trioxide (Sulphuric Anhydride)

Formula: SO_3

Properties: A white or colourless gas or liquid which evolves dense white fumes on exposure to air. Boils at 112·4°F. The gas is 2·8 times heavier than air. Soluble in water, forming sulphuric acid.

Uses and Occurrence: Is formed when concentrated sulphuric acid is heated. The gas does not constitute as serious a hazard in industry as does sulphur dioxide.

Mode of Entry into Body: As a mist or vapour, through inhalation. Acts locally on the skin.

Physiological Action and Toxicity: Is a primary irritant, affecting the eyes and mucous membranes of the respiratory tract. Is similar to sulphur dioxide in its action. It is probably as toxic as the latter substance, but does not constitute as great a hazard, in practice.

Signs and Symptoms: Irritation and burning of the eyes, lacrimation and redness; cough and irritation of the nose and throat.

Recommended M.A.C.: 2 ppm for an 8-hour working period.

Sulphuric Acid (Oil of Vitriol)

Formula: H_2SO_4.

Properties: A heavy, oily, strongly corrosive liquid. Is colourless to dark brown, depending upon purity. Has a pungent, irritating odour.

Specific Gravity: 1·835 times as heavy as water.

Boiling Point: 518°F.

Volatility at room temperatures is very slight.

Fuming sulphuric acid, or oleum, is a solution of sulphur trioxide in concentrated sulphuric acid. The percentage of dissolved sulphur trioxide may run as high as 30 to 65% SO_3. Fuming sulphuric acid produces fumes of SO_3 on contact with air.

Uses and Occurrence: Sulphuric acid is so widely used in industry that any brief list of its uses would be of little practical purpose.

Mode of Entry into Body: Through inhalation of the mist. Acts locally on the skin.

Physiological Action and Toxicity: The unheated or non-reacting acid presents little hazard to health. When allowed to act upon metal, bubbles of hydrogen are evolved which carry a mist of sulphuric acid droplets into the air. Such mists are irritating to the skin and to the mucous membranes of the eyes and respiratory tract. No toxic concentrations have been determined, but it is unlikely that sulphuric acid is more toxic than hydrochloric acid. The fumes of sulphur trioxide which are given off by oleum are toxic. See reference to "Sulphur Trioxide."

Signs and Symptoms: The presence of sulphuric acid mist in the air causes a stinging sensation on the exposed skin. Heavier concentrations produce coughing.

Recommended M.A.C.: The value generally accepted is 50 mgm. per 10 cubic metres of air.

Fire Hazard: In contact with water, fuming sulphuric acid may react explosively.

Synthetic Rubber

Artificial or synthetic rubber manufacture is a relatively new industry, owing much of its development to the shortage

of natural rubber during the war. Several different kinds are now being produced commercially. The materials are made by a variety of methods, for example (1) by chemical reaction, (2) by polymerization, and (3) by formulation. Substances which are used in their production include: acrylonitrile or vinyl cyanide, butadiene or vinyl ethylene, monomeric styrene or phenyl ethylene, isoprene, and chlorobutadiene or "chloroprene." These substances are discussed individually.

In addition to the above chemicals, the manufacture of synthetic rubber requires the use of many of the solvents, anti-oxidants and accelerators used in the production of natural rubber products. Many of these materials are toxic or are skin irritants. Though less toxic compounds have been substituted where possible, many materials which possess some degree of toxicity are still widely used.

Tar and Pitch

Tar is a dark-coloured, bituminous substance, liquid or semi-liquid at room temperature, possessing a characteristic "tarry" odour. It is insoluble in water but soluble in most organic solvents. On distillation a residue of pitch remains. The name "tar" has also been applied to the residue remaining after the partial distillation of coal (coal-tar) and to Barbados petroleum. The name "pitch" has been applied to bituminous substances obtained from the earth, to the residues left after the distillation and oxidation of mineral oils, to the residues left after the distillation of coal-tar and wood-tar, and to mixtures of various materials which have the general properties of pitch (*e.g.*, roofing pitch).

Mode of Entry into Body: Through inhalation of the fumes produced by heating. The materials may act locally on the skin.

Physiological Action and Toxicity: Inhalation of the "fumes" or vapour produced by heating tar or pitch are said to cause headache, dizziness, nausea, loss of appetite, cough and bronchitis, irritation of the respiratory tract, diarrhoea, albuminuria, and suppression of urine. Contact of the

vapour with the eyes may cause conjunctivitis and, in cases of prolonged exposure, corneal ulceration. The most important effect of exposure to these substances is the development of various skin conditions, such as acne, "tar itch", ulcers, papillomas, etc. After many years of exposure, malignant skin lesions may appear. "Mule spinners' carcinoma of the scrotum", "paraffin cancer", "chimney-sweeps' cancer" and cancer occurring in persons applying creosote to timber or working with certain mineral oils and coal residues have long been recognized as occupational in origin. The carcinogenic substances present in the tar and pitch have not been completely isolated; although 3, 4-benzpyrene is known to be an active constituent of tar, other unidentified carcinogens are also known to be present. Certain petroleums are much more active than others in causing skin cancer. The final products of distillation, particularly the fraction between 700°F and 825°F, have the greatest carcinogenic action.

Recommended M.A.C.: No values have been suggested.

Fire Hazard: Pine tar has a flash point of 130°F, pine pitch has a flash point of 285°F. Tars and pitches are combustible, but do not constitute a fire hazard at ordinary temperatures.

Tellurium and its Compounds

Symbol: Te.

Pure tellurium occurs as a dark gray powder which melts at 845°F. Chemically, it is a member of the sulphur group. It is used in the manufacture of coloured glass, in rubber compounding, and in the hardening of lead, bronze, tin, silver and magnesium. Addition of tellurium to iron increases the chill depth, hardness and machineability. Tellurium is an impurity in certain copper and lead ores. When these are refined, gaseous hydrogen telluride (TeH_2) may be liberated along with dusts containing tellurium oxide (TeO).

Mode of Entry into Body: Through inhalation of the dusts and the gas, hydrogen telluride; through ingestion of the dusts; and by absorption through the skin.

Physiological Action and Toxicity: Tellurium is readily soluble in the tissue fluids and is carried to and deposited in all tissues of the body. Part of the tellurium is converted to methyl telluride and is excreted in the sweat and breath, imparting to these a characteristic, garlic-like odour. Acute poisoning induced in animals by subcutaneous injection causes haemorrhagic gastroenteritis, and symptoms of restlessness, tremor, diminished reflexes, paralysis, convulsions, somnolence and unconsciousness, followed by death from asphyxia.

Industrial poisoning is uncommon, and no fatalities have so far been reported.

Signs and Symptoms: In industrial poisoning, the symptoms reported include "garlic" odour of the breath, metallic taste, dryness of the mouth, foul-smelling sweat, suppression of sweating, dry, itching skin, loss of appetite, nausea and vomiting, and somnolence and apathy.

Recommended M.A.C.: A concentration of 1 mgm per 10 cubic metres of air has been suggested as safe for repeated 8-hour exposures. However, to avoid the development of foul-smelling sweat and breath, a concentration of 0·1 mgm per 10 cubic metres has been recommended by some authorities.

Tetrachloroethane (Acetylene Tetrachloride; 1, 1, 2, 2—Tetrachloroethane)

Formula: $CHCl_2$–$CHCl_2$

Properties: A colourless, corrosive liquid having a chloroform-like odour. Non-inflammable. Specific Gravity: 1·59. Boils at 296°F. Is much less volatile (about 33 times less volatile) than ether. The vapour is 5·8 times heavier than air. Is soluble in alcohol and ether and other organic solvents and oils; is practically insoluble in water. In the presence of moisture, it slowly liberates hydrochloric acid. Is slightly corrosive to iron, lead, copper and nickel; is strongly corrosive to aluminum.

Uses and Occurrence: Used chiefly as a solvent, in degreasing and dry-cleaning. Is a solvent for gums, resins, waxes and cellulose compounds. Used in paint and varnish removers.

Mode of Entry into Body: Through inhalation of the vapour, and by absorption through the skin.

Physiological Action and Toxicity: Tetrachloroethane is generally considered the most toxic of the chlorinated hydrocarbons. It has a fairly strong irritant action on the mucous membranes of the eyes and upper respiratory tract; a concentration of 3 ppm produces a detectable odour. There is thus an initial warning effect. Its narcotic action is stronger than that of chloroform, but because of its low volatility, narcosis is less severe and much less common in industrial tetrachloroethane poisoning than is the case with other chlorinated hydrocarbons. The toxic action of tetrachloroethane is chiefly on the liver, where it produces acute yellow atrophy and cirrhosis. Fatty degeneration of the kidneys and heart, haemorrhage into the lungs and serous membranes, and oedema of the brain have also been found in fatal cases. Some reports indicate a toxic action on the central nervous system, with changes in the brain and in the peripheral nerves. The effect on the blood is one of haemolysis, with appearance of young cells in the circulation and a monocytosis. Due to its solvent action on the natural skin oils, dermatitis is not uncommon.

Signs and Symptoms: The initial symptoms resulting from exposure to the vapour are lacrimation, salivation and irritation of the nose and throat. Continued exposure to high concentrations results in restlessness, dizziness, nausea and vomiting and narcosis. The latter, however, is rare in industry. More commonly, the exposure is less severe, and the complaints are vague and referable to the digestive and nervous systems. The patient's complaints gradually progress to serious illness, with development of a toxic jaundice, liver tenderness, etc., and possibly albuminuria and oedema of the legs. With serious liver damage the jaundice increases and toxic symptoms appear, with somnolence, delirium, convulsions and coma usually preceding death.

Recommended M.A.C.: The maximum allowable concentration recommended by most authorities is 5 to 10 ppm for an 8-hour working period.

Tetrachloroethylene (Carbon Bichloride; Tetrachloroethene; Perchloroethylene)

Formula: $CCl_2=CCl_2$.

Properties: A colourless liquid, having an ether-like odour. Non-inflammable. Specific Gravity: 1·62. Boils at 250°F; is slightly volatile at ordinary temperatures, the vapour being 5·7 times heavier than air. Is practically insoluble in water, but completely soluble in most organic solvents.

Uses: As a solvent in dry-cleaning and degreasing, and for cellulose compounds.

Physiological Action and Toxicity: Is a strong narcotic, of about the same order as chloroform. Its toxic effect is low, and it is generally considered to be the least toxic of the chlorinated hydrocarbons. It is used fairly widely in industry, but to date there have been few reports of its effect on humans under industrial exposure.

Recommended M.A.C.: 100 ppm for an 8-hour working period.

Tetryl (Trinitrophenylmethylnitramine; "C. E. Powder")

Properties: A crystalline powder, insoluble in water, but soluble in many organic solvents.

Uses: A military propellant and explosive.

Mode of Entry into Body: Through inhalation and ingestion of the dust. May be absorbed through the skin.

