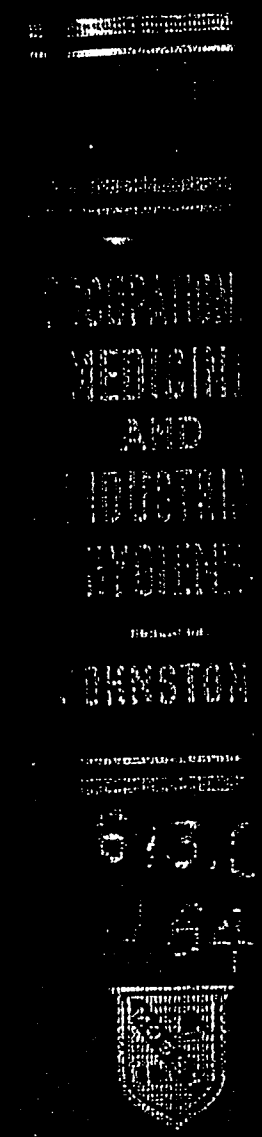
PLAINTIFF'S
EXHIBIT
01346.00
tabbies



PLAINTIFF'S
EXHIBIT
WV-009118
tabbies

RESEARCH LIBRARY
No. 2922
AUG 28 1953
EAGLE-PICHER CO.
JOPLIN, MO.

613.6
J 64

OCCUPATIONAL MEDICINE AND INDUSTRIAL HYGIENE

By

RUTHERFORD T. JOHNSTONE, A.B., M.D.

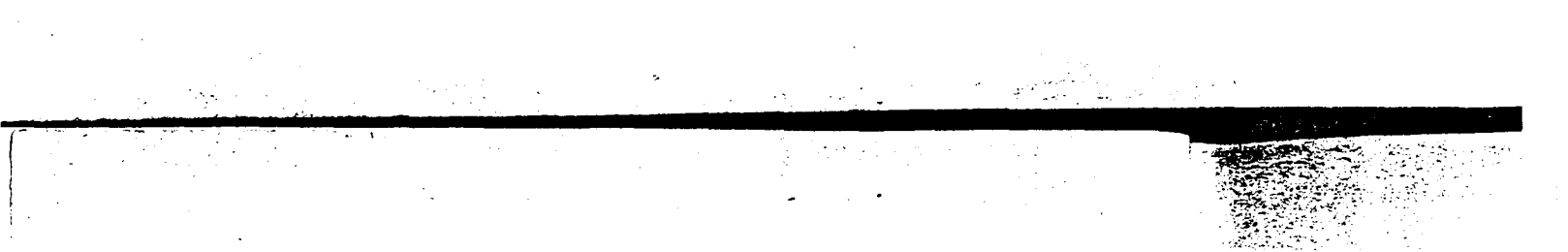
Consultant in Industrial Health; Lecturer at the University of California, Los Angeles

Formerly Assistant Professor of Medicine, University of Pittsburgh School of Medicine; Formerly Director of Department of Occupational Diseases, Golden State Hospital

With One Hundred Seventeen Illustrations

Seven in Color

St. Louis
THE C. V. MOSBY COMPANY
1948



CONTENTS

CHAPTER XXX

ASBESTOSIS

Asbestosis is a comparatively new disease. The first comprehensive reports regarding asbestosis came from the English writers in the years between 1920 and 1925. In America in the early thirties studies were made by Lanza, Sayers, Bloomfield, and others.

Asbestos is a hydrated magnesium silicate. Occupational exposure occurs in those trades where it is used for packing, insulating, or fireproofing, or where it is combined with cotton or other material in textile processes.

Signs and Symptoms.—As in silicosis,[1] the first sign is dyspnea, the cough is usually dry, and auscultation reveals few, if any, physical signs. Loss of weight is usually noticeable, the color is pale, and cyanosis is a fairly early sign. After some time, fine, crackling râles may be heard, and the chest appears emaciated and lacks the robust character noticed in silicosis. Early asbestosis must be differentiated from acute emphysema. If tuberculosis is present, the symptoms may take on the character of this disease, but this complication is far less frequent than in silicosis.

Pathology.—

PULMONARY CHANGES.—The pulmonary fibrosis of asbestosis is diffuse in character, peribronchial, and basal in location in contrast to silicosis in which the fibrosis is nodular in character and present in the upper part of the lungs. As the process continues, the fibrosis extends into almost all portions of the lung tissue. Bronchiolectasis and bronchiectasis are present within the substance of the fibroid areas, and bronchopneumonia and acute tracheobronchitis often cause the death of these patients. The pleurae, especially in the basal regions, are thickened and adherent. In some cases the pleural sac is completely obliterated; in others a fibrous or serous exudate may be present within it.

Action of Asbestos and Silica.—The difference between chrysotile asbestos and quartz silica in their mode of action has caused considerable speculation as well as experimentation. Gardner[2] particularly, at the Saranac Laboratory, carried on intensive study of these two materials. He felt that while the action of free silica is chemical in nature, the action of asbestos is mechanical. He stated:

"We have come to the conclusion that inhaled asbestos fibres are irritating not because they are silicates but because they are stiff fibres which mechanically irritate the lungs. Unlike the free silicas, these minerals will not stimulate fibroblasts in any part of the body; only those in the lungs are affected. It was inferred that these organs were

affected because the movements of respiration are so much more rapid and continuous than those of other viscera. Then it was discovered that if asbestos was ground very finely so that few of the fibres were longer that 2 microns in length the irritating property of the asbestos was practically destroyed. Inhalation experiments with such fine chrysotile asbestos were carried out over a period of several years. No fibrosis has developed in spite of the fact that an average atmospheric concentration of 125 million particles per cubic feet of air has been maintained. In contrast in a previously reported experiment one-third this concentration of long fibre asbestos dust produced well marked fibrosis after about two years."

If the effect of asbestos were chemical, one would expect that a decrease in size would accelerate tissue response. With free silica large particles have little effect, but as their size decreases cellular reaction becomes more vigorous and even constitutional symptoms may ensue. With fibrous asbestos the reverse is true.

The histology of early asbestosis does not suggest a chemical injury. Even under the most extreme conditions that can be created by artificial injection there is no preliminary phase of tissue necrosis with infiltration of leucocytes as occurs with high concentrations of very fine quartz. The connective tissue cells merely multiply very slowly in areas where the asbestos fibres are caught in the bronchioles. As collagen forms and contracts, the air spaces are obliterated by scar tissue. In experimental animals, at least, this change is not a progressive one after cessation of exposure to the dust as is the case in the response to quartz. Perhaps the reason is the deposition of the peculiar iron-containing coating on the surface of the inhaled fibres giving rise to the characteristic "asbestosis bodies" (Figs. 50 and 51).

Finally, it is most suggestive that among dozens of different silicate minerals only the five known as asbestos, which are unique because they are fibrous in structure, should be commonly recognized as pulmonary irritants. The variation in chemical composition within this group is greater than that between them and many other silicates. In fact, chrysotile asbestos has the same chemical formula as a nonfibrous silicate, serpentine, which is physiologically inert. Obviously irritation would seem to be associated with the physical rather than the chemical composition of these minerals.

One point of practical significance may be indicated by these observations: namely, that very finely ground asbestos is not dangerous. This conclusion has support in clinical observation, for it has long been known that at the Thetford Mills there was no clinical asbestosis even though in former years the atmosphere was very dusty and the dust was extremely fine. The fact that fabrication of fibres of the same mineral in American plants could produce disease was one of the puzzling fea-

Fig. 50.—Right lung, showing markedly thickened pleura with fusion of interlobar pleura. Dense fibrous tags over entire surface. (U. S. Public Health Service, Bulletin 241.)

Fig. 51.—Asbestosis bodies accompanied by giant cells. (U. S. Army Medical Museum, Specimen 52255.)

Fig. 52.—Section of a lung in asbestosis. (Gardner.)

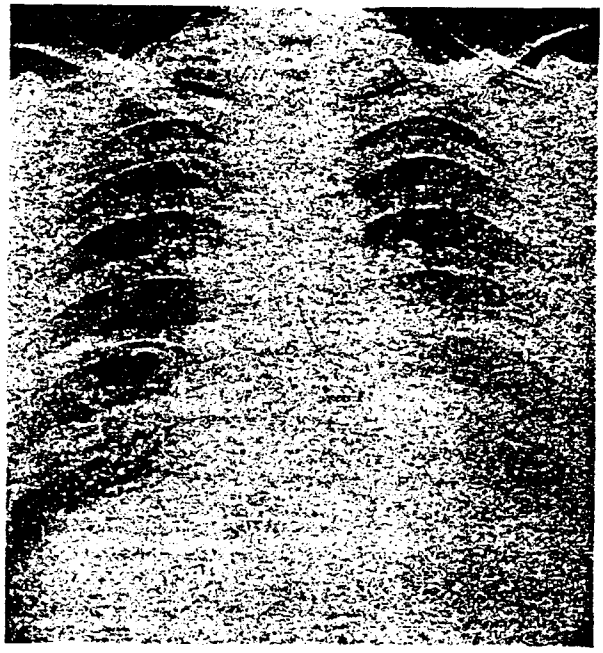


Fig. 53.—X-ray film of a patient with asbestosis. (Pendergrass.)

tures of this disease. But these experiments offer a plausible explanation. The fine dust in the mills is composed of serpentine and extremely short chrysotile fibres; that in the spinning and weaving mills contains many more long fibres.

