

FILE NAME: Brakes (BRK)

DATE: 1945 Apr 15

DOC#: BRK197

DOCUMENT DESCRIPTION: Industrial Hygiene Manual - Air Technical
Service Command

INDUSTRIAL HYGIENE MANUAL

ATSC No. 25-0-1

INDUSTRIAL TOXIC AGENTS

CHEMICAL FORMULA AND SYNONYMS

PRINCIPAL PROPERTIES

PRINCIPAL INDUSTRIAL USES

POISONING—SYSTEMIC

**MAXIMUM PERMISSIBLE CONCENTRATIONS
OF ATMOSPHERIC CONTAMINANTS**



U.S. Army Air Forces.

Office of the Surgeon

HEADQUARTERS AIR TECHNICAL SERVICE COMMAND

WRIGHT FIELD, DAYTON, OHIO

15 April 1945

FOREWORD

1. *Purpose and Scope:* This manual supplement is applicable to Medical Corps officers, Medical Service Corps officers, and enlisted personnel engaged in the control of occupational diseases, and will be used as a training aid for all industrial hygiene activities and tactical units within the AMC.

2. *General:* The contents of this manual describe toxic agents, suggested control measures, and health hazards, and include pictures of processes.

3. *Responsibility:* The office of the Surgeon, Hq AMC, is responsible for the preparation and revision of this manual.

4. *Initial Distribution:* Initial distribution of this manual will be accomplished by the office of the Surgeon, Hq AMC.

5. *Additional Copies:* Requests for additional copies of this manual should be submitted on AAF Form No. 104B, through channels, to the Commanding Officer, AF Technical Base, Wright Field, Dayton, Ohio, attn: World-Wide Publications Distribution Section (TSWPD).

BY COMMAND OF GENERAL McNARNEY:

J. F. McCLENDON
Colonel, USAF
Adjutant General

OFFICIAL:

J. F. McCLENDON
Colonel, USAF
Adjutant General

INDUSTRIAL HYGIENE MANUAL SUPPLEMENT

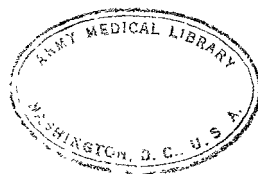
Pictures of Processes

Processes

Toxic Agents

Suggested Control Measures

Health Hazards



HEADQUARTERS AIR MATERIEL COMMAND
WRIGHT FIELD, DAYTON, OHIO

TABLE OF CONTENTS

<i>Section</i>	<i>Page</i>
I. Aero Repair.....	1
II. Assembled Engine Cleaning.....	2
III. Battery	4
IV. Blacksmith	4
V. Block Test.....	6
VI. Carburetor and Ignition.....	7
VII. Electroplating	8
VIII. Engine Cleaning.....	10
IX. Engine Disassembly.....	13
X. Foundry	15
XI. Heat Treating.....	16
XII. Hydraulic	17
XIII. Instrument Repair.....	18
XIV. Luminous Dial Repair.....	20
XV. Machine Shop.....	22
XVI. Minor Repair.....	23
XVII. Motor Vehicle and Miscellaneous Repair.....	24
XVIII. Office Appliances.....	26
XIX. Paint and Dope.....	27
XX. Plexiglas	29
XXI. Propeller	30
XXII. Radiator and Tank.....	31
XXIII. Rubber Tank Repair.....	33
XXIV. Sandblasting	35
XXV. Spark Plug Cleaning.....	36
XXVI. Spray Painting.....	37
XXVII. Trichloroethylene Degreasing.....	39
XXVIII. Welding	40

NL

Substance	Concentration
Tetrachloroethane	10 p.p.m.
Tetrachloroethylene	200 p.p.m.
Toluene	200 p.p.m.
	100 p.p.m.
Trichlorethylene	200 p.p.m.

Substance	Concentration
	100 p.p.m.
Turpentine	700 p.p.m.
	200 p.p.m.
Xylene	200 p.p.m.
	100 p.p.m.
Zinc oxide fume.....	15 mg/cu.m.

DUSTS

Millions of particles per cubic foot of air—light field count.