Physiological Action and Toxicity: The chief effect produced by exposure to tetryl is the development of dermatitis. Conjunctivitis may be caused by rubbing the eyes with contaminated hands or through exposure to air-borne dust. Iridocyclitis and keratitis have developed as a sequel to the conjunctivitis. Some authorities consider that tetryl may be a cause of tracheitis and asthma. Sensitization which frequently occurs as a result of exposure to tetryl may play a part in all these conditions. Tetryl workers may develop gastro-intestinal symptoms, though these complaints are more common among T.N.T. workers. Anaemia has been reported to occur frequently.

Signs and Symptoms: Erythema of the face and neck; dry, scaly or patchy, papular rash, oedema of the eyelids, conjunctivitis, epistaxis, ulceration of the nasal mucosa, anorexia, nausea, flatulence and abdominal cramps, dry cough, headache, irritability, sleeplessness and lassitude are all frequent complaints. Anaemia may be found on blood examination.

Recommended M.A.C.: 15 mgm per 10 cubic metres of air.

Fire Hazard: Tetryl is classed as a dangerous fire hazard.

Thallium and its Compounds

Symbol: Tl.

Thallium is a bluish-white, lead-like metal having a melting point of 576°F. It is of use chiefly for the production of its salts. The most important of these are thallium peroxide (Tl_2O_3), thallium chloride (TlCl) and thallium sulphide (Tl_2S). These salts are insoluble or only slightly soluble in water. The chloride is a white, crystalline powder, the sulphide a blue-black crystalline or amorphous powder, and the peroxide a brown or dark red powder.

Uses and Occurrence: The salts of thallium are used in the manufacture of pigments and dyes, artificial gems, depilatory preparations, disinfectants and rat poisons. Thallium is used in the manufacture of electric light bulb filaments, and certain kinds of glass.

Mode of Entry into Body: Through inhalation and ingestion of the dust. Thallium preparations may be ingested accidentally.

Physiological Action and Toxicity: Acute poisoning follows the ingestion of toxic quantities of the depilatory, or accidental or suicidal ingestion of rat poison. Children tolerate 8 mgm of thallium acetate per kilogram of weight, but adults and adolescents do not. Acute poisoning results in swelling of the feet and legs, arthralgia, vomiting, insomnia, hyperaesthesia and paraesthesia of the hands and feet, mental confusion, polyneuritis with severe pains in the legs and loins, partial paralysis of the legs with reaction of degeneration, angina-like pains, nephritis, wasting and weakness,

and lymphocytosis and eosinophilia. About the 18th day, complete loss of the hair of the body and head occurs. The symptoms resemble those produced by sympathectomy. Death may result.

Industrial poisoning is reported to have caused discolouration of the hair, which later falls out, joint pains, loss of appetite, fatigue, severe crampy pain in the calves of the legs, albuminuria, eosinophilia and lymphocytosis, and optic neuritis followed by atrophy. One case developed a posterior adhesion of the iris. Cases of industrial poisoning are rare.

Recommended M.A.C.: No values have been adopted.

Thorium and its Compounds

Symbol: Th.

Thorium is a heavy, gray, radioactive metal. The chief uses of the metal or its salts are for the manufacture of incandescent gas mantles, the preparation of certain radio-opaque pharmaceuticals, as catalytic agents, and, to a small extent, in making ceramics.

Physiological Action and Toxicity: The hazard associated with the use of radioactive materials is discussed in Section F. As systemic poisons, thorium and its salts are only slightly toxic. Damage to the liver and spleen and fibrotic changes in the regional lymph nodes have been reported following the clinical or experimental use of thorium dioxide. Exposure to thorium salts in industry, on the other hand, has been associated only with the production of local effects on the skin. In the manufacture of gas mantles, workers are exposed to thorium nitrate and to small percentages of cerium nitrate, sulphuric acid and beryllium nitrate. Cases of diffuse dermatitis, occasionally with swelling of the hands and fissuring of the skin, have been reported to occur in these workers.

Mesothorium was one of three radioactive ingredients used in the painting of luminous watch and clock dials in the United States between 1917 and 1924. The ingestion of the luminous paint through the habit of pointing or tipping the brushes with the lips resulted in 20 deaths, due in 5 cases to

bone sarcoma, in 2 cases to necrosis of the jaw, and in 13 cases to aplastic anaemia. Only luminizers who had been exposed for periods of more than 1 or 2 years were affected, and in many the disease did not become apparent until 5 or 6 years after they had ceased work.

Recommended M.A.C.: The recommended maximum allowable concentrations for gamma radiation and for thoron are given in Section F, under "Radium and other Radioactive Substances."

Fire Hazard: Thorium nitrate is an oxidizing material, and in contact with organic substances may cause violent combustion when ignited.

Tin and its Compounds

Symbol: Sn.

Neither tin nor any of its compounds are generally regarded as being industrial poisons. Stannic bromide (tin tetrabromide) is a white crystalline solid; stannic chloride (tin tetrachloride) is a clear, caustic liquid. Both these materials produce irritating fumes on exposure to air. The tetrachloride is used in the textile industry, in making ceramics, and as a bleaching agent in the sugar industry.

Physiological Action and Toxicity: Concentrations of 1 part of stannic tetrachloride vapour per million parts of air are fatal to mice in 10 minutes. Concentrations of 3 ppm are well tolerated by guinea pigs over several months of daily exposure. In humans, the inhalation of air containing 8·5 ppm causes irritation of the nose and throat with coughing. One man, who was exposed to fumes of tin tetrachloride over a period of years, was reported to have complaints of chilliness, particularly in the morning, pain in the throat, a sensation of heaviness in the epigastrium, anaemia, and traces of tin in the urine and faeces.

Recommended M.A.C.: No limits have been suggested.

Fire Hazard: Stannous oxide, when heated, decomposes with combustion. Stannic phosphide is a combustible.

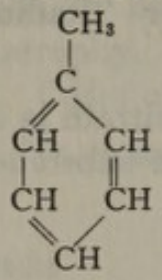
Titanium Dioxide

Formula: TiO_2

Titanium dioxide is a white powder used as a substitute for white lead in the manufacture of paint. No ill effects have been reported as the result of its use in industry.

Toluol (Toulene; Methyl Benzene, Methyl Benzol; Phenyl Methane)

Formula: $CH_3C_6H_5$ or



Properties: A colourless liquid having a benzol-like odour. Inflammable. Specific Gravity: 0·866; Boils at 232°F, and is less volatile than benzol, the evaporation rate being about 1/6 that of ethyl ether. The vapour is 3·14 times as heavy as air. Toluol is soluble in alcohol, benzol and ether; is insoluble in water.

Uses and Occurrence: Used as a solvent, and in the manufacture of explosives (T.N.T.), dyes, and other organic chemicals.

Mode of Entry into Body: Toluol, and the other homologue of benzol, xylol, are absorbed into the body as a vapour, through inhalation.

Physiological Action and Toxicity: Toluol and xylol are, like benzol, derived from coal tar, and commercial grades usually contain small amounts of benzol as an impurity. Though there is some disagreement among authorities, it is generally agreed that the order of toxicity is benzol, toluol, xylol. Weight for weight, toluol and xylol are stronger narcotics than benzol, but because of their lower volatility they are much less toxic than the latter substance in industry. Acute poisoning, resulting from exposures to high concentrations of the vapours, are rare with toluol, and even more rare with xylol. Inhalation of 200 ppm of toluol for 8 hours causes definite impairment of co-ordination and reaction time; with higher concentrations (up to 800 ppm) these effects are increased and are observed in a shorter time. In the few cases of acute toluol poisoning reported, the effect has been

that of a narcotic, the workman passing through a stage of intoxication into one of coma. Recovery following removal from exposure has been the rule. The occasional report of chronic poisoning describes an anaemia and leucopaenia, with biopsy showing a bone marrow hypoplasia. These effects, however, occur much less commonly than in employees working with benzol, and are not as severe.

Signs and Symptoms: Exposure to concentrations up to 200 ppm produces few symptoms. At 200 to 500 ppm, headache, nausea, loss of appetite, a bad taste, lassitude, impairment of coordination and reaction time are reported, but are not usually accompanied by any laboratory or physical findings of significance. With higher concentrations, the above complaints are increased, and in addition, anaemia and/or leucopaenia may be found in rare cases. A relative lymphocytosis is not uncommon.

Recommended M.A.C.: The recommended maximum allowable concentration for both toluol and xylol is 200 ppm for an 8-hour working period.

Fire Hazard: Toluol flashes at 45°F, has a minimum explosive limit of 1·27% and a maximum explosive limit of 7% by volume. Class 2 flammable liquid. Xylol flashes at 75°F, has a minimum explosive limit of 1% and a maximum explosive limit of 5·3% by volume. Class 3 flammable liquid.

Trichloroethylene (Acetylene Trichloride; "Tri"; Chlorylene)

Formula: $CHCl=CCl_2$

Properties: A colourless liquid, having a chloroform-like odour. Non-inflammable. Specific Gravity: 1·47. Boiling Point: 188°F. Is 3·8 times less volatile than ether, the vapour being 4·5 times heavier than air. Insoluble in water, but is completely soluble in all the common organic solvents. When exposed to open flame, it decomposes to form phosgene and hydrochloric acid.

Uses: Is widely used in industry as a solvent and degreaser; is used in the manufacture of drugs, dyes, and organic chemicals, paints and varnishes, rubber and soaps. Is used as an insecticide.

Mode of Entrance into Body: Occurs chiefly through inhalation of the vapour; it can be absorbed through the skin.

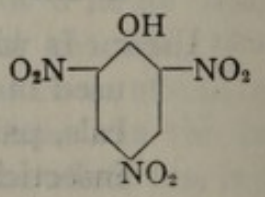
Physiological Action and Toxicity: Trichloroethylene has a narcotic action stronger than that of chloroform, and the picture in acute poisoning is one of narcosis, which, if severe, may result in unconsciousness and death from respiratory collapse. Recovery from acute poisoning usually occurs without lasting injury. Recently, trichloroethylene has been stated to have an effect on the heart similar to that of chloroform, with production of tachycardia and possible ventricular fibrillation, in exposures to high concentrations. The question of chronic poisoning by trichloroethylene is debatable at present. German reports indicate that the central nervous system in particular may be damaged, and damage to the liver, spleen and kidneys has also been reported. Experience in this country, in England and in the United States has, for the most part, indicated that organic injury rarely results from industrial exposures, though toxic jaundice and liver hypertrophy have been mentioned. Industrial poisoning in this country is usually of the narcotic type. Certainly trichloroethylene does not have the toxic effects of carbon tetrachloride.

Signs and Symptoms: Headache, dizziness, confusion and fainting are the usual complaints where concentrations are low. Higher concentrations produce unconsciousness; on recovery the patient exhibits post-anaesthesia retching and nausea. In contact with the skin, the natural oils are removed, and dermatitis may result.

Recommended M.A.C.: Most authorities recommend a maximum allowable concentration of 100 to 200 ppm for an 8-hour working day.