Microscopic Appearance.—In the early phases of the disease there is a thickening of the alveolar septa as a result of fibroblastic proliferation. The alveolar spaces contain numerous alveolar phagocytes. With progression of the disease, fibrosis becomes more marked and the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few remaining alveoli which lie in the area of fibrous tissue are lined by a low cuboidal epithelium and have a glandular appearance (Fig. 52).

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures (first described by McDonald*) known as asbestosis bodies. These bodies are slender in their center and bulbous at each extremity. Many are arranged like strings of graduated beads with the largest at the end of the chain.

X-Ray Findings.—Certain peculiarities exist here as contrasted to silicosis. In asbestosis, the lesions may be bilateral or largely unilateral. According to the experience of Pendergrass,[3] the roentgenologic findings in asbestosis are largely limited to the lower half or two-thirds of the lung fields, while in silicosis the upper portions of the lungs are also involved. In moderately advanced asbestosis, x-rays will reveal lessened ventilation of the lung fields, the parietal pleura is thickened, and there is a "ground-glass" appearance to the picture. The vascular shadows lose their identity. Nodulation is absent. Pendergrass[3] feels that a roentgenologic diagnosis of early asbestosis is unreliable and that the condition must be moderately or markedly advanced to make a differential diagnosis from the roentgenograms (Fig. 53).

Medicolegal Aspects.—It is felt that the time necessary to develop asbestosis is, on the average, from seven to nine years in a fairly high concentration; with a less severe concentration, from fifteen to twenty years. The allowable concentration is 10,000,000 particles per cubic foot of air, of a size between 0.5 and 5.

Once established, the disease is progressive, even after the cessation of exposure. The basis for diagnosis is similar to that given under Silicosis.

References

1. Lanza, A. J.: Asbestosis, J.A.M.A. **106**: 368 (Feb.), 1936.
2. Gardner, L. U.: Recent Developments in Relation to Silicosis, Indust. Med. **9**: 45 (Feb.), 1940.
3. Pendergrass, E. P.: Silicosis and Asbestosis, New York, 1938, Oxford University Press.

* McDonald, S.: Histology of Pulmonary Asbestosis, Brit. M. J., **2**: 1025, 1927.

RESEARCH LIBRARY
No. 1098
Date Received Jan 5/38
EAGLE-PICHER LEAD CO.

613.63
D83

INDUSTRIAL DUST

Hygienic Significance, Measurement and Control

BY

PHILIP DRINKER, S.B., CH.E.
Professor of Industrial Hygiene, Harvard School of Public Health

AND

THEODORE HATCH, B.S., S.M.
Instructor in Industrial Sanitation, Harvard School of Public Health and Harvard Graduate School of Engineering

FIRST EDITION
SECOND IMPRESSION

McGRAW-HILL BOOK COMPANY, INC.
NEW YORK AND LONDON
1936

COPYRIGHT, 1936, BY THE
McGRAW-HILL BOOK COMPANY, INC.

PRINTED IN THE UNITED STATES OF AMERICA

All rights reserved. This book, or parts thereof, may not be reproduced in any form without permission of the publishers.

THE MAPLE PRESS COMPANY, YORK, PA.

CHAPTER II

EFFECTS OF DUSTS AND FUMES UPON MAN

Exposure to dusts can produce four distinct types of disability: (1) the pneumoconioses, such as silicosis and asbestosis, are caused only by dust inhalation; (2) toxic dusts like lead, manganese, and cadmium can produce their effect as the result of

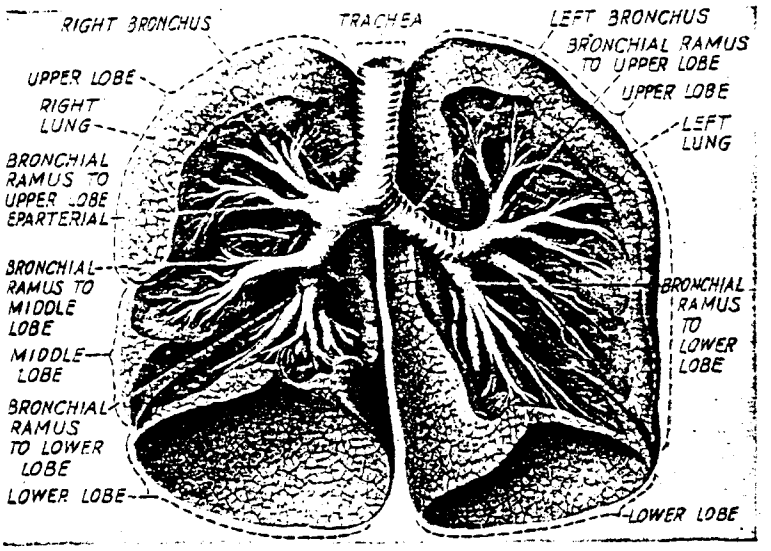


FIG. 10.—Lungs, bronchi, and trachea. (*After Sobotta and McMurrich.*)

either breathing or swallowing dust; (3) metal-fume fever is a temporary malady resulting from inhaling certain metallic oxide fumes; and (4) the allergic reaction or protein poisoning, as typified by hay fever, is the direct result of breathing pollen and other organic substances. In all four cases dust inhalation may be the sole cause of disability; only in the case of toxic dusts is there another mode of entrance.

Respiration and Dust Inhalation. The lungs are nonsymmetrical bilateral structures encased in a rather elastic cavity,

the chest, and they communicate with the nose and mouth through the trachea or windpipe. The left lung has two divisions or lobes and the right lung has three; the right lung is about 12 per cent larger than the left.

In the normal adult, the trachea is a tube some 1 to 2 cm. in diameter and 10 cm. long (Fig. 10); it is fortified with ringlike cartilage which gives it a strong and inelastic character. At the approximate level of the fourth rib, the trachea branches into the bronchi and these in turn subdivide into bronchioles which lead to the terminal air sacs or alveoli. It is in the alveoli that, as result of inspiration and expiration, gas exchange between blood and air takes place. Oxygen is taken up by the red blood cells and carbon dioxide is given off.

The amount of air breathed and the oxygen consumed vary with the task performed and with the individual's size. The rates at which an athletic man of 150 lb. breathes are shown in Table 1.

TABLE 1.—OXYGEN CONSUMPTION AND VOLUME OF BREATHING BY MAN[1]

	Oxygen consumed at 0°C. and 760 mm. pressure, liters per minute	Air breathed at 20°C., liters per minute
Resting in bed	0.24	6
Sitting	0.30	7
Standing	0.36	8
Walking 2 m.p.h.	0.65	14
Walking 4 m.p.h.	1.20	26
Slow run	2.00	43
Maximum exertion	3.00–4.00	65–100

[1] After Henderson and Haggard (125).

The ordinary individual seldom breathes more than 50 liters per minute, the high rates being limited to trained athletes, such as long-distance runners.

The inspiratory and expiratory phases are not exactly alike, but practically we may consider each as taking half the time required for a complete respiratory cycle and we can ignore the pauses that occur at the beginning and end of each inspiration. Thus with minute volume of 50 liters the actual inspiration is at the rate of 100 liters per minute and momentarily rather high

velocities occur in the trachea. The velocity in the alveoli proper, however, is practically zero at all times because each alveolus is a dead end. The slight surge back and forth with respiration must be very close to still-air conditions. Thus there cannot be any driving or high-speed impingement of dust particles into the lung tissue. Furthermore, there is no evidence of such impingement in the upper passages where velocities are highest.

The respiratory mechanism is not strong enough to withstand for long any appreciable impediment or resistance to respiration. The muscles are easily fatigued, with resulting embarrassment and distress. Practice or training teaches the individual to minimize the effects of the resistance, but no one, strong or weak, will perform even the mildest exercise for long if the respiratory impediment exceeds a few inches, water gage. This fact is of the utmost importance in the design of respiratory protective equipment, as will be shown in Chap. XIV.

The Fate of Inhaled Dusts. A dust particle the size of a common pollen grain (15 to 25 μ) is likely to be caught in the nasal passages or at the back of the throat. If it should enter the trachea near the center line, there is no reason why it should not pass on down to the bronchi, but it is not likely to reach the alveoli. Collection of such a particle is the result solely of chance impact against the moist walls of the respiratory tubes. Obviously such impact takes place most effectively with particles large enough to have appreciable momentum and rapidly ceases to be effective as the particles approach sizes at which they move as an integral part of the transporting gas. Lining the trachea and extending down to the lower ends of the bronchioles are myriads of cells with whiplike appendages, cilia, which carry upward any foreign bodies that chance to touch the wet mucus-bathed linings of the respiratory passages. The nasal passages likewise are bathed in mucus and lined with cilia. All the mucus is moving toward the exits of the nose and mouth and is never stagnant.