Substance	Concentration
Alundum	15
Asbestos	15
	5
Carborundum	15
Cement	15
Feldspar	10
Foundry	20
	15
	12
Granite	25
	10
Mica	50
	10
Nuisance	50
Pottery	4
Organic	50
Pyrophyllite talc	25
	10

Substance	Concentration
Silica (based on percentage of free silica in the dust)	
Count x%.....	
10%	50
("low")	50
10%	10
25—35%	10
"medium"	20
"high"	
over 75%	
over 90%	
Silicates	15
Slate	50
	15
Soapstone	50
Talc	50
	15
Total dust.....	50

SILICOSIS AND SIMILAR DUST DISEASES

The pneumoconioses represent a class or type of diseases of the lungs which develop slowly as a result of occupational exposure. The term "pneumoconiosis" may be applied to any pathological condition of the lung produced by the inhalation of dusts. It is common further to classify pneumoconiosis according to the chief constituents of the dust producing the condition; for example, silicosis produced by silica, asbestiosis produced by asbestos, siderosis produced by iron, anthracosis produced by carbon particles, etc. Since silicosis is the most important and control measures effective in preventing silicosis are applicable in the prevention of other forms of pneumoconiosis, this discussion will be limited chiefly to reporting the cause and prevention of Silicosis.

Definition of Silicosis: A disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes, and the development of miliary 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.

A. Principal Properties:

1. It occurs in two forms, free and combined. The free silicas as a group are definite compounds in the form of SiO_2 . The combined forms are silicates. Of the free silicas which occur in nature, quartz is the most common. Quartz is a hard mineral and chemically resistant to reagents. Other forms of free silica are Opal ($\text{SiO}_2 \cdot \text{H}_2\text{O}$), amorphous hydrated form, tridymite, cristobalite, and siliceous glass, or vitreous silica.

B. Principal Industrial Uses:

1. Owing to the fact that the earth's crust contains so great an amount of silica, it is obvious that those occupations concerned with the driving of tunnels, development of highways, and mining are frequently associated with a silicosis hazard. Those industries that have to do with the processing and industrial use of mineral products, such as the smelting and refining of ores, the use of sand and gravel for structural purposes, the carving of stone, particularly granite, the manufacture and use of certain abrasives, and the processing of the various forms of free silica, expose workers to this hazard.

C. Pulmonary Effects:

1. Modes of entry
 - a. Inhalation of free silica particles 10 microns and less in size.
2. Symptoms
 - a. The subjective and objective signs of silicosis vary according to the rate of development and degree of pulmonary fibrosis, and when infection is present, according to the type, extent, and duration of the complicating pulmonary infection. From a medical point of view, there is no sharp line of demarcation as to stages of silicosis. However, by means of a description by stages, the progressive nature of the disease may be better illustrated.
 - b. *First Stage:* Chest film indicates early nodular fibrosis, the individual may or may not exhibit such clinical evidence as slight shortness of breath upon exertion, and some cough. The general appearance is that of a healthy individual, and there is no appreciable decrease in capacity to perform usual duties.
 - c. *Second Stage:* More advanced nodulation is shown by X-ray, and there may be some evidence of localized aggregation of nodules and pleural thickening. Definite shortness of breath upon exertion, cough more pronounced, chest movements sluggish, and expansion usually decreased. The individual may be able to continue at his usual job, although less effectively.
 - d. *Third Stage:* Fibrous changes as shown by X-ray film are further accentuated. Nodules are larger and assume conglomerate form, showing large shadows corresponding to areas of dense fibrosis. Shortness of breath is marked and distressing upon slight exertion. Cough is increased, and may be dry, but is usually productive. Chest expansion is decreased even upon forced inspiration. The individual's capacity for work is seriously and permanently injured.
 - e. The diagnosis is based upon historical data, including the complete occupational history, past and present medical history; clinical, laboratory and X-ray findings. Except by autopsy, silicosis cannot be diagnosed definitely until X-ray examination reveals characteristic shadows of both lung fields. Other evidence of complicating pulmonary disease should be included in the diagnosis.

3. Predisposing Causes

- a. Age and geographical in itself is no great factor.
- b. Previous exposure seems to have a definite influence in predisposing to silicosis.
- c. Role of Infection: Infection developing along the respiratory tract may be of importance. Acute pneumonic conditions as well as the more chronic lung changes such as chronic bronchitis, bronchiectasis, bronchiolectasis, emphysema, asthma, and pleurisy all tend to decrease the ability of the lung to rid itself of foreign materials, through lessened lymphatic drainage and decreased power to force the bronchial secretions and foreign matter from the lungs.