Trinitrophenol (Picric Acid)

Formula: Trinitrophenol exists as four isomers, the most important being the 2,4,6-trinitrophenol,



which melts at 251°F. The solubility of the four isomers varies considerably.

Uses and Occurrence: Military explosive. Used in medicine as a germicide and in the manufacture of dyes.

Mode of Entry into Body: Through inhalation and ingestion of the dust and vapour. May be absorbed through the skin.

Physiological Action and Toxicity: The most important effect of trinitrophenol is the development of dermatitis. Its systemic toxicity is low, but a few cases of poisoning resulting from industrial exposure have been reported. Some cases showed symptoms of a gastro-intestinal character while others indicated the development of a neuritis. Ingestion of the substance may cause damage to the kidneys.

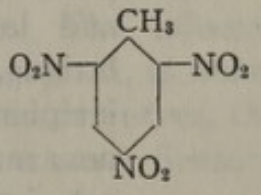
Signs and Symptoms: Dermatitis, irritation of the eyes, ulceration of the cornea, irritation of the respiratory tract with sneezing, rhinitis and chronic cough, yellow discolouration of the skin and hair, dizziness, bitter taste, nausea, vomiting and diarrhoea, epigastric pain and urinary symptoms have been reported.

Recommended M.A.C.: No limits have been adopted.

Fire Hazard: The 2,4,6-trinitrophenol explodes above 572°F.

Trinitrotoluol (Trinitrotoluene; T.N.T.)

Formula: There are 5 isomers of trinitrotoluene, of which the most important is the 2,4,6-trinitrotoluol,



Properties: When pure, trinitrotoluol occurs as pale yellow crystals. The 2,4,6-isomer melts at 178°F. It is slightly soluble in hot water, more soluble in alcohol, and very soluble in ether. The other 4 isomers also occur as crystalline solids. Their melting points vary. T.N.T. as used in warfare is composed chiefly of the 2,4,6-isomer.

Uses: Explosive.

Mode of Entry into Body: Chiefly by absorption through the skin. Absorption also occurs through inhalation and ingestion of the dusts and vapour.

Physiological Action and Toxicity: Trinitrotoluol is reduced in the body and excreted in the urine and bile, partly in conjugation with glycuronic acid. The skin, hair and nails may be coloured yellow. The irritant properties of the dust may cause irritation of the nose and throat and may produce dermatitis affecting the exposed surfaces most frequently. Gastro-intestinal complaints are common. In severe cases of poisoning, the liver, bone marrow and vascular endothelium are injured and there may be evidence of damage to the kidneys. The pathological findings reported include toxic hepatitis, acute yellow atrophy, focal necrosis and cirrhosis of the liver, hypoplasia or hyperplasia of the bone marrow, multiple petechial haemorrhages in many of the organs and in the skin, and toxic nephritis. Injury to the kidney is the most common cause of death, though aplastic anaemia may be the cause in rare cases. There is strong evidence to indicate that trinitrololuol exerts a haemolytic effect on the red blood cells.

Signs and Symptoms: Dermatitis usually occurs in the absence of any general complaints. It may take the form of tiny vesicles accompanied by itching and resembles the id reaction of epidermophytosis. In severe cases the skin may exfoliate. In other individuals, an erythema may develop, usually patchy and irregular in shape, progressing to form papules and later, vesicles. Complaints of lassitude, weakness, fatigue, and anorexia are probably the first symptoms of incipient systemic poisoning. Epigastric and liver tenderness may be present. The lips and ear lobes have a characteristic bluish colour, the exact cause of which is not entirely clear. A monocytosis may be present. The urine of T.N.T. workers is almost invariably of a dark "tea" colour. The development of jaundice is a serious sign of liver damage and may precede death from toxic hepatitis. Occasionally purpura develops, and more rarely an aplastic anaemia which is usually fatal. Examination of the urine reveals the presence of excretion products of T.N.T.

Recommended M.A.C.: 15 mgm per 10 cubic metres of air.

Fire Hazard: The 2,4,6-isomer explodes on heating to 536°F.

Tri-Ortho-Cresyl Phosphate

Formula: $PO–(OC_4H_6CH_3)_3$.

Properties: Tricresyl phosphate occurs as three isomers. The para-isomer is a crystalline solid, the ortho- and meta- forms are oily, nearly colourless liquids having a slightly pungent odour. The specific gravity of the liquids is about 1·165, and the boiling point around 770°F. Their volatility at ordinary room temperatures is very low. All the isomers are insoluble in water, but miscible with all common solvents, thinners and vegetable oils.

Uses and Occurrence: As a plasticizer in the manufacture of plastic materials, lacquers, coating materials; used in waterproofing and fireproofing compositions.

Mode of Entry into Body: Chiefly by absorption through the skin. Possibly by inhalation of the vapour when the material is heated.

Physiological Action and Toxicity: Most of the cases of tri-ortho-cresyl phosphate poisoning have followed its ingestion. In 1930, some 15,000 persons were affected in the United States, and of these, 10 died. The responsible material was found to be an alcoholic drink known as Jamaica ginger, or "jake". This beverage had been adulterated with about 2% of tri-ortho-cresyl phosphate. The affected persons developed a polyneuritis, which progressed, in many cases, with degeneration of the peripheral motor nerves, the anterior horn cells and the pyramidal tracts. Sensory changes were absent. Since 1930 there have been several other outbreaks of poisoning following ingestion of the material. Recently 3 cases of polyneuritis occurring in England in connection with the manufacture of the tri-ortho-cresyl phosphate have been reported. Absorption was probably through the respiratory tract, though there may have been some absorption through the skin. All three men made a good recovery.

From ingestion experiments with cockerels, it appears that tri-ortho-cresyl phosphate is more toxic than the meta-form, and much more so than tri-para-cresyl phosphate or triphenyl phosphate.

Signs and Symptoms: Irrespective of whether absorption has been by ingestion or by inhalation or skin absorption, the history is usually one of early, transient gastro-intestinal upset, with nausea, vomiting, diarrhoea and abdominal pain. These clear up, and are followed in 1 to 3 weeks by soreness of the lower leg muscles, "numbness" of the toes and fingers, and a few days later by weakness of the toes and bilateral foot-drop. After another week or so, weakness of the fingers and bilateral wrist-drop follow. There are no sensory changes. Recovery is slow, and the degree of residual paralysis depends upon the extent of damage to the nervous system. Many cases recover completely.

Recommended M.A.C.: No value has been set.

Fire Hazard: Flash point: 460°F.

Turpentine

Formula: $C_{10}H_{16}$

Properties: Turpentine is a volatile oil obtained from various species of pine. It is a mixture of terpenes, all of which have the formula $C_{10}H_{16}$; some terpenes are straight-chained hydrocarbons, others are cyclic. There are several grades of turpentine, depending upon the method of production. Gum spirit of turpentine is produced by the distillation of pine gum; wood or distilled turpentine is produced by the destructive distillation of pine wood, roots and brush, and contains small amounts of methyl alcohol, formaldehyde, phenols and pyridines.

Gum spirit turpentine has a specific gravity of 0·863 to 0·875, a boiling range of 311°F to 356°F, and is miscible with alcohol and most oils. Practically insoluble in water.

Uses and Occurrence: Turpentine is an excellent solvent for fats, resins, rubber and asphalt. It is used chiefly in the manufacture of paints, rubber, and polishing and cleaning

preparations. It is also used in the textile industry for cleaning fabrics, and in the printing industry for cleaning type and printing rolls.

Of recent years, the increasing cost of turpentine has resulted in a wider use of substitutes, which for the most part, are petroleum distillation products having a boiling range between 282°F and 426°F. Mixtures of natural turpentine and petroleum products are commonly used.

Mode of Entry into Body: Through inhalation of the vapour and by absorption through the skin. The liquid acts locally on the skin.

Physiological Action and Toxicity: The inhalation of turpentine vapour in concentrations of 750 to 1,000 ppm for periods of several hours causes irritation of the mucous membranes of the eyes and upper respiratory tract. There may also be symptoms of irritation of the central nervous system, followed by depression. With exposure to higher concentrations, the effects upon the nervous system become more pronounced. Damage to the kidneys has been reported following long periods of exposure to the vapour. Several cases of apparent injury to the kidney have also been attributed to absorption of turpentine through the skin. Death has been reported in several cases following the inhalation of the vapour in closed containers and after the ingestion of 6 ounces of the fluid. Turpentine is eliminated to some extent through the lungs, but the major portion is excreted through the kidneys, partly unchanged and partly combined with glycuronic acid. After absorption of turpentine, the urine may take on an odour resembling violets.

Locally, turpentine is irritant to the skin, causing erythema which may be followed by blistering. Certain individuals acquire a sensitivity to turpentine or to impurities present in it. Wood turpentine appears to be a more frequent cause of dermatitis than the gum spirit.

Signs and Symptoms: Irritation and burning of the eyes, lacrymation, irritation of the throat, cough, headache, dizziness, nausea and acceleration of respiration and pulse.

Vomiting and abdominal pain have been reported, but in most instances these complaints occurred in painters, and

may have been due to absorption of lead. Haematuria, with slight pain on micturition, has occurred following periods of long exposure to the vapour and after application of the liquid to the skin. Contact of the liquid or vapour with the skin may produce erythema which may be followed by blistering. Chronic eczematous conditions may develop in persons who have had many years exposure to turpentine.

Recommended M.A.C.: 100 to 200 ppm., for an 8-hour working day.

Fire Hazard: Flash point: 95°F. Is a class 3 flammable liquid. Minimum explosive limit: 0·8% by volume, of the vapour in air.

Type Metal

Type metals are of four grades, but all are alloys of lead, antimony and tin. The proportions of each metal vary in the different grades, the lead content varying between 75% and 95% of the total content, the antimony constituting between 2% and 18%, and the tin between 3% and 7%.

The hazard involved in the making of type metal is, of course, chiefly that of lead poisoning. During the re-melting of the metal, fumes of lead oxide are evolved. There is also some exposure to fumes of antimony oxide. For details of lead and antimony poisoning, refer to "Lead" and "Antimony".

Uranium and its Compounds

Symbol: U.

Uranium is a heavy, radioactive metal. Though formerly of little general industrial importance, with the recent advances in the field of atomic energy, considerable attention has been focused upon the possible toxicity of this element.

Physiological Action and Toxicity: The high incidence of pulmonary carcinoma occurring in European miners of uranium and other radioactive ores has been mentioned in Section F, under "Radium and other Radioactive Substances." Though there is some circumstantial evidence that radioactive materials such as uranium may cause lung cancer, there is as yet no conclusive proof.

As a systemic poison, uranium is considered to be one of the most toxic of the metals. Most of the evidence is experimental, however, since there are but few reports in the literature dealing with the toxic effects of uranium on humans. Administration of the soluble uranium nitrate to animals has resulted in toxic nephritis, albuminuria, glycosuria, gastro-intestinal disorders, degenerative changes in the liver and disturbances of the central nervous system. In man, exposure to uranium salts has been reported to cause purpura of the legs, leucopaenia and anaemia, though industrial exposure in Canada to the sulphate indicates that this salt has very low toxicity.