Within the alveoli are other cells, phagocytes, which are brought out in vast hordes by the stimulus of foreign bodies such as dust particles, which they engulf (Fig. 11). The dust-laden cells, which have the power of independent motion, may wander off through the walls of the lung tissue into the blood capillaries

surrounding the lungs, or they may pass to the finer bronchioles, from which they are removed by ciliary action; thus they eventually reach the mouth and are spit out or swallowed. Within the alveoli there are neither cilia nor mucus.

Most of the dust-laden cells, however, migrate into the lymphatics, a system of closed vessels running through the tissue that supports the blood vessels of the lungs, the bronchioles, and the bronchi (Fig. 12). The lymph, a watery fluid which flows through the lymphatics, has its own circulation. It is derived from the blood, into which it discharges near the heart. At the various bifurcations of the trachea and the bronchi, the lymph passes through glands or lymph nodes, one of whose functions is the filtration of foreign bodies. It is at these tracheo-bronchial lymph nodes that a great deal of dust is deposited by the phagocytic cells and it is here that fibrosis of healthy lung tissue starts, following quartz-dust inhalation.

FIG. 11.—Phagocytic cells containing dust. (*Courtesy J. Ind. Hyg.*)

Dust particles can pass from the blood vessels into the lymph circulation without having been phagocytosed (60). Such particles then can be picked up by phagocytes at any point in the lymph stream or in a lymph node, the latter being particularly suited to such phagocytosis.

Fenn (78) found that all dusts are not phagocytosed with the same readiness; he gave cells an equal chance to ingest various dusts and found that quartz was among those least preferred.

Gardner and Cummings (85) have pointed out that the motility or rapidity of migration of cells differs with different dusts.

PNEUMOCONIOSIS

It has been recognized for centuries that excessive dust inhalation generally produces a serious pulmonary disease which has been known in the various dusty industries as miners' asthma, miners' phthisis, grinders' rot, etc. Zenker (260) proposed the

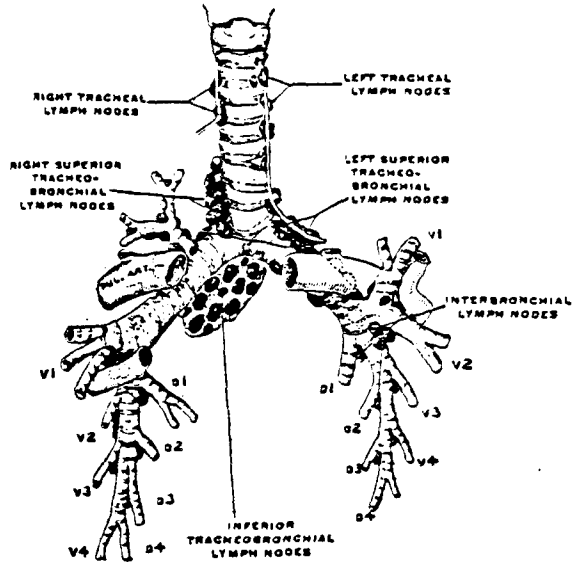


FIG. 12.—Position of lymph nodes in relation to the trachea, the bronchi, and the pulmonary artery. (*After Gray's Anatomy.*)

general name *pneumonokoniosis* (a *lung* containing *dust*) in place of the special names applied to certain industries. Recently this has been shortened to *pneumoconiosis* (134). The word originally implied that the lung had been seriously damaged by dust—enough to cause disability—but the meaning has been broadened in recent years to include all pulmonary manifestation of dust inhalation, whether the dust is injurious or harmless.

Silicosis and asbestosis are today the most important forms of pneumoconiosis. Other terms such as *silicatosis*, *anthracosis*, *siderosis*, etc., have been given to milder forms of the disease and

doubtless others will be added as studies of the dust problem progress.

Silicosis. For reasons still unexplained silicon dioxide as quartz (free silica) produces the worst form of lung fibrosis. The American definition of silicosis (3) states that it is 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. . . . " The International Silicosis Conference at Johannesburg in 1930 defined silicosis (133) as a "pathological condition of the lungs due to the inhalation of free silica dust. It can be produced experimentally in animals. It can be detected by clinical and radiological means, which can be confirmed with the above pathological condition with sufficient accuracy to separate it from other pneumonoconioses. It also affords a fair basis for legislative measures."

A recent note in the *Journal of the American Medical Association* (140) states that:

> Only silica (SiO_2) is capable of inducing silicosis but any other mineral dust under conditions of prolonged exposure and gross exposure may cause some increase in pulmonary fibrosis. If the relative potential harm of silica is rated as 100, these other nontoxic mineral dusts may be rated only on the order of 5 or 10. Moreover, the fibrosis is in itself not pathognomonic, since many other dusts, alkalies, acids, or vapors may induce somewhat similar if not identical X-ray markings.

In his summary of the situation in South Africa, Watkins-Pitchford (247) wrote in 1927 that dusts other than silica could "give rise to such nonpermanent and relatively harmless conditions—one can hardly call them diseases—as anthracosis, aluminosis, siderosis, etc."

In general, the damage done in silicosis (and asbestosis) is permanent; an unalterable tissue change takes place in the lungs. Of prime importance is the fact that prolonged exposure to these dusts results in a greatly increased susceptibility to tuberculosis, more so from quartz than from asbestos. Gardner has shown in the laboratory the great importance of this fact which had long been recognized in industry. Deaths from uncomplicated lung fibrosis caused by dust are infrequent.

Free quartz possesses a unique cytocidal power; it kills phagocytic cells, the physiological scavengers, which then deposit their dust load *in situ*. The dust thus dropped may or may not be picked up again and the killed cell is said to mummify. The surrounding tissue then begins to fibrose and to appear more or

FIG. 13.—Silicosis. Isolated silicotic nodules above; fine nodules below. (*After Gardner; courtesy U.S. Public Health Service.*)

less stringy, as is shown in Fig. 13; such a fibrotic nodule contains dust particles identifiable chemically and petrographically.

Characteristics of Silicosis. For the convenient diagnosis of silicosis or of silicosis complicated by tuberculosis, the pulmonary condition is usually placed in one of three stages. In the first stage, the disease does no measurable harm (and produces no disability). The victim can work just as well as ever. But as his condition progresses to the second stage, his respiration is affected; he is bothered by dyspnea or labored breathing. If his dust exposure continues (and often even if he is kept out of dusty air), he is likely to reach a third stage in which dyspnea becomes severe and he is likely to contract pulmonary tuberculosis, generally with fatal results. Whether he contracts tuberculosis or not,

the advanced silicotic is far below normal and is inordinately susceptible to all respiratory diseases (44).

Each of these three stages of silicosis is diagnosable by X-ray, provided a reliable case history, including dust exposure, is available. A comparison of the X-ray plates of a silicotic with those of a normal person of like age and physique shows, in the first stage, distinctive shadows, evenly distributed in both lungs. These may be missed or misinterpreted by any but an expert; but even to the layman the changes in the second and third stages are obvious.

FIG. 13*a*.—Barre granite cutter. Two isolated silicotic nodules. Cellular connective-tissue borders and hyaline fibrous centers. Thickened interlobular septum extending upward to the left of the right nodule. Note dilated lymph vessels in septum. (*After Gardner; courtesy U.S. Public Health Service.*)

When silicósis first came into prominence in England, then in South Africa, and later in the United States, the cases described and the X-rays published featured advanced cases, often complicated with tuberculosis. A comparison of the X-ray pictures with those of normal chests showed striking differences that could not be missed by anyone. Granted that the layman might misinterpret what he saw in the X-ray, he could not miss the differences between the dusted and the normal chests. Today those cases would probably be called "third stage" in the American nomenclature.

The advanced third-stage silicotics described by Lanza (127), by Pancoast and Pendergrass (196), and by earlier writers in South Africa and in England seem to be disappearing in the

United States, and in their stead are being featured in the literature the cases more difficult to diagnose, which so confuse the layman. There is no doubt at all that the rapidly fatal third-stage silicosis has virtually been eliminated in the South African gold-mining district (135).

Silicosis and Duration of Exposure. Silicosis may not become disabling until some years after dust exposure has ceased. Watkins-Pitchford (247) gives examples of Welsh miners who passed the physical examination for enlistment in the British army, fought through the World War, then came back to England, and died of silicosis. Britton and Head (29) give more detailed examples of similar latent effects in the United States.

The length of exposure necessary to produce silicosis has been the subject of a good deal of controversy in the past. In Great Britain, Bridge (28) gives the following table:

TABLE 2.—SILICOSIS AND ASBESTOSIS IN GREAT BRITAIN, THROUGH 1934[1]

	Number of deaths	Average age at death	Duration of employment, years		
			Maximum	Minimum	Average
Silicosis	261	55.4	60	2.3	34.8
Silicosis with tuberculosis	315	52.5	67.0	2.0	32.0
Asbestosis	41	41.0	27.0	1.5	12.9
Asbestosis with tuberculosis	26	38.0	29.0	0.8	9.9

[1] After Bridge.