4. Pathology

- a. Tissue reaction to dust: It has been shown that silica in solution or non-crystalline form exerts a toxic action upon the tissues which leads to the proliferation of fibroblastic cells, while other dusts have been either completely absorbed, leaving no scar tissue, or have remained unaltered in the form in which they were injected.
- b. Size of Dust Particles: Since dust, to exert its harmful action, must enter the finer divisions of the lung, the particle size of the atmospheric dust bears a definite relationship to the injurious effect produced. The silica must be present in the air in particles small enough to enter the finer air spaces and of such dimensions that the phagocytic cells may engulf them. The natural defenses of the respiratory tract probably prevent many particles larger than 10 microns from ever reaching the finer divisions of the lung, and such as do are likely to be expelled with the bronchial secretions. The soluble silica plays a definite part in the production of the disease, and the size of the particle also affects the rate of solution, due to the fact that the smaller the particles, the greater the total surface area exposed to the action of solvents.

5. Prognosis

- a. In the main, the prospects for recovery for cases of Silicosis are not favorable. Even when removed from dust exposure, the disease often tends to progress and to be complicated with tuberculosis. If the individual can be removed from his dusty occupation before serious damage to the lungs has occurred, his chances of maintaining his working capacity are fair.

6. Standard of Dustiness

- a. In an investigation of the granite cutting plants conducted by the U. S. Public Health Service, most of the workers were found to be exposed to an average of about 60,000,000 particles of dust less than 10 microns in the greatest diameter per cu. ft. of air. The dust contained 70% silica, of which about 35% was in the form of quartz or free silica. Under such conditions, there was an almost universal occurrence of silicosis among those exposed twenty years or longer, and a large proportion of workers developed pulmonary tuberculosis. It was found in this investigation that little or no evidence of lung injury could be observed in workers exposed to a concentration of between 9,000,000 and 20,000,000 particles, so that this report suggests in broad limits a tentative concentration of air dustiness which can be tolerated over a period of years without serious consequence. Obviously such an estimated limit would be valueless where conditions are dissimilar. Most accepted permissible limit is 5 million particles per cu. ft. (size range 5 to 10 microns). While obviously dust containing 90% silica is more harmful than dust containing 60%, it is not certain that the relative harmfulness is one of strict arithmetical proportion. Another value of dust counts lies in their use as a measuring rule to indicate the relative efficiency of devices and the method adopted to suppress dust within an industry.

D. Sanitary Corps Officer:

1. Measures for control of dust hazard.
2. Maintenance of existing control measures.
3. Supervision of protective devices for workers exposed to dust hazard.

E. Medical Control:

1. Pre-employment placement examination should include X-ray of lungs. All persons who have chronic infections of the upper respiratory tract should not be exposed to dusty occupations.
2. Periodic Examinations
 - a. A yearly complete physical examination which should include X-ray of lungs.

F. Suggested Silica Periodic Check:

1. Name, Address, Age, Race, Sex, Department, Age Began Work and Number of Years Worked.
2. Present:
 - a. Specific Occupation
 - b. Specific Industry
 - c. Number Years In:
 - (1) Dust
 - (2) Non-Dust
3. Past:
 - a. Specific Occupation
 - b. Specific Industry
 - c. Number Years In:
 - (1) Dust
 - (2) Non-Dust
4. Past Illness:
5. Present Illness:
 - a. Cough
 - b. Shortness of Breath
 - c. Fatigue
 - d. Others
6. Physical Examination:
7. Laboratory:
8. X-Ray
9. Remarks:

SELECTED REFERENCES

Riley and Dallavalle, Public Health Reports, vol. 54, No. 3, 3 March 1939, pp 352-359.

A Study of Quartz-Fusing operations with special Reference to the Measurement and Control of Silico Fumes.

Sayers and Jones, Public Health Reports, vol. 53, No. 33, 19 August 1928, pp 1453-1472.

Silicosis and Similar Dust Diseases.

Metropolitan Life Insurance Co., Industrial Health Section, Silicosis.

HEALTH HAZARDS

Dermatitis may be caused by carbon tetrachloride, petroleum distillate (gasoline and petroleum naphtha), benzol, toluene, acetone, amyl acetate, lead, ethyl alcohol, butyl alcohol, ethyl acetate, butyl acetate, turpentine, caustic cleaners, oils, and greases.