Uranium hexafluoride fumes on exposure to air, and vapourizes readily at ordinary temperatures. It is highly corrosive to glass and metals, and carbonizes some organic substances. Though no cases of illness due to this material have been reported, one might expect exposure to the fumes to cause a chemical pneumonitis with possibly additional toxic effects.

Recommended M.A.C.: No values have been recommended.

Fire Hazard: Uranium nitrate is an oxidizing material, and in contact with organic substances may cause violent combustion when ignited.

Vanadium and its Compounds

Symbol: V.

Vanadium is a light grey, crystalline metal. It is used as a catalyst, and in the manufacture of special alloy steels which retain their hardness when heated. Exposure occurs in the extraction of the ore and in the preparation of various vanadium compounds.

Mode of Entry into Body: Through inhalation and ingestion of the dusts.

Physiological Action and Toxicity: Vanadium compounds act chiefly as irritants to the conjunctivae and respiratory tract. There is still some controversy as to the effects of industrial exposure on other systems of the body.

The first report of vanadium poisoning in humans described rather widespread systemic effects, consisting of polycythaemia which was followed by red blood cell destruction and anaemia, loss of appetite, pallor and emaciation, albuminuria and haematuria, gastro-intestinal disorders, nervous complaints and cough, sometimes severe enough to cause haemoptysis. More recent reports describe symptoms which, for the most part, are restricted to the conjunctivae and respiratory system, no evidence being found of disturbances of the gastro-intestinal tract, kidneys, blood or central nervous system. Though certain workers believe that it is only the pentoxide which is harmful, other investigators have found that patronite dust (chiefly vanadium sulphide) is quite toxic to animals, causing acute pulmonary oedema.

Signs and Symptoms: Pallor, greenish-black discolouration of the tongue, paroxysmal cough, conjunctivitis, dyspnoea and pain in the chest, bronchitis, râles and rhonchi, bronchospasm, tremor of the fingers and arms, radiographic reticulation.

Recommended M.A.C.: No values have been set.

Vinyl Chloride (Chloroethylene; Chloroethene)

Formula: $CH_2=CHCl$.

Properties: A colourless gas, having an odour similar to that of ethyl chloride. Below 7°F it is a colourless liquid. Vapour density: 2·15 times heavier than air. Soluble in most organic solvents.

Uses and Occurrence: Used as a refrigerant, in organic synthesis, and in the manufacture of plastics.

Mode of Entry into Body: Through inhalation of the gas.

Physiological Action and Toxicity: Inhalation of vinyl chloride causes only slight irritating effects. The gas is narcotic, but is less powerful as an anaesthetic than chloroform. There is a wide range between the narcotic concentration and that causing death. Concentrations above 12%, by volume, are dangerous and may cause death through respiratory paralysis. Recovery from acute exposures is complete with no residual injury. The toxicity is low for repeated

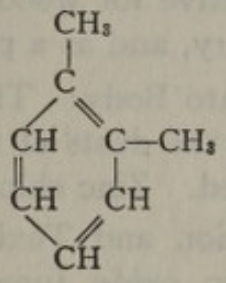
exposure to sub-narcotic concentrations, and liver and kidney damage have not been reported in experimental animals. Vinyl chloride is considered to be one of the least dangerous of the chlorinated hydrocarbons, no cases of industrial poisoning having been reported.

Signs and Symptoms: Dizziness, confusion, headache and, in exposure to high concentrations, stupor and unconsciousness.

Recommended M.A.C.: Several authorities have recommended that repeated daily exposure be limited to concentrations below 500 to 1,000 ppm.

Fire Hazard: Flash point: below 20°F; is a combustible gas.

Xylol (Dimethyl Benzene; Dimethyl Benzol; Xylene)

Formula: $C_6H_4(CH_3)_2$ or 

Properties: A clear, colourless liquid, having an aromatic, characteristic odour. Inflammable. Occurs as the ortho (formula above), meta and para forms. The ortho compound has a specific gravity of 0·876, boils at 291°F, and has a vapour density of 3·66. The physical and chemical constants of the meta and para compounds are very similar to those of the ortho-xylol. All forms are less volatile than benzol. Xylol is soluble in alcohol, ether and benzol; is insoluble in water.

Uses: Xylol is used as a solvent for rubber cements and in the manufacture of paints and varnishes. Used in microscopy, and in the manufacture of organic chemicals.

Physiological action and toxicity, signs and symptoms, and fire hazard are discussed under "Toluol".

Zinc and its Compounds

Symbol: Zn.

Zinc is a silvery white metal which melts at 786°F and boils at 1685°F. When heated to near its boiling point, white fumes of zinc oxide (ZnO) are given off.

Uses and Occurrence: Metallic zinc is used in the galvanizing of sheet iron, to prevent rusting; in the manufacture of non-ferrous alloys, such as brass and bronze; and in production of zinc electrodes, battery zincs, fuses, engravers plates, and other articles of common use. Exposure to zinc oxide fumes occurs in zinc smelters and welders, galvanizers, brass and bronze founders and men reclaiming junk metal. Zinc chloride ($ZnCl_2$) is a white granular, deliquescent powder used as a wood preservative and catalyst, and in the preparation of soldering fluxes and glass etching compositions; it is also used in the textile industry. Zinc chromate ($ZnCrO_4$), a yellow crystalline powder, is used as a pigment. Zinc sulphate ($ZnSO_4$), a colourless, crystalline solid, is used as a preservative for wood and hides, as a mordant in the textile industry, and as a pigment in paints.

Mode of Entry into Body: Through inhalation of the metallic oxide fumes; the dusts of the various zinc compounds may also be inhaled. Zinc chloride acts locally on the skin.

Physiological Action and Toxicity: The inhalation of freshly sublimed zinc oxide fumes may cause an acute febrile reaction which has been known amongst workmen as "smelter shakes", "brass chills", "brass founder's ague" and other similar names. Though zinc fume is the commonest offender, other metallic oxides may cause the condition, which is described under the title "Metal Fume Fever".

There are a few reports in the literature describing a chronic form of zinc poisoning, with production of gastro-intestinal disturbances and even gastric and duodenal ulcer. Exposure for more than 6 months to dust or fumes of the oxide, chloride, chromate or sulphate has been said to cause dermatitis, conjunctivitis, gastro-intestinal disturbances and anaemia. However, the existence of chronic zinc poisoning is by no means certain, since commercial zinc contains arsenic, and many zinc compounds are used in mixture with other materials such as lead carbonate. Chronic illness described as being due to zinc may have been due to other toxic substances present. Zinc chloride is strongly corrosive to the skin and mucous membranes. Inhalation of the powder may cause ulceration of the nasal septum which

may progress to actual perforation. When used as a flux in soldering, in the form of a "grease", spattering of the material on the skin may cause deep, penetrating burns or ulcers, which exhibit little inflammatory reaction about their edges. The ulcers are very slow-healing.

Recommended M.A.C.: The recommended maximum allowable concentration for fumes of zinc oxide is 150 mgm per 10 cubic metres of air.

Fire Hazard: Hydrogen is liberated by the action of acids and caustic soda on zinc. The metallic dust may form explosive mixtures with air. Zinc dust, in bulk, in a damp state may ignite spontaneously on exposure to air.

Chapter IV

THE OCCUPATIONAL DERMATOSES

From an industrial viewpoint, occupational skin diseases are very important. They constitute at least sixty to seventy per cent of all cases of occupational disease and are responsible for much suffering and loss of working time.

Protective Quality of the Skin

The cells and secretions of the skin perform an important function—that of protection against external irritants. Thus, the cells of the horny layer will withstand the action of moderately strong acids, though they are attacked by alkalis and fat solvents. Perspiration affords protection by diluting those irritants which are soluble in water while the sebaceous secretions, which consist of cholesterol and liquid waxes, form a protective coating. In addition, the pigment of the skin acts as a protective agent against light.

Under certain conditions perspiration and sebaceous matter may promote the action of irritants on the skin. Excessive sweating, for example, may render the skin more liable to attack by making it sodden. Sweat may also act as a solvent for an irritant which can then permeate the skin. Sebaceous secretions may admit irritating fat solvents to their ducts and to the hair follicles.

A thick oily skin is more resistant than a dry skin to the action of fat solvents such as soaps, turpentine, benzol, trichloroethylene and carbon tetrachloride. Individuals with hairy arms and legs and seborrhoeic skin are more likely to develop acne-like lesions and folliculitis upon contact with clothing soiled with oils, greases or waxes.

Causative Agents

Chief among these are (1) *mechanical* action such as friction, pressure and trauma, which cause callosities, bursitis, tenosynovitis, cuts and abrasions; (2) *physical* agents such as heat, cold, electricity, sunlight and radiations from radioactive materials or x-rays; (3) *biological* agents such as bacteria, fungi and parasites; (4) *chemicals*. Of these four groups, the last is the greatest offender since more workers are exposed to chemicals than to the other causative agents.

Mechanical and Physical Agents

The lesions produced in workers by the action of mechanical and physical agents do not differ, as a rule, from those produced outside industry. The diagnosis is usually made without difficulty, and the association, where it exists, between the lesions and the workman's occupation is easily determined in most cases.

Biological Agents

Certain occupations involve exposure to dermatitis-producing bacteria, fungi and parasites. Secondary infection with pyogenic organisms is common in occupations where the skin is subject to cuts, scratches, abrasions or punctures.

Since a large percentage of the general population is subject to mycotic infections of the skin, the role of occupational exposure in causing these infections is often difficult to assess. Differential diagnosis is further complicated by the fact that fungus infections may occur in persons suffering from occupational dermatitis; or occupational dermatitis may be superimposed on a superficial fungus infection.

Chemical Agents

The chemical agents causing occupational dermatoses may be divided into primary irritants and sensitizers. A primary cutaneous irritant is a material which will produce damage when in contact with any person's skin in sufficient strength for an adequate length of time. A cutaneous sensitizer is an agent which does not necessarily cause demonstrable cutaneous changes on first contact, but may induce specific changes in the skin so that further contact on the same or other parts of the body will cause dermatitis.

A primary irritant may also act as a sensitizer. In this case, initial exposure may condition the skin so that further contact, even with dilute solutions, may produce a dermatitis.

Individuals vary greatly in their natural resistance to skin irritants. Thus, blonde persons are usually more susceptible than brunettes. As distinct from such inborn susceptibility, a worker may develop a sensitivity to a chemical as a result of previous exposure. This acquired sensitivity may be quite specific for the chemical in question, or an acquired sensitivity may result in the worker becoming sensitive to other substances which are related chemically to the original

sensitizer. Thus, a worker sensitized by exposure to phenol, may at the same time become sensitive to cresol, and possibly other caustics.