Harrington (106) of the U.S. Bureau of Mines has assembled the data on length of exposure required to give definite silicosis; these show very clearly that first-stage silicosis may develop in as short a time as eight months. Among foundry workers where the silicosis risk is low, McConnell and Fehnel (178) and Pope (200) found first-stage silicosis only after long employment. But in severe quartz-dust exposure, where cases may occur in a matter of a few months, the condition is very likely to progress and to become complicated by tuberculosis whether the man leaves his dusty occupation or not. Thus the length of exposure that will produce the disease varies with the working conditions and individual susceptibility.

Silicosis and Tuberculosis. It is well-known that the tuberculosis mortality in the dusty trades is high. Collis and Yule (44)

TABLE 3.—RESPIRATORY DISEASES IN PERSONS EXPOSED TO SILICA DUST

Age	Ratios of mortality to that of standard population as 1000	
	Respiratory diseases	All other diseases
20	1202	704
25	1556	1055
35	2720	1160
45	3628	1474
55	4264	1550
20–65	3067	1396

showed that the mortality from causes other than respiratory diseases is also significantly higher for persons exposed to silica dust than for those without such exposure. But their figures for mortality from respiratory diseases (Table 3) are the most striking.

The experience of the Metropolitan Life Insurance Company (159) supports Collis' and Yule's data (Fig. 14). Starting at about the same level, the statistics for industrial males show a rapid increase in the mortality rate with age, reaching a maximum at 45 to 54 years. At this point the death rate is more than three times higher than among the ordinary policy holders with the company and one and one-half times the rate for males in the United States. It is true that dust is not the only industrial factor that contributes to tuberculosis; there are other environ-

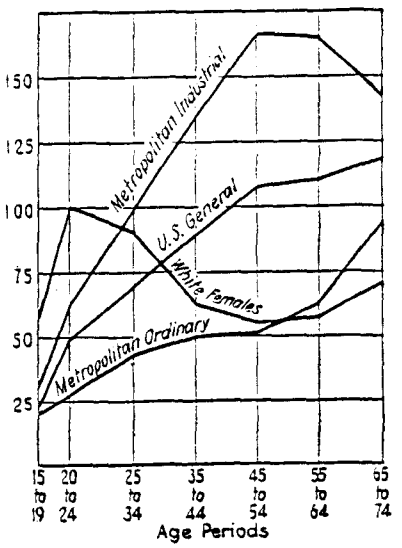


FIG. 14.—Tuberculosis—all forms—1931. Death rates per 100,000 white males by age periods. (*After Lanza and Vane; courtesy Am. Rev. Tuberculosis.*)

mental conditions of great importance which act to raise the death rate for industrial workers. Compared with female industrial workers, however, the males, who are more generally employed in the dusty trades, show a much higher tuberculosis death rate in the later years of life.

The importance of the dust hazard, in its effect upon both the tuberculosis death rate and the death rate for all causes, is shown in Table 4, in which are compared the number of actual cases and the number of expected cases (based upon general experience) among workers in the chief dusty trades. These data, which represent the combined experience of twelve life insurance companies, show excess mortality varying from 114 to 450 per cent of the expected rate for all causes and from 103 to 1833 per cent for tuberculosis. In general, the mining, quarrying, and stone-dressing operators show a higher hazard than the general manufacturing workers.

Disability from Dust Inhalation. There is no medical or social reason for branding a man with first-stage simple silicosis as disabled. Both industry and the man are done serious harm; the man is prevented from seeking gainful occupation, for no employer wants to hire a man whom the courts or the compensation boards have labeled as unfit or partially unfit. The employer, by hiring such a man, opens the door at once to suits involving the slightest mishap to the disabled.

In the British Empire, where silicosis legislation has been in force for some years, only the man with advanced silicosis, simple or infective, is disabled. Men with early, simple silicosis are not disabled, nor are they a menace to their fellow employees. The mere fact that a barely perceptible X-ray change is visible to the expert roentgenologist is not ground for claiming disability since most people over forty show some chest changes.

In the opinion of the special Industrial Disease Commission of Massachusetts (173), it is only when tuberculosis has become superimposed on silicosis that the victim becomes disabled or a menace to others. Thus a man with early, simple silicosis is not disabled and should not be prevented from working, an opinion seconded vigorously in the Saranac Silicosis Symposiums (211). In Canada the worker with simple silicosis is not refused further employment. The industry is enabled, by sensible legislation, to continue employing the man, and it is recognized

TABLE 4.—NUMBER OF DEATHS EXPECTED FROM SPECIFIED CAUSES AND NUMBER WHICH ACTUALLY OCCURRED AMONG PERSONS ENGAGED IN CERTAIN OCCUPATIONS EXPOSED TO SILICA DUST[1]

Occupation	Deaths from all causes			Deaths from respiratory tuberculosis		
	Actual	Expected	Percentage actual of expected	Actual	Expected	Percentage actual of expected
Metal-mine operatives (underground):						
Gold and silver mines	44	9.77	450	9	1.12	804
Lead and zinc mines	22	5.03	437	11	0.60	1833
Copper mines	83	22.63	367	24	2.63	913
Iron mines	33	12.12	272	4	1.54	260
Quarry operatives	37	22.45	165	7	2.66	263
Stone cutters:						
Granite and sandstone	29	17.47	166	16	1.64	976
Marble and limestone	12	10.55	114	3	1.02	294
Metal industries:						
Grinders	46	28.65	161	7	3.33	210
Buffers and polishers	89	64.10	139	10	6.80	147
Chippers (not ship)	38	14.14	269	8	1.30	615
Core makers and sand-molders	32	22.65	141	3	2.92	103
Iron and steel foundries:						
Molders, founders, and casters	202	130.14	155	24	13.38	179
Drillers, flask makers, machine hands, mixers, etc	45	31.03	145	7	3.03	231

[1] Ordinary department mortality experience of twelve life insurance companies, 1915-1920 (159).

fully that, if he has silicosis, he got it in Canada and Canada will take care of him.

In the case of a first-stage silicotic without tubercular infection, there is no method, as yet, for determining his lessened efficiency. The lung change, if any, caused by his dust inhalation is too small to measure.

McCann and Hurtado (177) have estimated disability from lung fibroses of various origins but have not yet succeeded in developing a method that is simple enough for routine use by compensation boards, insurance examiners, and the like. Their procedures involve various simple measurements of respiratory function but they have not established any methods of detecting fibrosis which are more sensitive than the present X-ray examination.

Of course, industry would welcome a diagnostic routine for detecting early harm from dust or, better, for forecasting harm. No such method is available and it seems fruitless to expect such help. The pneumoconioses are not diagnosed until harm is demonstrable by X-ray.

Collis (42) has stressed the difference between the age groupings in pulmonary tuberculosis and in silicosis, with or without tubercular infection superimposed. Tuberculosis is most prevalent between the ages sixteen to twenty-four, while silicosis rarely becomes disabling until later in life. Inasmuch as a good many years of dust exposure are required before the average case of silicosis (or asbestosis) becomes evident, it is obvious that it is not likely to be acquired early in life. However, there is an epidemiological side to the question which is of great importance and it is that which Collis especially emphasized. The average healthy individual has acquired a fairly effective immunity or resistance to tuberculosis. His chest X-ray is likely to show one or more healed scars or fibrosed areas from tuberculosis. Such an individual, Cummings emphasizes (211), is a better silicosis risk than the man who shows no evidence of past exposure to pulmonary tuberculosis. Consequently it is wise to select for dusty jobs men who are past the age of forty and not young men just taking up a trade.

Asbestosis. The X-ray picture of the typical asbestotic chest is confusing to the layman. The effect is described as a diffuse fibrosis. Characteristic silicotic nodules are absent and even

the expert roentgenologist withholds his diagnosis until the case history is complete.

The pathology produced by asbestos is not like that of silicosis. The asbestos fibers group about the neck of an alveolus and shut it off, causing what is known as *atelectasis*. There is no definite migration or transportation of the dust particles to the lymph nodes and no formation of the fibrous nodules as shown in Fig. 13. As the atelectatic areas increase, the reduction in lung

FIG. 15.—Asbestosis bodies in sputum. (*After Ellman* (75); *courtesy J. Ind. Hyg.*)

area causes serious dyspnea or labored breathing. Lanza (158) suggests that the enlarged hearts noted frequently in his cases of second-stage asbestosis may be the result of the increased work of the heart resulting from this condition; it takes more work to pump blood through the atelectatic than through the normal lung.

In silicosis it seems to be a general rule that, after a certain point, the victim's condition grows worse even if his dust exposure has ceased. But Wood and Gloyne (258) state that they have seen patients with asbestosis "whose condition appears to have

remained stationary since stopping work in the factory," but they advise definitely that the asbestotic individual be removed from his dusty job. Merewether (183) and Lanza are less certain on this point.

Asbestosis Bodies. In the lungs of patients who died after prolonged exposure to asbestos dust and in the sputum of men with considerable asbestos-dust exposure are found what first were called *curious bodies* and later *asbestosis bodies* (Fig. 15) (75). While somewhat similar bodies occur in the lungs of coal workers and even of normal persons, it is admitted that asbestosis bodies in sputum are characteristic of asbestosis. Stewart (223) gives considerable diagnostic weight to their presence.