1. **CARBON TETRACHLORIDE.**—If in sufficient concentration may cause an acute narcosis. Most common effect is chronic poisoning which injures primarily the kidneys and liver.
2. **PETROLEUM DISTILLATES** (gasoline and petroleum naphtha).—May produce an acute anesthetic action or may produce a chronic type of poisoning usually associated with nervous symptoms.
3. **BENZENE** (benzol).—Is a very toxic material and usually presents a picture of aplastic anemia. This type of clinical picture is seen after prolonged exposure. The acute type of poisoning produces inebriation.
4. **TOLUENE.**—If in sufficient concentration has a narcotic action. May produce death. Toluene is methyl benzene.
5. **ACETONE.**—There are no cases of poisoning reported in man. Experimentally on animals, has an acute narcotic effect.
6. **ACETATE** (amyl, butyl and ethyl).—Slight possibility of causing irritation of eyes and respiratory passages and mildly anesthetic. There are no fatal poisoning cases reported in man.
7. **ALCOHOL** (butyl).—May produce blood changes with renal and hepatic degeneration if inhaled over a long period of time.
8. **TURPENTINE.**—Inhalation of heavy fumes causes symptoms of a mild anesthetic poison. It is excreted by the kidneys and nephritis is not an uncommon result of long exposure to turpentine fumes.
9. **CAUSTIC CLEANER.**—May produce dermatitis and conjunctivitis along with caustic effect.
10. **OILS AND GREASES.**—Usual damage is an oil dermatitis which can be controlled by proper corrective measures.

SECTION XVII

MOTOR VEHICLE AND MISCELLANEOUS REPAIR

PROCESS.—Repair of gasoline motors and related equipment.

TOXIC AGENTS

1. Carbon monoxide.
2. Gasoline.
3. Lead.
4. Asbestos.
5. Oils and greases.

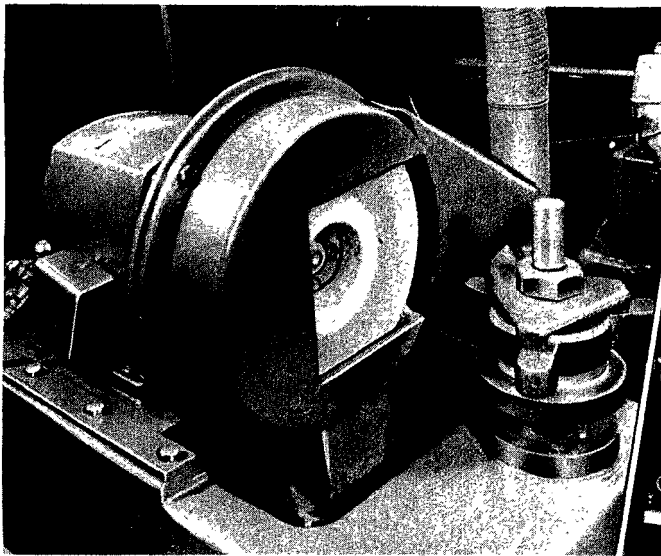
SUGGESTED CONTROL MEASURES

1. Local exhaust ventilation.
2. Protective clothing, gloves, aprons.
3. Approved-type air line respirators for continued exposure or approved-type toxic dust respirator for intermittent exposures to dusty operations.
4. Protective hand creams.
5. Strict personal hygiene.

