

MANUAL

PLAINTIFF'S
EXHIBIT
B&R-111

ACCIDENT
PREVENTION
MANUAL
for Industrial Operations

5th EDITION



NATIONAL SAFETY COUNCIL Chicago, Illinois 60611

COPYRIGHT 1964 NATIONAL SAFETY COUNCIL

All Rights Reserved

No portion of this book may be reproduced by any process without written permission of the National Safety Council, 425 North Michigan Avenue, Chicago, Ill. 60611

Library of Congress Catalog Card Number: 64-24124

25M106463

Printed in U.S.A.

Stock No. 121.35

THE LIBRARY
THE UNIVERSITY
OF TEXAS

PREFACE

The dominant theme of this Manual is safety; the pervading purpose is accident prevention. Industrial accident prevention has as its primary objective the prevention of injuries to persons; an auxiliary goal is the prevention of damage to equipment and other property.

The favorable reception of the first four editions of this book by safety engineers and others who have the responsibility for accident prevention has provided the incentive for this revised Fifth Edition.

The object was to update the material and extend its scope while being more specific in both word and illustration to reduce the bulk of the book. For improved readability, a slightly larger and clearer typeface has been used. For easier reference, the index includes as many synonyms as possible, but it has been considerably shortened by including only major references under each subject. If a subject is discussed on successive pages, only the first page is referenced.

Two new chapters have been developed: "Audio-Visual Aids" and "Industrial Noise." Many others have been completely revised. For more logical organization, some chapters have been retitled and material has often been regrouped.

Some duplications will be found among the sections. The Editors think this facilitates the use of a section and avoids the necessity for frequent reference to other parts of the book. Where a significant amount of additional information is in another chapter, cross references are provided.

The preparation of each chapter was directed by a National Safety Council staff specialist in the field. In some cases, new material was developed by recognized authorities who are identified by the footnote on the first page of each chapter. All worked with many collaborators. For all of these men, the most satisfying reward is the knowledge that they have contributed to the more effective performance of the industrial safety men of today, and to the education of future safety men.

To increase the effectiveness of those responsible for industrial accident prevention, much detailed material had to be included, but it is not the purpose of this Manual to serve as a complete handbook of all design, fabrication, operation, inspection, and management principles. Rather, it attempts to set forth the important points to be considered when evaluating an industrial accident prevention program including the safe operation of equipment and processes.

This Manual does not constitute an official code or standard, and it does not obviate prescribed regulations or minimum safe practices. When practices are described, it is not intended that they supplant other practices that have proven satisfactory, nor is their description intended to discourage innovation and originality in contributing to ever-more-effective industrial accident prevention.

No one prescribed safety formula can be superimposed on all accident-prevention programs. As a suggestive guide the Manual necessarily speaks in broad and general terms; its contents must be adapted to the specific circumstances of the particular operation. Ultimate responsibility, therefore, must rest in the selection of the technique or method for correcting any given condition or situation.

THE EDITORS

INDUSTRIAL HYGIENE

the larger particles are trapped by the hairs in the nose. Other dust particles are removed from the air as it passes over the moist mucous membranes of the nose, throat, and other portions of the upper respiratory system. The bronchi and other respiratory passages are covered with a large number of tiny, hairlike *cilia* or microscopic whiplashes, which aid in the removal of dust trapped on these moist surfaces. The *cilia*, all bending in one direction, make a fast stroke toward the mouth and a slower return stroke. This action tends to push mucous and deposited dust upward to the mouth so that the particles can be expectorated or swallowed.

Retention of dust

Many studies have been made in an effort to determine the amount of dust that is retained in the lungs, but there is no simple answer to this question. It has been shown that the size of the dust particles, the rate of respiration, the density of the dust in the air, the efficiency of the dust-catching mechanism, and probably many other factors are involved.

Sizes of particles inhaled

Although an occasional dust particle of larger size will enter the lungs, particles less than $3\ \mu$ in diameter are the most likely to do so and thus have the greatest opportunity to cause a physiological reaction. In silicotic lungs, for example, dust particles under $3\ \mu$ greatly outnumber larger ones, and many particles are less than $1\ \mu$.