Pneumoconiosis in Animals. Silicosis has been produced experimentally by quartz dusting guinea pigs, rabbits, mice, cats, and domestic fowl, thus emphasizing the specificity of quartz dust to biological tissue. In like manner asbestosis has been produced in experimental animals.

The Equidae, such as horses and mules, apparently are not susceptible to ordinary pulmonary tuberculosis but there is no reason to believe they have any special immunity or resistance to silicosis. The normal silica content of horses' or mules' lungs is not known but it would seem wholly reasonable to use the lungs of animals which have worked 5 to 15 years underground in mines as physiological dust samples. A brief report on this subject was published by Haynes (121) but data such as one needs are singularly lacking.

TOXIC DUSTS

Poisoning from inhaling toxic dusts is much more likely than poisoning from swallowing them. Dusts that reach the lungs may pass directly into the blood stream, thence to the heart, and immediately be pumped all over the body. Distribution to all the body tissue is thus brought about rapidly and effectively. But dust taken in with the food goes to the stomach and the major part passes out in the feces. Some is picked up by the portal blood circulation and moves on to the liver. That portion which causes poisoning must first pass through the liver, which is an effective filter and detoxifier; only then can it enter the general circulation.

The practical significance of this physiological distinction between the two ports of entry of dust is considerable. Hamilton (103) states,

> A great deal of money has been wasted by well-meaning employers who sought to protect lead furnacemen or oxide roasters or white lead grinders against poisoning, by providing baths and lunchrooms and clean overalls and mouth washes and such, instead of preventing the escape of lead into the air the men were obliged to breathe, and unfortunately this has sometimes been done under a physician's advice. It must never be forgotten that the great majority of industrial poisons enter the body with the inspired air and that while a workman eats only three times a day he breathes sixteen times a minute during the eight or ten hours of his working day.

Goadby (88) found that cats dusted with lead acquired lead poisoning more easily than did a control animal which was fed over ten times the total lead dosage for the dusted animals. Drinker and Shaw (61) showed how efficiently the liver removed foreign dust particles injected into the blood stream. Minot (188) emphasized the much greater danger of lead poisoning from inhaled than from swallowed dusts. Blumgart (24) showed that the absorption of lead directly in the nasal passages was rapid and might be "of a magnitude far in excess of the minimal dose by mouth."

All this experimental evidence confirms the view so often expressed by the late Sir Thomas Legge (162), but which is only now beginning to be accepted, namely, that there is far more danger of being poisoned by inhaling toxic dusts than by ingesting them in food or drink.

Metal-fume Fever. Of interest in connection with the breathing of dusts is metal-fume fever, a transient noncumulative malady that results from breathing rather heavy concentrations of metal fumes like zinc oxide, copper oxide, magnesium oxide, lead, probably lead oxide (227), and manganese dioxide.

About 2 to 8 hr. after a heavy exposure to the well-dispersed metallic or metallic-oxide fumes, the victim experiences chills followed by fever like that of malaria or the protein reaction following a typhoid inoculation. The patient's fever may reach uncomfortable heights (we have recorded 104°F.) and the next day he feels debilitated but generally can go to work. With

the fever goes an increased white-blood-cell count or leucocytosis like that experienced in any infection. By the next morning the fever has abated but the leucocytosis persists (Fig. 16). Then the victim is fairly immune; generally he can take another inhalation without experiencing a second attack (70). In industry it is a commonplace that attacks on successive days are unlikely.

The cause of this peculiar malady is probably the absorption of protein material which results from the action of the inhaled

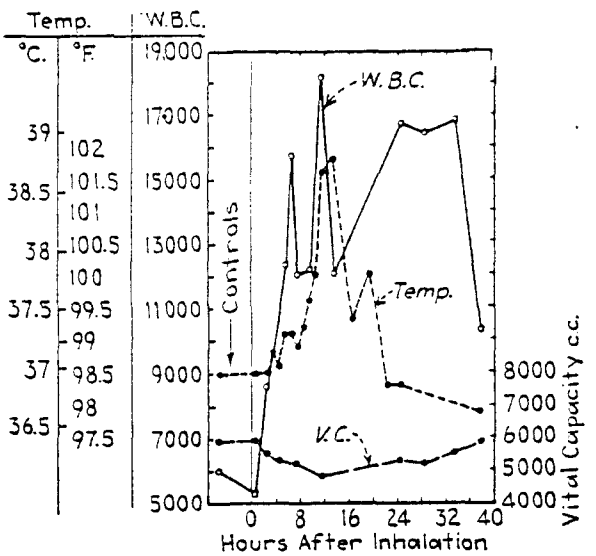


FIG. 16.—A typical attack of metal-fume fever, showing increase in leucocyte count, body temperature, and drop in vital capacity. Note that fever abates before the white count returns to normal. (*After Sturgis et al.; courtesy J. Ind. Hyg.*)

fume particles upon the tissue of the respiratory passages. In our own experiments we found that the chills and fever were acquired far more easily if one took a few deep breaths at a rate of say five breaths per minute than at the normal rate of twelve to fifteen breaths per minute. Slow, deep breathing insures penetration into the alveolar spaces.

Electric or acetylene welding in a confined space may generate metal-fume concentrations in excess of those used by Lehmann or Drinker in studying metal-fume fever. At present there are no data on the effects of breathing dense concentrations for

prolonged periods. There is reason to believe that the effects of heavy concentrations would be more severe than the ordinary metal-fume fever reaction (233).

ALLERGIC REACTIONS

An undetermined number of organic dusts produce allergic reactions like hay fever or specific kinds of skin reaction—dermatoses. Sensitivity varies greatly and unpredictably with the individual. The manifestation may take the form of a bothersome dermatitis or again may be a true poison in the commonly accepted sense. It is unnecessary for the offending dust to reach the depth of the lungs—giant-ragweed pollens, for example, which are unlikely to reach the alveoli, undoubtedly can produce their effect after being caught in the upper respiratory passages. Should the offending substance be finely ground (*e.g.*, broken-up pollens), it would reach the alveoli and as a result probably all physiologic reactions would be accelerated.

Landis (157), whose contributions to the field of pulmonary tuberculosis and pneumoconiosis are notable, stated of organic dust that it "never produces the condition known as pneumonokoniosis. That some organic dusts give rise to protein intoxication, such as asthma, threshers' fever, etc., is known."

THE DUSTY TRADES

Owing to the lack of occupational and mortality statistics on silicosis and other dust diseases in this country, it is impossible to state accurately the number of men exposed to injurious dusts. Hoffman (129) estimated in 1918 that there were approximately 4 million workers engaged in dusty occupations but of these only 1.7 million were exposed to metallic and mineral dusts. Recently Van Siclen (244) prepared the following estimate of the number of men in various industries exposed to dust capable of producing silicosis (Table 5).

Lanza and Vane (159) carried the study further and have estimated the number within each industry actually exposed to silica-bearing dust, whereas Van Siclen simply gave the total number employed in each industry. The data in Table 6 are compiled from the figures given by Lanza and Vane, who point out that in addition to these there are "thousands of workers

TABLE 5.—NUMBER OF MEN ENGAGED IN CERTAIN DUSTY TRADES INVOLVING (MORE OR LESS) EXPOSURE TO SILICA DUST[1]

Process	Total Exposed
Metal mining	62,268
Nonmetallic mining, omitting tunnel and foundation excavation	23,665
Bituminous coal mining, underground	450,513
Bituminous coal mining, open pit	8,219
Anthracite mining, underground	143,063
Anthracite mining, independent washeries	269
Smelting, nonferrous plants	13,166
Smelting, ferrous plants	24,960
Cement plants	33,368
Asbestos products	8,092
Abrasive industry	3,873
Clay products	93,336
Cutlery	14,991
Glass manufacture	67,527
Granite, slate, marble, and other stone products	28,715
Hones, whetstones, and similar products	174
Iron and steel	39,697
Mineral fertilizers	20,926
Minerals and earths, ground	1,679
Nonferrous metal alloys and products	79,183
Pottery, including porcelain ware	33,409
Sand-lime brick	566

[1] Adapted from Van Sicien.

TABLE 6.—ESTIMATED NUMBER OF MEN EXPOSED TO SILICA-BEARING DUST IN VARIOUS INDUSTRIES[1]

Process	Percentage of all employees	Number exposed
Metal mining	100	62,000
Anthracite coal mining	20	30,000
Quarrying of granite, ganister, sandstone, etc.	100	22,000
Smelting and refining	100	18,000
Foundry workers	100	200,000
Potteries, glassworks, stone products	50	70,000
Grinders, buffers, sand blasters, vitreous enamelers	?	62,000
Estimated total for chief mining, quarrying, and manufacturing industries	...	450,000

[1] Adapted from Lanza and Vane.

who have been exposed to silica dust in these various occupations and are now engaged in other forms of work."

Silicosis occurs in other industries as well as those listed above. Among these may be mentioned bituminous coal mining, rock excavation in connection with building and highway construction, and various public-utility jobs. The total number of men engaged in these miscellaneous industries is put by Lanza and Vane at 2,000,000, of which at least 5 per cent are said to be exposed to silica dust. Hence, one must increase the estimate in Table 6 by 100,000. Thus, the number of men exposed in United States industries to harmful amounts of silica dust is approximately 500,000.