In many instances, when a worker first comes in contact with a sensitizing agent, he develops a dermatitis. However, if the worker is able to continue on the job, his dermatitis may clear up after a period of time; he has become "hardened". Hardening may be permanent, or it may disappear after exposure is discontinued for some time.

In general, chemicals exert a harmful action on the skin. They act in many ways. The keratin may be softened by the solvent action of alkalis and soaps. Concentrated acids combine with the water in the skin and cause chemical burns. The fat may be removed from the skin by fat solvents. Heavy metal salts may cause precipitation of proteins in the skin. Some chemicals such as hexamethylene tetramine (urotropin) when in contact with the moisture of the skin, hydrolyse and form irritating compounds. Chemicals, such as arsenic, petroleum, coal tar products, and aniline compounds stimulate the horny layer of the skin and may lead to new growths. Another group of chemicals, *e.g.*, the nitro and nitroso compounds, cause hypersensitivity of the skin after varying periods of contact. Photosensitization can be induced by coal tar and petroleum products, and by some dyes and sulphonamide drugs.

Some of the more common materials causing occupational dermatitis are oils and greases, solvents, acids and alkalis, chromic acid and its salts, nickel, synthetic resins (plastics) and waxes, cyanides, rubber ingredients, drugs used for treating injuries, and explosives.

Oils and Greases are used in a great many industries and account for a relatively large number of cases. Most cases occur amongst machine workers using cutting oils. Cutting oils are usually of two types, soluble and insoluble. The soluble oil is a water emulsion containing some mineral oil, soap and other minor materials. The insoluble oil is usually a mineral oil containing sulphur or chlorine or both. Machinists in contact with such oils, particularly with the insoluble variety, develop oil acne which often becomes disabling. The acne is usually localized on the arms, shoulders, body and legs but not, as a rule, on the face. Most of the lesions occur around the hair follicles which have been blocked by oil and dirt. If scratched or abraded, these areas may become infected, with the formation of small boils or pimples. Other irritating materials may also be present in the oil.

The occurrence of numerous cases of infected dermatitis in a shop may suggest exposure to a common bacterial agent. A culture will often show the oil to be contaminated with such organisms as Staph. aureus and B. pyocyaneus but this bacterial contamination is generally considered to be of little importance. It is more likely that the infection is due to organisms present on the worker's skin, which are introduced through scratches and abrasions. Cutting oils seldom cause dermatitis if special attention is paid to personal and shop cleanliness.

Industrial Solvents such as petroleum distillates, trichloroethylene, alcohol, toluol, and lacquer thinners remove the fat from the skin, causing it to crack and fissure. Many cases of occupational skin diseases are due to the harmful practice of using solvents as hand cleansers. When solvents are spilt on clothing, the clothing should be removed immediately to avoid a burn.

Acids and Alkalis are the cause of much trouble. In strong concentrations, they produce an effect similar to a burn. In more dilute application, damage to the skin may follow prolonged contact. Weak alkaline solutions act by dissolving the natural fats out of the skin, which then becomes soft and sodden.

Chromic Acid and its derivatives are a common cause of dermatitis. Sensitivity to bichromates is not uncommon. Cuts or abrasions contaminated with bichromates tend to ulcerate.

Nickel and more particularly, nickel salts are often a cause of dermatitis. This type of dermatitis appears most frequently when atmospheric conditions are hot and moist, the incidence being highest in the summer months.

Plastics and Waxes play an important role as causative agents of occupational skin diseases. The two most important resins in this group are the phenol-formaldehyde and urea-formaldehyde types which are used in moulded goods, in wood plastics and in cloth as a filler or size. Dermatitis from these sources is limited in the main to those persons handling and working the material before it is set or moulded. A worker rarely becomes affected from the finished resin. When this happens, it is thought that other ingredients present, such as hexamethylene tetramine, may have been responsible.

Chloronaphthalene waxes are used for electric insulation on wires and condensers. Such waxes cause an acne-like condition. The skin

on the face and on the abdomen is most commonly involved. Practically all exposed workers will develop the condition if proper control measures are not introduced.

Cyanide solutions are the cause of dermatitis among electroplaters and among workers employed in the cyanide extraction of gold. Hot cyanide solutions used in the hardening of tools are hazardous, causing a burn on contact with the skin. When this occurs, the cyanide must be removed from the burned area as quickly as possible to prevent absorption and subsequent systemic poisoning.

Rubber Ingredients. Chemicals used in the rubber trade have been responsible for much dermatitis. Compounding room and mill workers are those usually affected although skin conditions may occur in other jobs.

The main causative agents are the accelerators and antioxidants, not the crude rubber. The most irritating accelerator is probably hexamethylene tretramine. Wherever this chemical is used, dermatitis is prevalent. Fortunately, hexamethylene tetramine has been largely replaced in the rubber trade by other accelerators which are less irritating to the skin. Rubber that blooms after vulcanization occasionally causes dermatitis. It is thought that this bloom contains accelerators and antioxidants or their break-down products. Persons coming in contact with a product containing such rubber may be affected. Sulphur monochloride is another irritating chemical sometimes found in rubber products that have been cured by being exposed to the vapour. If the sulphur monochloride is not removed or neutralized, skin eruptions may develop upon contact with the rubber.

Dermatitis is not prevalent among workers in synthetic rubber plants. Such plants are new, and use modern methods of handling which incorporate the experience gained in the natural rubber industry. Many of the chemicals used are similar to those employed in the treatment of natural rubber.

Explosives. Military and peace-time explosive chemicals are one of the greatest potential skin hazards in industry. Over fifty per cent of workers who come in contact with mercury fulminate develop skin eruptions, the face and eyelids being the usual sites involved. The condition clears up quickly on removal of the person from exposure. Since "hardening" of the skin to mercury fulminate does not take place, such removal must be permanent.

Tetryl, used extensively for filling fuses, is a primary irritant but may also act as a sensitizer. T.N.T. sometimes causes skin lesions, the incidence being lower than with mercury fulminate and tetryl exposures. Extreme care is necessary when handling T.N.T. as it is poisonous and can be absorbed through the skin.

Dermatitis-Producing Drugs. Certain skin diseases are caused by materials used in the first aid treatment of cuts and abrasions. Sulphonamide preparations which have a sensitizing action are among the chief offenders in this respect. For this reason, such products should be applied to the skin only when their use is definitely indicated.

Diagnosis of Industrial Dermatitis

It is beyond the scope of this book to attempt to give any detailed description of the lesions produced by the various irritants and sensitizers encountered in industry. In certain instances, *e.g.*, chrome, characteristic lesions are produced. In other cases, especially where the causative agent is a primary irritant, such as caustic soda, acids, etc., the causal relationship is obvious. In the majority of cases, however, there are no specific lesions, and the relationship between the dermatitis and the exposure is obscure. The excellent textbook "Occupational Diseases of the Skin", by Schwartz, Tulipan and Peck, is recommended for detailed information on the diagnosis and treatment of occupational dermatoses.

In general, a diagnosis of industrial dermatitis must be based on a careful history of the development of the skin condition, including detailed inquiry into the workman's exposure. In examining the patient, complete removal of all clothing is essential. The following criteria will assist in establishing or eliminating the occupational factor as a possible cause of the dermatitis. Where a diagnosis of occupational dermatitis is made, most of these criteria must be fulfilled.

1. The dermatitis appears during a period of industrial exposure or within a reasonable period after cessation of exposure (usually not longer than 2 or 3 weeks.)
2. The dermatitis was not previously present, other than as a similar dermatitis in a similar exposure.
3. The dermatitis disappears or is improved by termination of the exposure.

4. The dermatitis tends to recur or to exacerbate upon the worker's return to exposure.
5. The dermatitis usually occurs first in the exposed areas; if a solid or liquid, the forearms may be effected; if a vapour or dust, the face, neck, and other parts of the body may be involved.
6. The appearance of the dermatitis should be consistent with known cases resulting from a similar exposure. In general the acute type will appear as erythema, oedema, vesicles, oozing and crusting.
7. Fellow workers in a similar exposure are, or have been, similarly affected.

Knowledge of the working processes and substances used in the patient's occupation is essential to diagnosis. The plant chemist can often assist in determining the composition of materials to which the worker is exposed.

Patch tests, properly performed and interpreted by those experienced in the technic of this test, are often of value in making a diagnosis.

Difficult cases should be referred to a dermatologist in order that valuable time may not be lost before commencement of proper treatment. Many conditions clear up readily under suitable treatment but become intractable if treatment is delayed.

Provincial Divisions of Industrial Hygiene are prepared to investigate exposures and render assistance in the diagnosis of difficult cases.

Prevention or Control

Occupational dermatitis develops because of:

1. Lack of knowledge regarding the harmful nature of the materials used.
2. Improper selection of workers.
3. Lack of periodic inspection of workers.
4. Poor personal hygiene.
5. Failure to provide or maintain protective measures.

1. *Lack of Knowledge*

Proper precautions can only be taken when industrial materials are known to present a skin hazard. The employer should be familiar

with those substances used in his plant that are likely to cause skin trouble, and with the measures he should adopt to protect his employees' health. The employee should know the dangers associated with improper handling of these harmful substances, and be instructed in the steps that he should take to protect his own health.

2. *Selection of Workers*

In plants where materials with known irritating or sensitizing properties are used, selection of workers is of special importance. Workers who are tidy and clean are less liable to suffer from skin disease than those who are careless in matters of personal cleanliness. Fair skinned persons are generally more sensitive to skin irritants. Workers with oily skin resist the effects of solvents better than those with dry skin. Probably the most important types to exclude are individuals who have a history of skin allergy or of repeated or chronic skin eruption.

3. *Periodic Inspection of Workers*

It is important that workers who are handling skin irritants and sensitizers be examined periodically for evidence of irritation. Frequent inspection may reveal cases in the early stages when they are more amenable to treatment. Furthermore, the early discovery of cases enables measures to be taken to eliminate or reduce the exposure, thereby preventing a widespread outbreak.

4. *Personal Hygiene*

It is important that workers wash carefully at the end of the work period, ensuring that all offending material is removed from the skin. If the skin is not properly cleaned, some of the irritant will remain, maintaining continuous exposure and allowing the skin no chance to recuperate. Eight hours contact, followed by 16 hours free from irritants, materially changes the picture in many instances. Length of exposure is just as important as the exposure itself. This applies in particular to workers exposed to cutting oils and to chemicals used in the explosives and rubber industries.

If a major skin hazard is present in a plant, extra washing facilities will be required. In some instances it may be necessary for workers to have a shower at the end of each shift. If this is to be done, one shower is necessary for every three or four workers. Similarly, the number of wash basins may have to be increased to one for every three or four workers. Hot water, soap and towels should be supplied,

as these are necessary prerequisites to proper washing. Harsh soap, abrasives, and other drastic methods of skin cleaning should not be used. Management should not supply or permit workers to use solvents on their skin for cleaning purposes. In many cases, such practice has produced dermatitis. If the worker is careful and is properly protected, washing at the end of the work period with hot water and mild soap should be adequate.