In the case of very fine fibrous asbestos dust, an exception occurs in the size of particles inhaled. Many fibers up to $100\ \mu$ long have been found in the lungs of asbestos workers at autopsy. A typical fibrosis caused by asbestos is produced by fibers that are 20 to $50\ \mu$ in length, but only a few microns wide.

Physiological Effects of Dusts, Fumes, and Mists

The physiological reactions caused by the inhalation of airborne particulate matter will vary with different types of dusts, fumes, and mists. The reactions include:

1. The cardiopulmonary reaction which consists of the pneumoconioses, such as *silicosis* and *asbestosis*. In certain cases,

specific types of lung pathology result and the heart may be affected (*cor pulmonale*) when the fibrosis is advanced. In other cases, there is mainly just an accumulation of a relatively inert dust in the lungs.

2. The systemic reactions which are caused by toxic dusts of such elements as lead, manganese, cadmium, and mercury, by their compounds, and by certain organic compounds.
3. Metal fume fever which results from the inhalation of finely divided and freshly generated fume of zinc or possibly of magnesium or of their oxides. This is a transient condition.
4. Allergic and sensitization reactions which may be caused by inhalation of, or skin contact with, such materials as organic dusts from flour, grains, and some woods and dusts of a few organic and inorganic chemicals. There are also bacterial and fungal infections which occur from inhalation of dusts containing active organisms, such as wool or fur dust containing anthrax spores or wood bark or grain dust containing parasitic fungi. (See the section "Biological Agents and Other Occupational Infections" later in this chapter.)
5. Irritation of the nose and throat, which is caused by acid, alkali, or other irritating dusts or mists. Some dusts such as soluble chromate dusts may cause ulceration of the nasal passages or even lung cancer.
6. Damage to internal tissues, which may result from inhaled radioactive materials such as radium and its daughter products and from other radioisotopes that emit highly ionizing radiation. (See Chapter 42, "Ionizing Radiation.")

Pneumoconioses

Pneumoconiosis comes from three Greek words that mean "lung," "dust," and "abnormal condition." The present generally accepted meaning of the word is merely "dusty lung." The kind of dust inhaled determines the type of condition or injury. A number of organic dusts are capable of producing lung diseases, but not all these diseases are classified as pneumoconioses because they are not all a "dusty condition" of the lung.

INDUSTRIAL HYGIENE

In very rare cases, enough dust has been inhaled to cause mechanical blockage of the air spaces. (Flour dust has been known to cause this condition.) Some dusts may be essentially inert and remain in the lungs indefinitely with no recognizable irritation, and a few (like limestone dust) may be gradually dissolved and eliminated without harm.

Silicosis is the most important lung disease caused by the inhalation of mineral dust. It is well-known in industries where crystalline free silica dust is present, such as foundries, glass manufacturing, granite cutting, mining, and tunneling in quartz rock. It is found throughout the world, and in the past it has had many names, such as miner's asthma, grinder's consumption, miner's phthisis, potter's rot, and stone-mason's disease. The same occupational disease, however, is meant by all these names, and it is caused by dust from crystalline free silica, usually quartz.

Silicosis has been defined as "a disease due to breathing air containing silica characterized anatomically by generalized fibrous changes and the development of milium nodulation 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 by characteristic X-ray findings."

Silicosis has been known to manifest itself after widely differing periods of exposure to silica dust. Apparently, development of the disease depends upon:

- The amount and kind of dust inhaled.
- The percentage of free silica contained in the dust.
- The form of the silica.
- The size of the particles inhaled.
- The duration of the exposure.
- The powers of resistance of the individual concerned.
- The presence or absence of a complicating process such as infection.

Many theories have been advanced over the years to explain why crystalline free silica acts as it does in the lungs. It is now believed that the fibrosis produced is caused by the hardness or sharpness of the

particles, but by a combination of slight solubility with a physiochemical effect and an immunological effect—but no one is certain of the exact mechanism of the disease. Experimental work on the reasons for the development of silicosis is still going on in various parts of the world. If the precise mechanism of silicosis could be determined, better medical preventive measures might be developed and possibly a cure could be found.