We have no estimates of the number of workers exposed to asbestos dust in this country. In England, with an industrial population about one-third that of the United States, there are approximately 2200 asbestos workers (184).[1]

Dust Concentrations in the Dusty Trades. Clean country air contains as little as 0.1 to 0.2 mg. of dust per cubic meter, a large portion of which is organic matter. The concentration varies with meteorological conditions and with human activity. In manufacturing towns it may reach a level of 2.0 mg. or more. The air within a modern air-conditioned building should not exceed 0.2 to 0.3 mg. per cubic meter regardless of the concentration outside. In industrial establishments the dust concentration varies enormously; it will be less than 1 mg. per cubic meter in a well-controlled plant and over 500 mg. at the working face of a mine in which no dust-control measures are employed. An explosive concentration of a dust like wheat flour is about 10 to 15 grams per cubic meter. A dustiness of this concentration is very unstable because the suspension settles rapidly; an explosion usually follows a disturbance that jars dust off rafters and other dust-catching surfaces so that momentarily an explosive concentration exists.

Because of the great variation in dustiness in industry, it is impossible to summarize completely concentration values for

[1] Since writing the above we have seen a statement by Lanza from a paper presented on June 13th, 1935 at the Atlantic City Meeting of the American Medical Association to the effect that about 10,000 persons were exposed to asbestos dust. McPheeters (in *J. Ind. Hyg. and Tox.*, April, 1936) states that 9237 persons were exposed in 1929.

the dusty trades. Figures are available from many countries but these are not directly comparable with one another because the dust-sampling instruments and methods of determination are not alike. In tabulating published data, therefore, we have been limited to figures for American industries which were determined by the impinger technique, using low-power light-field illumina-

TABLE 7.—AVERAGE DUST COUNT IN CERTAIN DUSTY TRADES

Industry	Dust Count in Millions of Particles per Cubic Foot of Air
Slate-finishing mills:	
Floormen	1598.0
Loaders	1276.0
Disk crusher operators	312.8
Talc mining:	
Jackhamer drillers	2159.8
Muckers	44.8
Talc-finishing mills:	
Crushers and cylinder men	14.0
Packers	50.1
Marble carvers	19.1
Marble cutters	32.8
Granite quarrying:	
Leyner drillers	144.4
Jackhamer drillers	112.1
Plug drillers	36.9
Cement mill, average of all operations	26.0
Granite cutting:	
Hand-pneumatic-tool operatives	59.2
Machine-pneumatic-tool operatives	35.9
Attendant labor	17.0
Anthracite coal mining:	
Mining and miners' helpers	231.5
Attendant labor	31.1
Bituminous coal mining:	
Coal cutters and coal loaders	112.3
Attendant labor	3.9
Silverware manufacturing:	
Dusty processes	5.2
Nondusty processes	1.7
Municipal dust (street cleaners):	
Congested district	4.1
Residential district	1.8
Cotton industry:	
Carding room	8.6
Weaving and spinning room	4.5

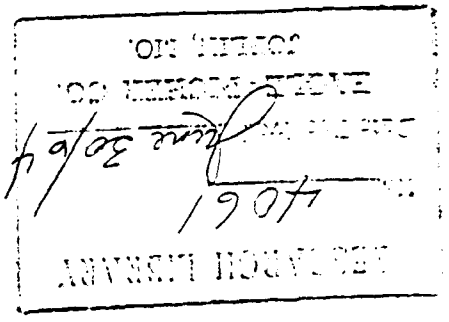
TABLE 8.—DUST CONCENTRATION IN CERTAIN INDUSTRIES; GOOD AND POOR PLANTS COMPARED

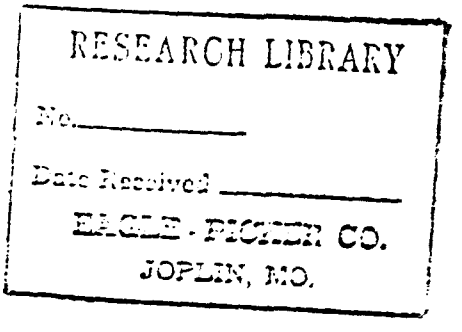
Industry and process	Control measures	Quartz, percentage	Dust concentration Millions per cubic foot		
			Maximum	Minimum	Average
Sand pulverizing:		80–98			
General plant air	Fair ventilation		20.0	2.2	12.0
Grinding	Exhaust ventilation (fair)				21.0
Bagging	None				695.0
Bagging	Exhaust ventilation (fair)				63.0
Bagging	Exhaust ventilation (good)				7.0
Abrasive-blast cleaning:		42–95			
Sand:					
Inside room	Average ventilation		3140	232	969.0
Outside room	Average ventilation		28	23	25.0
Barrel	Poor maintenance		353	1.6	33.1
Barrel	Fair maintenance		12	0.6	4.1
Table	Poor maintenance		82	41	55.0
Table	Fair maintenance		102	0.2	12.0
Cabinet	Poor maintenance		365	6	127.0
Cabinet	Fair maintenance		44	0.7	5.2
Artificial abrasive or steel:		2.5–3.6 (from sand on castings)			
Inside room	Average ventilation		3272	48	155.0
Outside room	Average ventilation		15	8	11.0
Barrel	Average maintenance				15.0
Table	Average maintenance				11.0
Cabinet	Average maintenance				8.6
Rock drilling and handling:		0–84			
Open excavation-dry drilling	None		487	0.06	58.0
	Wet drilling				
	Good exhaust hood				
Underground:					
Granite	Wet drilling				47.0
Dolomite	Wet drilling		14	5.4	10.0
Dry drilling	Good exhaust hood		4.6	1.7	2.9
Mucking	Dry, good ventilation				83.0
	Dry, good exhaust hood				8.0
Metal grinding:		30–95			
Sandstone wheels	Wet grinding		250	4.3	79.0
	Poor ventilation		62	6.3	27.0
	Good exhaust hood		2	0.2	0.7
Foundries:					
Grey iron:					
General air			76	3	17–9[1]
Core making			48	2	11–6
Molding			475	3	39–9
Shakeout			66	3	22–17
Grinders			184	5	63–6
Sand conditioning			263	22	74
Facing mills			2608	32	193
Steel:					
General air			34	1	9
Core making			15	1	8
Molding			160	3	22
Shakeout			33	2	20
Grinders			36	1	17
Facing mills			275	4	119
Malleable iron and nonferrous:					
General air			67	4	17–3
Core making			19	3	9–4
Molding			45	3	22–4
Shakeout			97	13	51–5
Grinders			13	6	10–5
Sand conditioning					56

[1] Figures in this column are for plants with good dust-control equipment.

tion (page 117). The data in Table 7 (18) summarize the results obtained in the dust studies conducted by the U.S. Public Health Service.

In Table 8 we have brought together other data from published and private reports. An attempt has been made to indicate the range in dustiness as well as the average figure and to show the difference between the conditions in good and poor plants. These figures are characterized by the wide range in dustiness exhibited by a single process, by the variations between good and poor plants, and by the variations from one industry to another.




RESEARCH LIBRARY
No.
Date Received
EAGLE-PICHER CO.
JOPLIN, MO.

INTERNATIONAL LABOUR OFFICE

OCCUPATION

AND

HEALTH

ENCYCLOPAEDIA OF HYGIENE, PATHOLOGY
AND SOCIAL WELFARE

Volume I

A – H

GENEVA
1930

IMPRIMERIE
NOIRCLERC & FÉNÉTRIER (S. A.)
3, Rue Stella, 3
LYON
Directeur : C. SERRIÈRE

Contents of Volume I

the mayor, which must be renewed every week. The Californian law enjoins that no child under at least 16 years of age can be employed in a studio unless duly authorised and after medical examination carried out by a medical man in the Child Welfare Service. These examinations are made every three months. Children taking part in film production must not be employed more then eight hours a day, including lessons lasting four hours given by a qualified teacher, paid, but not selected, by the studios. Employment of children before 8 a.m. and after 5 p.m. is prohibited.

Bibliography

PERI. "La patologia professionale degli artisti drammatici." *Il Ramazzini*, 1913. Florence.

For other sources, see *Bibliography of Industrial Hygiene*, published quarterly by the International Labour Office.

***

Asbestos

French: *Amiante, Asbeste, Pierre à coton.* — German: *Asbest, Amianth, Bergflachs.* — Italian and Spanish: *Amianto.*

Chemical Properties

Asbestos is a brilliant filamentous mineral, oily to the touch, white, silvery grey, greenish or bluish. Its density is 2.3-2.9. Its chemical composition consists, according to its origin, of calcium silicate and magnesia or of a magnesia silicate accompanied by quantities of iron, more or less extensive. Analysis of different varieties of asbestos reveals a silica content of 40-50 per cent. — 41.2 in Canadian asbestos, 41.8 in Siberian asbestos, and 51.1 in the African variety — while oxide of magnesia varies between 2.3 (African) and 41.7 (Canadian), oxide of iron between 2.52 (Canadian) and 35.8 (African). In Siberian asbestos there is besides 16.39 per cent. of alumina. When cut up it presents a silky cotton-like form which lends itself easily to weaving. It is incombustible, prevents heat loss, is a thermic and electric insulator and is not attacked by most acids.