5. *Protective Measures*

In addition to careful selection and periodic inspection of workers, and the provision of facilities for maintaining personal cleanliness, there are other protective measures which assist greatly in preventing the occurrence of occupational dermatitis.

(a) *Substitution of Materials*

In many instances, non-irritating or less irritating materials can be used in the factory without interfering with operations. This method of substitution is widely practised in industry. A good example is to be found in the rubber industry where the chief chemical irritant, hexamethylene tetramine, has been largely replaced by safer materials. It is true, however, that in some industries there are no safe substitutes and the workers are required to handle materials likely to cause skin trouble.

(b) *Mechanical Methods*

Contact with dermatitis-producing chemicals can often be eliminated or greatly reduced by the provision of mechanical methods for handling and processing materials. It is often possible to completely enclose certain processes, thereby eliminating the hazards they present. Where harmful dusts or vapours are produced, suitable ventilation may be necessary to solve the problem. Even when not particularly harmful, their removal helps to keep the workshop clean, and thus encourages the workmen in maintaining habits of personal cleanliness.

(c) *Protective Clothing*

Protective clothing for the workers is important. In some instances a complete change of clothing is necessary each day. In most cases, however, protection can be afforded by supplying aprons, gloves, and rubber boots. The introduction of artificial rubber has made available aprons, gloves, etc. that will

withstand the effects of oil. Such equipment has been of great assistance to large groups of workers.

It is important to ensure that gloves are clean inside. Gloves whose inner surfaces are contaminated with irritating materials are worse than none. In certain instances rubber gloves are not satisfactory, *e.g.*, in handling explosive chemicals. For this work, ordinary cotton gloves should be supplied daily or at shorter intervals depending on the exposure.

(d) *Protective Creams*

Protective creams are of value in many cases. They should be selected on the basis of their suitability to the chemicals being handled, as no one cream will serve all purposes. One of the advantages of a protective cream is that the worker can wash his hands and exposed parts without recourse to harsh washing methods. While protective creams are of definite value in certain exposures, they should not be relied upon as the sole method of preventing skin contact with irritating chemicals. The application of such creams does not eliminate the need for such preventive measures as maintaining personal cleanliness, substitution of mechanical methods of handling, provision of protective clothing and substitution of less harmful materials.

(e) *First Aid Treatment of Accidental Injuries*

Small scratches, cuts and abrasions should always be treated promptly, especially where a worker is exposed to strong irritants. Protective dressings should be applied and if necessary, the workman should be removed from exposure. Lack of protective dressings, or failure to remove the workman from exposure, results in aggravation of the original injury, often with serious consequences.

(f) *Removal of Worker from Exposure*

In some cases, especially when the worker is sensitive to some material used in his work, he must be transferred to another job. When this is done, it is important that the new job does not involve an exposure which may aggravate the condition. The generally-held impression that "outdoor" work is free from skin hazards may be misleading. A worker with dermatitis is not likely to benefit if he is sent outside to unload a car of soda ash or to paint a building.

LIST OF OCCUPATIONS WITH THEIR SKIN IRRITANTS

Acetylene Makers
- Calcium carbide

Acid Workers
- Cyanides
- Hydrochloric acid
- Nitric acid
- Sulphuric acid

Aeroplane Dope Makers
- Acetone
- Amyl acetate
- Benzol
- Carbon tetrachloride
- Tetrachloroethane

Alkali Salt Makers
- Chlorine
- Hydrochloric acid

Bakers
- Dough
- Dust
- Heat
- Potassium persulphate

Barbers and Hairdressers
- Arsenic
- Bromethane
- Capsicum
- Carbon tetrachloride
- Colocynth
- Mercury
- Paraphenylene diamine
- Quinine
- Resorcin
- Soap
- Sulphur
- Tar

Battery (dry) **Makers**
- Amyl acetate
- Benzol
- Chromium compounds
- Hydrochloric acid
- Mercury
- Tar

Beatermen (paper and pulp)
- Alkalis
- Chlorine
- Dyes

Bleachers
- Chloride of lime
- Chlorine
- Chromium compounds
- Hydrochloric acid
- Hydrofluoric acid
- Nitric acid
- Potassium hydroxide
- Sodium hydroxide

Bricklayers
- Lime

Bronzers
- Ammonia
- Amyl acetate
- Arsenic
- Benzol
- Cyanides
- Hydrochloric acid
- Mercury
- Methanol (methyl alcohol)
- Petroleum hydrocarbons

Cap Loaders
- Mercury

Carbolic Acid (phenol) **Makers**
- Benzol
- Phenol
- Sulphuric acid

Cartridge Dippers
- Hydrochloric acid
- Nitric acid
- Sulphuric acid

Cellulose Workers
- Carbon disulphide

Cementers (rubber shoes)
- Benzol
- Carbon disulphide
- Carbon tetrachloride
- Methanol (methyl alcohol)
- Petroleum hydrocarbons

Cement Mixers (rubber)
- Benzol
- Carbon disulphide
- Carbon tetrachloride
- Petroleum hydrocarbons

Cement Workers
- Lime
- Pitch
- Resin

Chlorine Makers
- Hydrochloric acid

Chromium Platers
- Chromium compounds

Cloth Preparers
- Acids
- Alkalis
- Lime
- Potassium salts
- Soap
- Sodium salts
- Sodium silicate

Coal Tar Workers
- Aniline
- Benzol
- Cresol
- Phenol
- Tar

Colour Makers
- Ammonia
- Antimony
- Arsenic
- Benzol
- Bromine
- Cadmium
- Chlorine
- Chromium
- Lead
- Sulphuric acid
- Tetrachloroethane
- Thallium

Compositors
- Aniline
- Antimony
- Petroleum hydrocarbons

Compounders (rubber)
- Aniline
- Antimony
- Arsenic
- Chromium compounds
- Petroleum hydrocarbons

Cotton Sizers
- Acids
- Aluminium salts
- Arsenic salts
- Calcium salts
- Magnesium salts
- Phenol
- Zinc chloride

Degreasers
- Benzol
- Carbon disulphide
- Carbon tetrachloride
- Petroleum hydrocarbons
- Tetrachloroethane
- Trichloroethylene

Dentists
- Mercury

Detonator Cleaners
- Mercury

Detonator Packers
- Mercury

Dishwashers
- Bacteria
- Caustic soaps
- Fungi
- Grease

Disinfectant Makers
- Chloride of lime
- Chlorine
- Cresol
- Formaldehyde
- Mercury
- Thallium

Druggists
- Chloride of lime
- Iodoform
- Soap
- Sodium salts
- Sugar

Dry Cleaners
- Benzol
- Carbon disulphide
- Carbon tetrachloride
- Methanol (methyl alcohol)
- Oxalic acid
- Petroleum hydrocarbons
- Turpentine

Dye Makers
- Acetone
- Acridine
- Ammonia
- Aniline
- Antimony
- Arsenic
- Benzol
- Bromine
- Chloride of lime
- Chlorine
- Chromium compounds
- Cresol
- Cyanides
- Dimethyl sulphate
- Formaldehyde
- Formic acid
- Hydrochloric acid
- Mercury
- Methanol (methyl alcohol)
- Methyl chloride
- Nitric acid
- Nitrobenzols
- Oxalic acid
- Phenol
- Phenylhydrazine
- Phosgene
- Picric acid (trinitrophenol)
- Sulphuric acid
- Turpentine

Dyers
- Acetone
- Amyl acetate
- Aniline
- Chromium compounds
- Hydrochloric acid
- Hydrofluoric acid
- Petroleum hydrocarbons
- Picric acid (trinitrophenol)

Electroplaters
- Antimony
- Arsenic
- Benzol
- Carbon disulphide
- Carbon tetrachloride
- Chromium compounds
- Cyanides
- Formic acid
- Hydrochloric acid
- Mercury
- Nitric acid
- Petroleum hydrocarbons
- Sulphuric acid

Embalmers
- Formaldehyde
- Mercury

Enamelers
- Amyl acetate
- Arsenic
- Benzol
- Carbon disulphide
- Chromium compounds
- Petroleum hydrocarbons
- Tetrachloroethane
- Turpentine

Engravers
- Benzol
- Hydrochloric acid
- Oxalic acid
- Sulphuric acid
- South African boxwood
- Turkish boxwood

Etchers
- Hydrochloric acid
- Hydrofluoric acid
- Nitric acid
- Phenol
- Sulphuric acid

Explosive Workers
- Acetone
- Ammonia
- Amyl acetate
- Amyl alcohol
- Aniline
- Benzol
- Carbon disulphide
- Cresol
- Formaldehyde
- Mercury
- Methanol (methyl alcohol)
- Nitric acid
- Nitroaniline
- Nitrobenzols
- Nitroglycerine
- Phenol
- Picric acid (trinitrophenol)
- Sulphuric acid

Farmers
- Arsenic

Felt Hat Makers
- Arsenic
- Mercury
- Methanol (methyl alcohol)
- Sulphuric acid

Fertilizer Makers
- Ammonia
- Cyanides
- Hydrochloric acid
- Hydrofluoric acid
- Manganese
- Nitric acid
- Phosphorus
- Sulphuric acid

Fish Dressers
- Brine

Flax Spinners
- Lime
- Brine

Flour Mill Workers
- Dust
- Fungi
- Parasites

Fur Handlers
- Arsenic
- Dyes
- Mercury

Fur Preparers
- Arsenic
- Dyes
- Mercury
- Nitric acid

Furniture Polishers
- Amyl acetate
- Chromium compounds
- Methanol (methyl alcohol)
- Petroleum hydrocarbons
- Petroleum oils
- Turpentine

Galvanizers
- Ammonia
- Arsenic
- Hydrochloric acid
- Nitric acid
- Sulphuric acid

Garage Workers
- Petroleum hydrocarbons

Gardeners
- Arsenic

Gas (illuminating) Workers
- Ammonia
- Benzol
- Cyanides
- Phenol
- Tar

Gas-Mantle Impregnators
- Thorium

Glass Mixers
- Antimony
- Arsenic
- Hydrochloric acid

Gold Refiners
- Arsenic
- Cyanides
- Hydrofluoric acid
- Mercury

Ink Makers
- Bromine
- Chlorine
- Chromium compounds
- Formaldehyde
- Hydrochloric acid
- Methanol (methyl alcohol)
- Nitroaniline
- Oxalic acid

Insecticide Makers
- Arsenic
- Carbon disulphide
- Formaldehyde
- Phosphorus

Jewelers
- Amyl acetate
- Cyanides
- Hydrochloric acid
- Mercury
- Nitric acid
- Sulphuric acid

Lampblack Makers
- Petroleum
- Phenol

Laundry Workers
- Chloride of lime
- Chlorine

Lime Burners
- Lime

Lime Pullers (tannery)
- Lime

Linoleum Makers
- Acrolein
- Amyl acetate
- Methanol (methyl alcohol)
- Petroleum hydrocarbons
- Sulphuric acid
- Turpentine