Amorphous free silica differs from crystalline free silica in physical structure and in physiological effects. In the amorphous state, molecules of silica are randomly oriented and may be naturally converted to opal and diatomaceous earth (kieselguhr) or artificially converted into such forms as silica gel, silica fume, and fused silica or quartz.

If amorphous silica is heated to a high temperature, as in calcining, forms of crystalline free silica called cristobalite and tridymite result, intermediate forms of amorphous silica are known as crypto-crystalline (ultra-microcrystalline). Inhalation of these crystalline forms can readily cause diatomite pneumoconiosis.

When diatomaceous earth is calcined, particularly in the presence of a trace of alkaline flux, appreciable quantities are converted to cristobalite. As a result of studies made by the U.S. Public Health Service, it has been recommended that the threshold limit value for crude or amorphous diatomite be placed at 20 mppcf (million particles per cubic foot), but that the atmospheric concentration for dust containing cristobalite be kept under 5 mppcf.

Various commercial products containing particles of silica under 1 μ in size are available. The physiological effects of these products have not been well defined. Until more experience with human beings is available, these products should be handled with care.

*American Public Health Association. 1790 Broadway, New York City. "Report (Joint) of the Committee on Pneumoconiosis and the Committee on Standard Practices in Compensation of Occupational Diseases." Year Book, 1933.

Free silica is uncombined silicon dioxide (SiO_2). Silicates contain silicon and oxygen combined with other elements in more complex molecules. The SiO_2 reported in chemical analyses for mineral and geological reports is the *total* of the silicon dioxide present, both the free silica (if present), and the silica combined in the mineral. Such analyses are not reliable indications of the silicosis potential of the material, because it is the uncombined or free silica that is most important in industrial dust exposure. So that an exposure can be properly evaluated, the percentage of uncombined silica must be determined by petrographic analysis using a polarizing microscope or, preferably, by X-ray diffraction analyses and special analytical chemical procedures.

There has been some experimental evidence that some dusts may tend to inhibit the action of silica on the body, but this inhibiting action is so slight and uncertain that it must be discounted in practice. In fact, there is also evidence that the nonsiliceous components of a dust mixture containing free silica may provoke a disabling condition more severe than that caused by the silica acting alone.

With the exception of asbestos and some talcs, the silicate dusts do not ordinarily cause a serious disabling lung condition such as is produced by free silica. Much higher levels of silicate dusts than of free silica dust can be tolerated. In many industries, men have worked with silicate dusts that contained no free silica without development of disability or of nodulation in the lungs. The X-ray may show shadows indicating dust deposits in the lungs, but the pneumoconiosis is essentially harmless. However, partially disabling pneumoconioses have been reported where men have worked for long periods of time in very high concentrations of certain silicate dusts. Disabling pneumoconioses from exposure to abnormally high concentrations of mica, tremolite talc, and kaolin dusts have been described in the literature. The clinical signs are not the same for these silicate dusts as for free silica, but the symptoms can be marked.

Asbestosis. Several minerals having a fibrous character are classed as "asbestos"—hydrated silicates of magnesium with variable amounts of iron, calcium, sodium, potassium, and aluminum present as impurities.

The fine airborne fibers of asbestos can pass through the upper respiratory tract to the lower parts of the lungs to cause irritation and to form "asbestos bodies" where the fibers are encapsulated. This diffuse fibrosis probably begins as a "collar" about the terminal bronchioles. There is evidence that other minerals having a fibrous character (except glass fiber) can produce a reaction similar to that of asbestos.

Following a study by the U.S. Public Health Service of the asbestos textile industry,* it was recommended that the dust concentration be kept at less than 5 mppcf to prevent asbestosis. Evaluation of an exposure to asbestos dust is based on the *total* amount of dust, since the concentration of injurious fibers will be kept within safe limits if the fine dust is kept below the suggested threshold limit.

Miscellaneous pneumoconioses. Even though a dust is classified as harmless, amounts above the TLV can lead to trouble by causing a pneumoconiosis, mechanically irritating the walls of the respiratory system, or interfering with ordinary lung processes. Mica dust and kaolin dust are two good examples of dusts that ordinarily are considered benign but amounts above the TLV can cause a troublesome pneumoconiosis. Mica pneumoconiosis has been observed in grinding operations where mica dust, but no free silica was present. There were marked changes in the X-ray pictures of the lungs and some disability. The cases occurred where the dust exposures were massive over many years.