Of the three different mineral varieties known by the name of asbestos, it is serpentine asbestos or chrysolite and hornblende asbestos which are the most commonly used; the first of these is the most sought after variety. Industrial asbestos is chiefly derived from Canada, Russia, Italy, and the Transvaal.

Technology

Extraction involves two distinct operations: (1) quarrying of the asbestos-bearing rock, generally mined in open quarries or, in countries having a severe winter climate, in galleries; (2) separation of the asbestos from the surrounding rock. The rock is cut in terraces, sometimes reaching a depth of some 150-200 ft. Quarrying is more economical and effective than underground work even despite exposure to weather. Drilling and blasting are engaged in as in ordinary stone quarrying. The mineral got from the quarry is rough sorted, different grades being chosen for length of fibre and sent to "cobbing sheds" where dressing is carried out. This consists of separating the abestos fibres from the surrounding rock and may be done (*a*) by hand — the stone being broken by a small sledge hammer and the fibre thrown thereafter into one box and the waste into another — or (*b*) by machine ("mills").

Hand separation ("picking and sorting") is not difficult since the fibres lie in layers more or less loosely attached to the rock and can frequently be picked off with the fingers; but hand dressing is not thorough and the waste material from the cobbing tables contains much fibre, the further utilisation of which represents large profits to the mine, and these fine pickings have to be dressed mechanically. They are passed after drying to crushers where they are broken by successively finer-set rollers and further reduced by cylindrical fiberisers and the cyclone machine, which reduces them to fine powder. The cotton-like fibres come to the surface and are mechanically withdrawn by suction. The pulverised rock and ground asbestos are then separated by passing them through a shaking screen. In some mills the particles of iron present are taken up by strong electric magnets. The crude fibre on separation from the waste rock resembles mineralised wool. The asbestos is graded according to the length of fibre on a series of cylindrical screens. It is then carded and packed in sacks and placed on the market in bulk.

Industrial Uses

The uses to which asbestos is put are very varied. The long fibres are used in the textile industry for the manufacture of cloth, braid, rope, theatre curtains, decoration, upholstery, hangings, and for finishing pistons for steam engines. The shorter fibres together with an agglomerating agent are used in the manufacture of felt, paper, cardboard, varnishes, covering agents (especially for strong

rooms), cements, rubber tyres for automobiles and bicycles. Asbestos is also used as an electrical insulator for covering electric wiring for high and low pressure currents, in the manufacture of tiles, planks, panelling and exterior coverings for buildings, mattresses, heat insulating gloves, firemen's clothes, laboratory equipment, porcelain, balls for gas fires (made of fireclay and asbestos), etc.

Dangers and Hygiene

The worker engaged on the extraction and manufacture of asbestos is constantly exposed to danger from dust, more especially, however, in the operations of spinning and weaving.

The baneful influence of the dust in relation to the production of tuberculosis is brought out in the *Annual Report of the Chief Inspector of Factories for England and Wales for 1910*, which records 5 deaths from tuberculosis amongst 40 workers engaged in an asbestos weaving factory, where the most dangerous process was revealed to be the weaving of asbestos mattresses composed of bags of woven asbestos filled with short asbestos fibre. They were placed on a table and beaten out flat by a man with a wooden flail, which process occasioned the production of much dust. Women sewed the mattresses into sections using asbestos threads and as they worked beside the beaters they also of necessity inhaled much dust.

Towards the beginning of 1924 a fatal case of poisoning by asbestos dust was reported at Rochdale (England). The victim was a woman who had been employed in an asbestos factory. A post-mortem examination revealed death as due chiefly to a fibrosis of the lungs due to the inhalation of mineral particles and in part to tuberculosis. A microscopic examination of the lungs and of dust samples taken from three different workshops of the factory revealed the same black particles present in each, alike in form and dimensions.

Another fatal case was studied in 1927 by Cooke and Hill (Great Britain).

A striking example of the noxious property of asbestos dust is instanced by the following case: in an asbestos spinning and weaving factory (Calvados) there were 40 deaths in the five years 1890-1895. The mortality rate decreased considerably with the installation of a good system of local ventilation over the carding machines. (See also article "Tuberculosis".) In an asbestos weaving factory in Saxony the workers complained of headaches and loss of appetite. These affections were caused by fumes given off by a rubber solution with which the asbestos cloth had been coated to eliminate dust.

An investigation carried out in Canada in 1911 revealed that hygienic conditions were greatly improved as a result of the adoption of an effective system of ventilation. Nevertheless, a local doctor with long experience stated that respiratory troubles, and in particular tuberculosis, were of very frequent occurrence amongst asbestos workers.

Rambousek drew attention to the risk of lead poisoning during the weaving of asbestos when a thread of lead is added to the weft, as is sometimes the case.

Marked frequency of chronic conjunctivitis with hypertrophy of the rims of the pupils has also been noted amongst the asbestos workers of the Island of Cyprus. The workers in question were chiefly engaged in grinding, screening, and packing the asbestos in sacks.

The weaving of asbestos has only developed importance during the last twenty years. Now that it is widely practised the application of local exhaust systems during such processes as that above described is called for. All processes from extraction onwards unquestionably involve a considerable hazard, and American and Canadian life insurance companies generally refuse asbestos workers on account of the assumed deleterious conditions in the industry.

The lack of more accurate and detailed data in medical literature regarding this industry in its various branches, including the utilisation of by-products, is to be deplored in view of the self-evident importance of asbestos dust as a predisposing cause of pulmonary tuberculosis, more especially since the rapidly-increasing development of industries utilising asbestos adds greatly to the urgency of studying the conditions with a view to their amelioration.

All provisions already described for withdrawal of dust should be enforced in this industry.

Legislation

In *Italy* boys under 15 and girls under 21 years of age are excluded from workshops where dyeing and weaving of asbestos are engaged in, unless effective

means are taken to prevent the diffusion of dust throughout the atmosphere.

BIBLIOGRAPHY

AURIBAULT. "L'hygiène et la sécurité des ouvriers dans les filatures et tissages d'amiante": *Bull. de l'Insp. du Travail*, 1906, pp. 120-132. Paris.

SIMPSON, F. W., in *Brit. Med. Journ.*, 26 May 1928, p. 885. London.

UNITED STATES DEPARTMENT OF LABOUR, BUREAU OF LABOUR STATISTICS. *Bulletin No. 231*, pp. 176-180.

***

Ashes, Cinders

French: *Cendres.* — German: *Aschen.* — Italian: *Ceneri.* — Spanish: *Cenizas.*

Ashes present problems from the point of view of industrial hygiene, not only when cinders from hearths are in question (dangers from hot dust, presence of certain products, etc.), but especially when they are employed for special purposes. Thus, for instance, treatment of goldsmiths' ash by lead may cause injury by reason of the metallic dust which it liberates.

Hygienic measures assure covering of any openings giving on to the public streets, thorough ventilation of the workshop, the installation of hoods for the furnaces and cupels connected to the chimney by a strong exhaust. If the treatment is effected by means of mineral acids, application of measures of protection against toxic fumes is required, viz. condensation of lead fumes. Noisy machinery should be isolated, and measures applied for evacuation of residues and the protection of workers from the heat and glare of the furnaces.

Pearl ash from combustion of organic material, such as shoots of vine, wine lees, vinasse from beetroot, etc., is composed of impure potassium carbonate and its treatment liberates sharp, piquant, thick, and disagreeable fumes and injures vegetation in the neighbourhood. Factories should be erected at a long distance from dwelling houses and calcination should be effected in closed ovens. Gases and fumes should be led to special hearths or requisite measures taken for their destruction. The chimney must be a very high one. It would be advisable to collect the incandescent mass of carbonate of potassium in a special apparatus so constructed that the gases can be led to the hearth or into apparatus where they can be burnt. Hoods should be installed above the evaporation boilers. The water should be led to a sewer: the residues require fairly frequently to be removed and can be utilised as manure.

By the expression "dust shot with a lead content" is meant the impure oxides which form on the surface of each bath containing lead or lead alloys. This scum or black dross is removed as it forms and is often put aside on the floor, where it becomes mixed with other substances. In appearance it resembles ashes, hence the French expression "*cendres de plomb*".

Kelp ash is lixiviated for the extraction of the salts of potassium. The ash is got by burning different kinds of sea-weed known as kelp or wrack and thrown up on the seashore. Dried in the sun, they are thereafter reduced to ashes in furnaces lined with refractory bricks. The product of combustion "kelp ash" is in the form of a solid compact block, from which the salts of potassium are extracted. The blocks are then ground and put into special apparatus for gradual dilution with water. The solution is concentrated in iron boilers and poured into vats where, after cooling, the potassium chloride is deposited in crystals, which are then subjected to purification.

Hygienic precautions to be recommended are impermeable flooring, cement walls to the height of one metre, hoods provided above the boilers to draw off the fumes to the chimney, all openings to the public highways to be kept closed, and neutralisation of the water used before it enters the sewers.