HEALTH HAZARDS

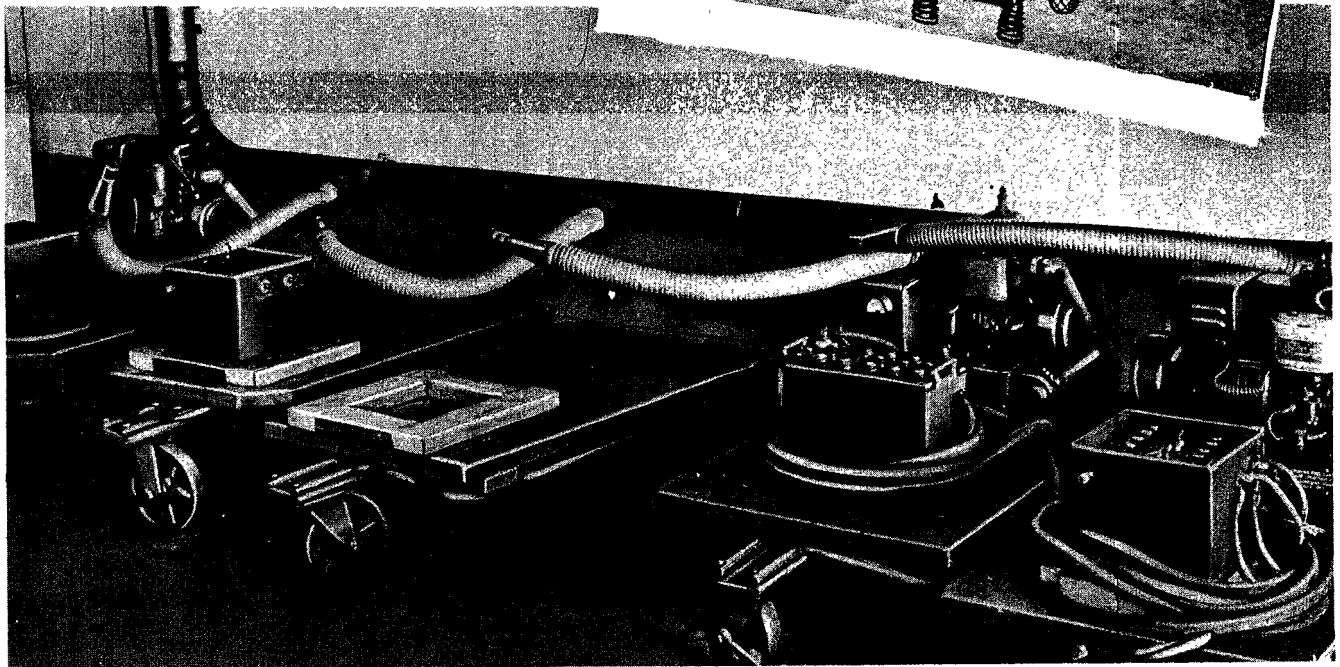
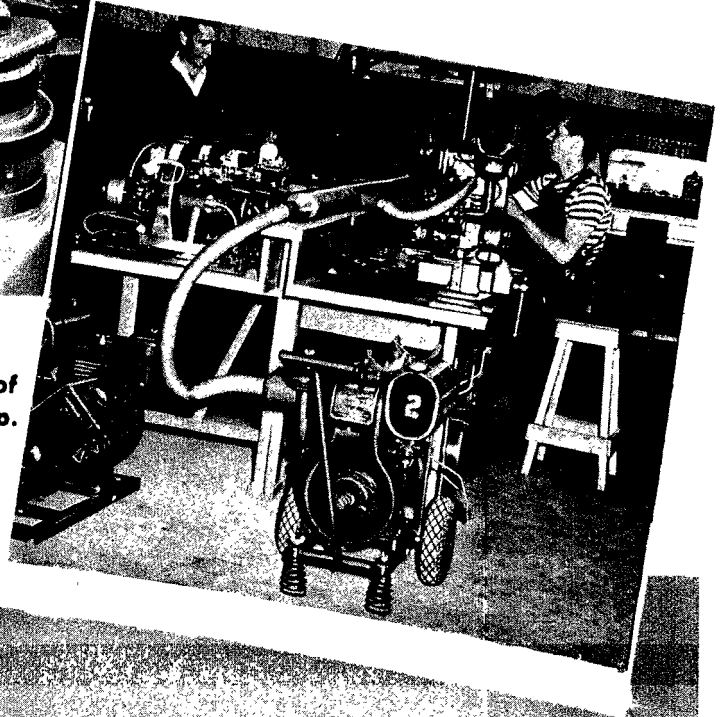
Dermatitis may be caused by petroleum distillate (gasoline and petroleum naphtha), oils, and greases.

1. **CARBON MONOXIDE.**—If in sufficient concentration, will produce anoxemia, causing unconsciousness and death.



*Exhaust ventilated grinding wheel
for brake shoe grinding.*

*Exhaust ventilating system for removal of
carbon monoxide during motor tune-up.*



Exhaust ventilating system for removal of carbon monoxide during motor tune-up.

2. **GASOLINE.**—May produce an acute anesthetic action or may produce a chronic type of poisoning usually associated with nervous symptoms.
3. **LEAD.**—May produce any of the symptoms and signs of plumbism.
4. **ASBESTOS.**—A prolonged exposure may produce asbestosis.
5. **OILS AND GREASES.**—Usual damage is an oil dermatitis which can be controlled by proper corrective measures.