Toxic dusts and fumes

Systemic reactions are caused by toxic dusts and fumes of various elements and their compounds and by certain organic compounds. All metallic fumes are irritating, especially when freshly generated. Industrially important metals and their compounds that can have a toxic effect when the dust or fumes are inhaled include arsenic, antimony, cadmium, chromium, lead, manganese, mercury, selenium, tellurium, thallium, uranium, and a few others.

*U.S. Public Health Service, Washington, D.C. "A Study of Asbestosis in the Asbestos Textile Industry." Bulletin No. 241. 1938.

T
nesi
Only
and
A
with
tion
toxi
disr
are
amo
and
level
then
In
is th
into
pou
hygi
enco
ing,
work
L
anim
daily
lead
exce
lead
mul
of r
conc
fore
caus
to a
B
ic di
of th
bery
pour
tion
pour
lium
temi
dust
conc
can
Hyg
prot
W
ciall
abra
form
ple
time
M
caus

INDUSTRIAL HYGIENE

The effect of some metals, such as magnesium and zinc, appears to be transient. Only limited data are available on the exotic and rare earth metals.

Although the dusts and fumes from metals with low toxicity do not need as much attention as the dusts and fumes from highly toxic metals, they should not be neglected or disregarded. The metals with low toxicity are more readily controlled because greater amounts can be tolerated, but their dusts and fumes should be kept at reasonable levels since excessive amounts of any of them can be harmful.

Inhalation of the dust of lead compounds is the most common mode of entry of lead into the system, and ingestion of lead compounds can add to the problem if personal hygiene is poor. Workers therefore should be encouraged to wash thoroughly before eating, and lunchrooms should be apart from work areas.

Lead is a normal constituent of plants and animals, and people ingest and excrete it daily even though they are not exposed to lead in their daily work. When intake rates exceed the normal excretion rates, however, lead builds up in the body. When the accumulation reaches a sufficient level, symptoms of poisoning or intoxication appear. The concentration of airborne lead should therefore be kept below the threshold limit because of lead's high toxicity and its tendency to accumulate in the human system.

Beryllium intoxication is a severe systemic disease that can result from the inhalation of the dust or fume of metallic beryllium, beryllium oxide, and soluble beryllium compounds. (There is no evidence of intoxication from the ingestion of insoluble compounds.) Only the inhalation of the beryllium-bearing dusts or fumes produces systemic disease. Accordingly, control of such dusts and fumes to keep them below the concentration specified by ACGIH (American Conference of Governmental Industrial Hygienists) threshold limit values is a basic protective measure.

When the soluble salts of beryllium, especially beryllium fluoride, contact cuts or abrasions on the skin, deep ulcers may be formed which heal very slowly, and complete surgical excision of the ulcer is sometimes required to effect healing.

Metal fume fever is an acute condition caused by a brief high exposure to the fresh-

ly generated fumes of metals such as zinc, magnesium or their oxides. Symptoms appear from 4 to 12 hr after exposure and consist of fever and shaking chills. There is complete recovery usually within one day, and ordinarily the employee can return to the same job without recurrence. However, after a period in which there has been no contact with the fume, for example, after a layoff, resumption of exposure is likely to bring on an attack.

Metal fume fever is caused by heavy concentrations of fumes. Zinc oxide fume is the most common source, but cases caused by the inhalation of fumes from magnesium oxide, copper oxide, and other metallic oxides have also been reported. The condition does not occur from the handling of these oxides in powder form. Apparently, it results only from the inhalation of extremely fine particles freshly formed as fume (called a "nascent fume"). Cadmium, mercury, and other metals may also produce a fever followed by the toxic effects of the element.

Organic Solvents

The widespread industrial use of organic solvents presents a major problem to the industrial hygienist, the safety engineer, and others charged with the responsibility for maintaining a safe, healthful working environment. Getting the job done without hazard to employees or property is dependent upon the proper selection, application, handling and control of solvents and an understanding of their properties.