Linotypers
- Antimony

Machinists
- Cutting compounds
- Lubricants
- Oils

Masons
- Lime

Match-Factory Workers
- Carbon disulphide
- Chromium compounds
- Phosphorus
- Potassium hydroxide
- White pine
- Sodium hydroxide
- Sulphuric acid

Mercerizers
- Acids
- Alkalis

Mercury Workers
- Mercury

Mixers (rubber)
- Aniline
- Arsenic
- Benzol
- Chromium compounds
- Petroleum hydrocarbons

Mordanters
- Acids
- Alkalis
- Aluminium salts
- Antimony compounds
- Arsenates

Chromates
Copper salts
Iron salts
Lead salts
Phosphates
Silicates
Tin salts
Zinc chloride

Mottlers (leather)
Dyes

Nickelplaters
Zinc chloride
Nickel sulphate

Nitroglycerine Makers
Nitric acid
Sulphuric acid

Oil Extractors
Acetone
Carbon disulphide
Tetrachloroethane

Oil Flotation Plant Workers
Petroleum

Oil Purifiers
Sulphuric acid

Oil Refiners
Sodium hydroxide

Paint Makers
Acetone
Amyl acetate
Aniline
Arsenic
Benzol
Carbon disulphide
Chromium compounds
Hydrochloric acid
Mercury
Methanol (methyl alcohol)
Petroleum hydrocarbons
Phenol
Tar
Turpentine

Painters
Acetone
Amyl acetate
Aniline
Arsenic
Benzol
Carbon disulphide
Chromium compounds
Mercury
Methanol (methyl alcohol)
Petroleum hydrocarbons
Tar
Turpentine

Paper-Box Makers
Dyes
Glues

Paper Makers
Canadian spruce
Chlorine
Formaldehyde
Sulphuric acid
Sodium hydroxide

Paraffin Workers
Acetone
Carbon disulphide
Carbon tetrachloride
Petroleum
Tar

Parchment Makers
Zinc chloride

Pencil Makers
Pyridine
Mountain cedarwood

Petroleum Refiners
Ammonia
Hydrochloric acid
Petroleum
Petroleum hydrocarbons
Sulphuric acid
Tar

Photograph Engravers
Benzol
Chromium compounds
Methanol (methyl alcohol)
Nitric acid

Photographic-Plate Cleaners
Alkalis

Picklers (metal)
Hydrochloric acid
Nitric acid
Sulphuric acid

Pitch Workers
Arsenic
Tar

Plasterers
Lime

Plumbers
Caustic soaps
Hydrochloric acid
Zinc chloride

Polishers
Petroleum hydrocarbons
Turpentine

Printers
Aniline
Arsenic
Petroleum hydrocarbons
Turpentine

Pyroxylin-Plastics Makers
Acetone
Amyl acetate
Benzol
Butyl alcohol
Cyanides
Methanol (methyl alcohol)
Petroleum hydrocarbons
Nitric acid
Sulphuric acid

Refiners (metals)
Arsenic
Mercury
Nitric acid
Sulphuric acid

Refrigerating Plant Workers
Ammonia

Refrigerator Makers and Repair Men
Acrolein
Ethyl bromide
Ethyl chloride

Ropemakers
Alkalis
Dyes
Tar

Rubber Workers
Acetone
Aniline
Arsenic
Benzol
Carbon tetrachloride
Chromium compounds
Formaldehyde
Formic acid
Methanol (methyl alcohol)
Petroleum hydrocarbons
Tetrachloroethane
Turpentine

Salt Preparers
Brine

Sewer Workers
Ammonia
Carbon dioxide

Shell Fillers
Nitroglycerine
Picric acid (trinitrophenol)

Shoe Finishers
Ammonia
Amyl acetate
Amyl alcohol
Benzol
Methanol (methyl alcohol)
Petroleum hydrocarbons

Silk Workers
Acids
Alkalis
Dyes

Slaughter and Packing-House Workers
- Bacteria
- Brine
- Fungi
- Parasites

Smelters
- Arsenic

Soap Makers
- Acrolein
- Benzol
- Formaldehyde
- Formic acid
- Hydrochloric acid
- Methanol (methyl alcohol)
- Nitroaniline
- Potassium hydroxide
- Sodium hydroxide
- Sulphuric acid

Sodium Hydroxide Makers
- Sodium hydroxide

Solderers
- Cyanides
- Hydrochloric acid
- Zinc chloride

Stockyard Workers
- Bacteria
- Fungi
- Parasites

Sugar Refiners
- Ammonia
- Carbon dioxide
- Hydrochloric acid
- Sulphuric acid

Tannery Workers
- Ammonia
- Amyl acetate
- Aniline
- Arsenic
- Chloride of lime
- Cyanides
- Formaldehyde
- Formic acid
- Hydrochloric acid
- Mercury
- Oxalic acid
- Petroleum hydrocarbons
- Sodium hydroxide
- Sulphuric acid

Tar Workers
- Tar

Taxidermists
- Arsenic
- Mercury

Temperers
- Cyanides
- Petroleum
- Sulphuric acid

Tinners
- Zinc chloride

Typists
- Dyes
- Chlorinated hydrocarbons
- Petroleum hydrocarbons

Type Cleaners
- Benzol
- Methanol (methyl alcohol)

Upholsterers
- Methanol (methyl alcohol)

Veterinarians
- Bacteria
- Fungi
- Parasites

Vulcanizers
- Aniline
- Antimony
- Benzol
- Carbon dioxide
- Carbon disulphide
- Carbon tetrachloride
- Chromium compounds
- Methanol (methyl alcohol)
- Petroleum hydrocarbons

Washers
- Alkalis
- Chloride of lime
- Soap

Watchmakers
- Potassium cyanide
- Trichloroethylene

Waterproofers (paper)
- Formaldehyde
- Paraffin

Wax-Ornament Makers
- Arsenic

Welders
- Benzol
- Mercury

Wood Preservers
- Arsenic
- Mercury
- Phenol
- Tar

X-Ray Workers
- Radiant energy

Zinc Chloride Makers
- Chlorine
- Hydrochloric acid

Zinc Electrode Makers
- Mercury

Zinc Miners
- Arsenic

Zinc Refiners
- Arsenic
- Antimony

CHAPTER V

PROVINCIAL WORKMEN'S COMPENSATION ACTS

This chapter provides a general description of Workmen's Compensation Acts, with particular emphasis on those aspects affecting the practising physician. A copy of the appropriate provincial act should be in the possession of persons who deal with compensation cases.

Purpose of the Acts

Workmen's Compensation Acts exist in every province except Prince Edward Island. They provide compensation for disability and medical treatment in case of injury due to an accident arising out of employment or to an occupational disease covered by the Acts.

An industry coming under a Compensation Act is made responsible for medical expenses and part of the loss of earnings of workmen incurred as a result of injury suffered in employment. An injured workman who comes under the Act has no right of action at law against his employer. In all provinces compensation cannot be assigned, charged or attached without approval of the Board. A workman cannot waive the benefits that he is entitled to under the Act.

Administration

Each provincial Act provides for the appointment by the Lieutenant-Governor in Council of a Workmen's Compensation Board to administer the Act. The Board has exclusive jurisdiction in all matters to which the Act applies. These include:

Determination and payment of compensation to workmen or dependents.
Payment of medical expenses incurred by injured workmen.
Classification of industries according to accident hazard.
Fixing of assessment rates appropriate to each class with preferential or merit rating in favour of industries with good accident records.
Collection of assessments.

Accident Fund

Each Act provides for the creation and maintenance of an accident fund from assessments levied upon employers in industries covered by the Act. These industries are divided into classes according to

their accident records and have corresponding assessment rates. All compensation, medical and administrative costs are paid from the accident fund. Payment of compensation is not affected by default of an employer in paying his assessment or by his insolvency.

Scope of Acts

The provincial Workmen's Compensation Acts vary in scope, but in general, cover most of the industries in each province. Farming and domestic services are the only large occupational groups not covered in any provincial Act. The following table which varies from province to province, lists the general range of industries and occupations covered by the Acts.

Automobile repair shops
Bakeries
Building and allied trades
Cleaning and dyeing
Dairies
Electric power systems
Engineering and construction
Fishing
Freight and passenger elevators
Grain elevators
(A) Hospitals
(B) Hotels
Lumbering
Lumber, coal and wood yards
Manufacturing of:
  Animal products
  Chemical and allied products
  Iron and steel products
  Non-ferrous metal products
  Non-metallic mineral products
  Textiles and allied products
  Vegetable products
  Wood and paper products
Mining
Municipal corporations
Navigation
Painting and decorating
Plumbing
Power laundries
Printing and publishing
Public utilities
Quarrying
Railways
Refrigeration plants
(C) Restaurants
Road, air and water transport
Service stations
(D) Shops
Telephone and telegraph systems
Theatres
Window cleaning

(A) Listed only in British Columbia, Saskatchewan, Ontario and New Brunswick.

(B) Listed only in British Columbia, Alberta, Saskatchewan, Ontario and New Brunswick.

(C) Listed only in British Columbia, Alberta, Saskatchewan, Ontario.

(D) Listed only in British Columbia, Alberta, Saskatchewan, New Brunswick.

In all provinces except Alberta, establishments in certain industries employing below a specified number of employees are excluded from the Acts.

In Quebec, municipal corporations and certain companies, such as telephone, telegraph, railway and navigation systems, arrange and pay for medical aid according to the Board's tariff. This arrangement formerly obtained in Ontario, but following a recent amendment to the Ontario Act, arrangements for medical aid are made through the Board.

Coverage

Accidents

In any employment covered by a compensation Act, compensation is paid for disability when "personal injury by accident arising out of and in the course of the employment is caused to a workman." Whereas negligence on the part of the employer or the workman does not affect payment of a claim for compensation, no payment is paid when the injury "is attributable solely to the serious and wilful misconduct of the workman unless the injury results in death or serious disablement." This wording of the Ontario Act is reproduced in the statutes of Quebec and Alberta. In British Columbia, Manitoba, Saskatchewan and Nova Scotia, the law is similar except that compensation for injury caused through wilful misconduct of the workman is payable only when disability is permanent.

The New Brunswick Act rules that no compensation shall be paid if the injury is intentionally caused by the workman or is principally due to his intoxication or serious and wilful misconduct or to a fortuitous event unconnected with the industry in which the workman was employed.

Occupational Diseases

In all provinces, when a workman is disabled or dies as a result of an occupational disease covered in the Act, he or his dependents are entitled to pension if the disease is due to the nature of any employment in which he was engaged at any time within twelve months previous to the date of his disablement whether under one or more employments. In Ontario, the twelve months qualification does not apply.

The diseases for which compensation is payable, are listed in a schedule to each Act. In Ontario, since 1947, any disease peculiar

to an industrial process and resulting in disability is to be compensated. While the Ontario Act provides for a wide coverage, only conditions related to an industrial process are compensable.