Labeling

The labeling of solvents to indicate their properties and health and fire hazards is an extremely important method for recognizing and evaluating the hazards. In fact, if a solvent is not properly labeled, it should not be used. The purchasing department can greatly help the safety department by notifying suppliers that only properly labeled solvents will be accepted in the plant.

Uniformity in language and layout is desirable to simplify understanding of solvent use. The MCA's *Guide to Precautionary Labeling of Hazardous Chemicals*, Manual L-1,* recommends the following subject mat-

*Manufacturing Chemists Assn., 1825 Connecticut Ave. NW., Washington, D.C.

ELEMENTS OF INDUSTRIAL TOXICOLOGY

in the handling of wet dross from a light metals plant, in the cleaning of tank cars which have held sulfuric and hydrochloric acids, in the cleaning of pipes with an acid contaminated with arsenic, in the precipitation of cadmium with metallic zinc, and in other operations.

Arsine rapidly destroys the red cells. After it has been inhaled, the first signs are those of lack of oxygen. After some time the body begins to excrete the damaged red cells, which appear as a dark or bloody urine. The kidneys may then become too clogged to operate at all. Arsine, in very small amounts, is also capable of producing a chronic type of poisoning, but it is more typically a source of acute episodes.

The possibility of arsine formation should always be considered when there is freshly formed hydrogen around ores of the heavy metals, since arsenic is widely distributed in small amounts and only a trace of it is required to produce enough arsine to be troublesome.

One of the more common sources of arsine incidents in recent years has been the handling of dross from the aluminum refining of metals. Probably the most significant observation about these cases, aside from the fact that the moisture in the atmosphere is enough to form arsine, is the fact that the people involved in them have not been aware of the garlic-like odor which is supposed to be a warning of the presence of arsine.

See Hygienic Guide.

Asbestos is a hydrated magnesium silicate found in the minerals chrysotile, amianthus, actinolite, and tremolite. Practically all the commercial asbestos is either chrysotile or amianthus, and about 90 per cent of that used in this country is chrysotile. It is used almost exclusively for heat insulation or for textiles which must resist high temperature, such as heat-resistant clothing and brake linings.

Inhalation of excessive quantities of asbestos fiber can produce a fibrosis in the lungs similar to that found in silicosis but somewhat milder and usually not so rapidly progressive. Like silicosis, the condition develops and advances slowly and is equally resistant to treatment. There is also good evidence that inhaled asbestos causes lung cancer.

See Hygienic Guide.

Barium, in the form of its soluble salts, is toxic on both ingestion and inhalation and highly caustic when applied to the skin. The carbonate and sulfide are sufficiently soluble to be toxic, although not very caustic. The sulfate, which is used as a contrast medium in X-ray work, is too insoluble to show any toxicity, and soluble sulfates are specific antidotes for ingested barium salts.

Since these salts produce violent stimulation of all muscle, upon ingestion they produce severe disturbances of the digestive system. After absorption, the barium salts increase blood pressure by constriction of the arterial muscle, slow down heart beat by an action similar to that of digitalis, and first excite and then paralyze the central nervous system. In spite of the high inherent toxicity, there are few cases of industrial poisoning with barium.

Barium is a heavy metal, and the dust of the insoluble salts will collect in the lungs and give rise to the condition known as baritosis. Its nodular appearance on X-ray film may be mistaken for that of silicosis. Men showing the sign of baritosis are apparently not disabled or inconvenienced in any way.

See Hygienic Guide.

Benzene (benzol) is a colorless, flammable, volatile liquid with a rather pleasant aromatic odor. Its fire hazard is between ethyl alcohol and ethyl ether. The greatest hazard is that of chronic poisoning by inhalation of comparatively small amounts over a long period of time. It is one of the two or three most dangerous organic solvents in commercial use, and acts primarily on the blood-forming organs, producing severe anemia, bleeding under the skin, and great reduction in the ability of the blood to clot.

The most convenient measurement of damage is given by a blood count. People who may be exposed to benzene vapor should have blood counts at regular intervals and significant changes in the count should be closely investigated.

Headaches, dizziness, fatigue, loss of appetite, irritability, nervousness, nosebleed, and other signs of abnormalities of the blood may or may not be present in chronic poisoning.

A single heavy exposure to benzene vapor may produce a serious acute effect, similar