In all provinces except Alberta, compensation will not be paid, if at the time of entering into employment, the workman has wilfully and falsely represented himself as not having previously suffered from the disease for which compensation is being claimed.

Waiting Period

Under each Act, a fixed period must elapse following the occurrence of the disability before compensation becomes payable. This "waiting period" varies from three to seven days, and in all provinces having an Act, compensation is paid for the waiting period if disability continues beyond it. Under all the Acts, medical aid is given from the date of the accident.

In Nova Scotia, Ontario and Quebec, compensation is payable only if the disability continues for seven days or more, in which case compensation is payable from the date of the disability.

In Alberta, British Columbia, Manitoba and Saskatchewan, no compensation is payable if the injury disables the workman for three days or less, but if the disability lasts for more than three days in Saskatchewan, four days in New Brunswick, six days in Alberta and British Columbia or fourteen days in Manitoba, compensation is payable from the date of the disability.

Medical Aid

Under each Act, the cost of medical aid for injured workmen is borne by the Accident Fund and is extended as long as treatment can be of benefit or until the physical condition stabilizes. Pensions are then paid to cases with residual permanent disability. In all provinces, medical aid includes surgical, nursing and hospital services and the provision and repair of crutches, artificial limbs and other prosthetic apparatus. In Ontario, treatment may be given by persons registered under the Drugless Practictioners Act and in Alberta by persons "licensed to practise the healing art."

Employers' Medical Aid Schemes

Under all the Acts, employers' schemes for medical aid to employees may be approved by the Board if it considers them to be as favourable

to the workmen as the provisions of the Act. Such an approved scheme may replace the arrangement for medical aid in the Act, insofar as the treatment of industrial diseases and accidents is concerned. Except for British Columbia and Manitoba, an approved scheme entitles the employer to reimbursement out of the Accident Fund or to a reduction in his assessment rate.

In all provinces except Alberta, British Columbia and Nova Scotia (coal mining), contributions by workmen to a medical aid scheme approved by the Board are forbidden by the Act.

First Aid

In all provinces, employers may be required by the Board to maintain such first aid equipment and service as the Board may direct. In British Columbia the Board may install first aid equipment and charge the cost to the employer who fails to comply with this provision. Regulations under the Compensation Acts have been issued in all provinces setting out the minimum first aid equipment and staff required according to the number of employees.

Rehabilitation

In every province, the Board may adopt and pay for any expedient measure which will aid in getting injured workmen back to work and lessen any handicap resulting from their injuries.

Function of Physicians

Choice of Doctor

In all provinces, a workman may initially select his own physician but the legislation provides that the Board shall decide in any cases where a difference of opinion arises. If required by the Board, the workman must submit to examination by a medical referee chosen by the Board.

In New Brunswick, Nova Scotia, Ontario and Saskatchewan, a workman must, if required by his employer, submit to a medical examination by a physician chosen and paid by his employer but only in accordance with the regulations of the Board.

In Alberta, in cases of dispute, the Board, after consulting the workman's physician, must nominate two recognized specialists in the class of injury or ailment for which compensation is claimed and the workman may select one of them to conduct the examination.

In all provinces, the fees for medical aid are fixed by the Board.

Submission of Claims for Compensation

Each provincial Act, except in Quebec and Alberta, requires the employer to notify the Board within three days of any accident to a workman involving loss of pay or necessitating medical aid. The Quebec Act allows eight days and the Alberta Act 24 hours for this purpose. When an employer indicates that a physician has rendered medical aid, the attending doctor is sent an account form on which his claim for remuneration is made. Facts and conditions justifying payment of compensation or medical aid must be shown before a claim can be allowed. The physical disability resulting from the accident, with a clear and definite description of the objective signs and symptoms, must be shown. In corresponding with the Board, the name and address of the workman and the claim number should always be given.

It is important that accident reports be forwarded by the employer, the physician and the employee to the Board as soon as possible following the occurrence of an injury involving loss of pay or necessitating medical aid. The Board assesses each claim upon the reports submitted and unnecessary hardship is imposed upon an injured workman if settlement of his claim is delayed through tardy submission of reports.

Reporting of Occupational Diseases

The Ontario Factory, Shop and Office Buildings Act requires the reporting of occupational diseases by the physician to the Department of Health and by the employer to the Department of Labour. This regulation is important in that the complete reporting of occupational diseases would enable the Department of Health to distribute periodic reports of the sources and incidence of occupational diseases.

Occupational Diseases

Each Compensation Act contains a table of occupational diseases for which compensation is payable. Beside each disease is listed the industrial processes in which the disease must arise to be compensable. While this might appear to restrict the scope of the Acts, certain cases of occupational disease not listed in an Act may be interpreted by the Board as being compensable, depending on other sections of the Act. Therefore, a claim should be presented for the Board's consideration whenever a disability is considered to be peculiar to or characteristic of a worker's occupation.

The table on the following page contains a complete list, as of September 1948, of diseases for which compensation is payable under the Workmen's Compensation Act in each province except Ontario and Prince Edward Island. In Ontario, in addition to those diseases listed under the Act, any disease peculiar to or characteristic of a particular industrial process, trade or occupation, and resulting in disability, is compensable.

COMPENSABLE OCCUPATIONAL DISEASES

Disease	B.C.	Alta.	Sask.	Man.	Que.	N.B.	N.S.
Acetates, alcohols, poisoning by a solvent containing	x						
Ammonia poisoning or its sequelae			x			x	
Ankylostomiasis	x		x	x		x	x
Anthrax	x	x	x	x	x	x	x
Arsenic posioning or its sequelae	x	x	x	x	x	x	x
Asbestosis					x		
Benzene (benzol) poisoning and poisoning by its homologues, nitro and amino derivatives, aniline and others, or the sequelae	x	x	x		x		
Brass, zinc or nickel poisoning or its sequelae			x		x		
Bronchial asthma, due to organic dust in handling grain, furs, feathers or wool	x						
Bronchitis, tracheitis, pulmonary oedema or gastric irritation caused by contact with welding gases and fumes	x						
Bursitis			x		x		
—acute, elbow	x					x	x
—prepatellar	x						
Cadmium poisoning			x		x		
Cancer, epitheliomatous or ulceration of the skin or of the corneal surface of the eye due to tar, pitch, bitumen, mineral oil or paraffin, or any compound, product or residue of any of these substances			x				x
Carbon bisulphide poisoning or its sequelae			x			x	
Carbon dioxide poisoning or its sequelae	x		x			x	
Carbon monoxide poisoning or its sequelae	x		x		x	x	
Cedar, hemlock, spruce or alder poisoning	x						
Cellulitis, subcutaneous hand	x	x					x
Chlorinated hydrocarbons (carbon tetrachloride, trichlorethylene, tetrachlorethane, trichlornaphthalene, methyl bromide and others) poisoning by, or the sequelae	x		x		x		
Chlorine poisoning			x				
Chrome poisoning	x		x		x		
Compressed air illness	x		x		x	x	

COMPENSABLE OCCUPATIONAL DISEASES—*Concluded*

Disease	B.C.	Alta.	Sask.	Man.	Que.	N.B.	N.S.
Conjunctivitis and retinitis due to electro and oxy-acetylene welding	x		x	x	x	x	
Cyanide poisoning			x				
Dermatitis (venenata) (See Below).	x	x	x	x	x		x
Formaldehyde and its preparations, poisoning by			x				
Frostbite							x
Glanders		x	x			x	
Infected blisters	x		x		x		
Inflammation of the synovial lining of the wrist joint and tendon sheaths	x		x		x		
Lead poisoning or its sequelae	x	x	x	x	x	x	x
Mercury poisoning or its sequelae	x	x	x	x	x	x	x
Miners', stone workers', or grinders' phthisis			x				
Nitrous fumes, poisoning by, or its sequelae			x	x			
Petroleum products and their fumes, respiratory, gastro-intestinal or physiological nerve and eye disorders due to contact with			x				
Phosphorus poisoning or its sequelae	x	x	x	x	x	x	x
Pneumoconiosis	x	x	x		x		
Silicosis	x	x	x	x	x	x	x
Solvent volatile, poisoning by	x		x				
Sugar, infection by handling						x	
Sulphuric, hydrochloric or hydrofluoric acid, poisoning by			x				
Sulphur poisoning	x					x	
Tooth erosion due to exposure to acid mist	x						
Tuberculosis, pulmonary in hospitals and sanitoria under the Act	x						
Vascular disturbances in the upper extremities due to continuous vibration from pneumatic or power drills, riveting machines or hammers	x		x				
Wood alcohol, poisoning by			x				
X-rays, radium or other radio-active substances, any disease or disability due to exposure to	x		x		x		
Zinc refining, ulceration of the mucous membranes of the nose or throat due to acid fumes in	x						

Dermatitis

The definition of dermatitis in the list of compensable diseases varies in each province as follows:

British Columbia

Dermatitis caused by direct contact with cheese, sugar or cereals, poison ivy or poison oak, alkalis or soaps, hides, uncooked meats, fish or poultry, cedar bark fluff or palco wool, cement, copra; glue in manufacture of plywood, airplanes or in woodworking plants; cutting-oil in machine work; oil containing alcohol, formaldehyde, phenol or phenol derivatives; any process in the canning or packing of fruit or vegetables, using teak or mahogany dust, using thioglycolates or other irritant substances in the hair dressing industry; any operation in preserving wood products involving the use of acids, alkalis or acids and oils; exposure to cyanide, magnesium or its alloys; manufacture of brooms or brushes; manufacture or use of rock-wool, slag-wool or glass-wool.

Alberta

Infection or inflammation of the skin or contact surfaces due to oils, cutting compounds or lubricants, dust, liquids, fumes, gases or vapours.

Saskatchewan and Nova Scotia

Dermatitis venenata in any industrial process involving the handling or use of irritants capable of causing or producing dermatitis venenata.

Manitoba

Occupational dermatitis and occupational ulcerations and infections of the skin caused by harmful and noxious factors pertaining specifically to the employment carried on by the following classes of workers: abattoir and stockyard workers; boiler washers (steam); bricklayers; cement (Portland) workers; dyers (in clothes cleaning establishments); furriers and fur workers; laundry workers; lime workers; masons; metal platers (including galvanizers and bronzers); plasterers (including lime white-washers); painters (including paint mixers and French polishers); printers (including engravers, electrotypers and lithographers); tanners (leather including hideworkers).

Ontario

(The blanket coverage of occupational diseases obtaining in Ontario applies to any dermatitis peculiar to or characteristic of a particular industrial process, trade or occupation.)

Quebec

Dermatitis venenata in any process involving the use of acids and alkalis or acids and oils capable of causing dermatitis venenata.

New Brunswick

(With the exception of infection by handling sugar, no specific definition of dermatitis is given in the Workmen's Compensation Act of New Brunswick.)

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