The Recommendation adopted at the International Labour Conference at Washington (1919) regarding the protection of women and children against lead poisoning demands the exclusion of women and young persons under eighteen years of age from work involving manipulation, treatment, and reduction of ashes containing lead.

***

Asphalt

(Mineral Pitch, Hard Bitumen)

French: *Asphalte, Poix minérale, Bitume.* — German: *Asphalt, Erdpech, Erdharz.* — Italian and Spanish: *Asfalto.*

Earth and rocks impregnated with bitumen are called asphalt. In mineralogy, however, the name asphalt is also given to bitumen in a free state. It is highly important to distinguish between these two products. Asphalt is generally in the form of fine grained rock, black or dark brown in colour according as it is more or less

HANDBOOK of DANGEROUS MATERIALS

No. 2468
Da NOV 26 1951
JOPLIN, MO.
660.2
La 9

Handbook of Dangerous Materials

By

N. IRVING SAX

Toxicologist, General Electric Co.
Schenectady, N. Y.

Assisted by

M. J. O'HERIN

Fungicide Laboratory
General Electric Co., Schenectady, N. Y.

and

W. W. SCHULTZ

General Engineering Laboratory
General Electric Co., Schenectady, N. Y.

BOOK DIVISION

REINHOLD PUBLISHING CORPORATION

330 West Forty-Second St., New York 18, U.S.A.

1951

COPYRIGHT 1951 BY
REINHOLD PUBLISHING CORPORATION

All rights reserved

PRINTED IN U.S.A. BY
THE MAPLE PRESS COMPANY, YORK, PA.

Arsenical Mixtures or Compounds, N. O. S.* Solid

Hazardous Properties: Poisonous materials. See **Arsenic.**

Shipping Regulations:†

Poison B 352, 354* Poison label 200 lbs. (max. lot)

Arsenical Dip, Liquid (Sheep Dip)

Hazardous Properties: Poisonous liquid. See **Arsenic.**

Shipping Regulations:†

Poison B 338, 349* Poison label 55 gals. (max. lot)

Arsenical Dust

Hazardous Properties: A poisonous material. See **Arsenic.**

Shipping Regulations:†

Poison B 352, 355* Poison label 200 lbs. (max. lot)

Arsenical Flue Dust

Hazardous Properties: Poisonous material. See **Arsenic.**

Shipping Regulations:†

Poison B 352, 355* Poison label 200 lbs. (max. lot)

Arsenous Acid, Solid

Hazardous Properties: A poison. See **Arsenic.**

Shipping Regulations:†

Poison B 352, 361* Poison label 200 lbs. (max. lot)

Arsenous and Mercuric Iodide Solution, Liquid

Hazardous Properties: A poisonous liquid. See **Arsenic** and **Mercury.**

Shipping Regulations:†

Poison B 338, 349* Poison label 55 gals. (max. lot)

Arsine

*Maximum Allowable Concentration in Air:** 0.05 parts per million.

Hazardous Properties: See **Arsenic.** Caution! Arsine can be liberated from unusual places. For instance, in cleaning out steel tanks which contained concentrated sulfuric acid it is possible to liberate arsine; although concentrated sulfuric acid does not react with steel, dilute acid does react with it momentarily, liberating hydrogen, which reacts with the arsenic, forming arsine. This is but one of the very numerous possibilities for industrial exposure to this toxic material.

Synonyms: Arsenic hydride; arseniuretted hydrogen

Formula: AsH_3

M. Wt.: 77.9 *M. P.:* −113.5°C

B. P.: −55°C *Density:* 3.484 g/liter

Description: Colorless gas

Shipping Regulations:† Poison label (1).

Arsphenamine

Hazardous Properties: A toxic material. See **Arsenic.**

Synonyms: 3-Diamino-4-dihydroxy-1-arsenobenzene hydrochloride; 606

Formula: $C_{12}H_{12}As_2N_2O_2.2HCl.2H_2O$ *M. Wt.:* 475.0

Description: Light yellow hygroscopic powder

Shipping Regulations:† None.

"Artic"

Trade mark of a refrigeration grade of methyl chloride. A proprietary material. See under component as listed.

Asbestos Particles or Asbestos Dust

*Maximum Allowable Concentration in Air:** 5 million particles per cubic meter.

Hazardous Properties: Asbestos is a class name for various minerals of a fibrous nature. The commercial material is mainly fibrous serpentine, known as chrysotile. The implication is that this material merely constitutes a nuisance dust. The figure given for the M. A. C. is probably the safe limit. There is a specific lung disease known as asbestosis, which may be caused by this material; it can also cause chronic conjunctivitis with hypertrophy (6). Asbestosis is a comparatively new disease which was first described by English writers (1920 to 1925). Finely ground asbestos is said not to be dangerous, and this theory is supported clinically. The time necessary to develop asbestosis is from 7 to 9 years, if the exposure concentration is fairly high. Lower concentrations are likely to require 15 to 20 years of exposure. Once established, the disease is progressive, even though exposure has stopped. It would seem that the irritation due to this material is related to its physical nature rather than its chemical composition. The first sign is dyspnea; the cough is usually dry and auscultation reveals few if any physical signs; loss of weight, pallor and cyanosis are also early symptoms. Later, emaciation and chest symptoms appear. There is a distinction between this disease and silicosis, *i.e.*, the chest appears emaciated and lacks the robust character noticed in silicosis. Occupational exposure occurs in the packing, insulating and fire-proofing industry or where this material is combined with textiles.

Treatment and Antidotes: Rest and removal from exposure are indicated. Consult a physician.

Storage and Handling: Personnel exposed to high concentrations or to unknown concentrations of asbestos particles should wear respiratory protection of an approved design.

Asphalt

Hazardous Properties: Asphalt causes skin eruptions which vary roughly in relation to the specific gravity and boiling point of the asphalt or bitumen. It can cause dermatitis. The fumes evolved are noxious and unpleasant with a strong odor so that kettles of this material

ELKINS

CHEMISTRY OF INDUSTRIAL TOXICOLOGY

WILEY

RESEARCH LIBRARY
No. 2175
Date Received 9/18/50
EAGLE-PICHER CO.
JOPLIN, MO.

CIVIL
EL 5

The CHEMISTRY of INDUSTRIAL TOXICOLOGY

HERVEY B. ELKINS, Ph.D.

Chief of Laboratory
Division of Occupational Hygiene
Massachusetts Department of Labor and Industries

JOHN WILEY & SONS, INC., NEW YORK
CHAPMAN & HALL, LIMITED, LONDON

COPYRIGHT, 1950

BY

JOHN WILEY & SONS, INC.

All Rights Reserved

This book or any part thereof must not be reproduced in any form without the written permission of the publisher

PRINTED IN THE UNITED STATES OF AMERICA

CHAPTER XI

NATURAL AND INDUSTRIAL PRODUCTS

MINERALS

The most important minerals, from the standpoint of industrial hygiene, are those containing free silica. These include sand, sandstone, flint, and quartz, which consist mainly of silica in the form of quartz; diatomaceous earth, which is essentially amorphous silica; and granite, which contains from 20 to 40% quartz.

Minerals that contain combined silica (silicates) but no free silica are incapable of causing silicosis. Asbestos, however, causes a fibrotic lung condition known as asbestosis, which resembles silicosis in many respects and can be almost as serious. Mica dust is also considered somewhat hazardous to inhale and in addition may cause skin irritation.

Non-siliceous minerals, like limestone, marble, dolomite, etc., which do not contain toxic elements, do not ordinarily present any significant dust hazard. Minerals containing toxic elements, such as calcite and cryolite (fluorine), and pyrolusite (manganese) may cause systemic poisoning if the dust is inhaled or ingested in quantity. As a rule, such minerals are less reactive than synthetic compounds of the same elements, however, and may be relatively inert and innocuous in comparison.

A detailed discussion of silica and other mineral dusts, their occurrence, effects, and evaluation, is given in *Industrial Dust*, by Drinker and Hatch.[219]

ABRASIVES

Abrasives containing free silica, such as sandstone grinding wheels, flint sandpaper, and sand used for abrasive blasting processes, all present a silicosis hazard. Most artificial abrasives, however, consist of silicon carbide, aluminum oxide, boron carbide, or other inert and non-toxic substances. Rouge, or iron oxide, used for polishing, is also harmless for practical purposes.

Steel shot or grit commonly replaces sand for cleaning castings in the modern foundry, and in itself produces no dust hazard.

GLASS

The industrial hygienist is interested in glass primarily when it is in the form of glass wool or Fiberglas. This useful material causes considerable skin irritation when it comes in intimate contact with the skin. The possibility of lung injury from inhalation of the fine particles has been raised repeatedly; but, according to the best evidence, pulmonary effects cannot arise from this source.

COAL AND ITS PRODUCTS

Coal itself, even bituminous coal, is relatively inert, and inhalation of its dust is more inconvenient than harmful. When soft coal is destructively distilled, however, not only are gaseous and volatile liquid hydrocarbons (benzene, toluene, etc.) produced, but tars and pitches of varying degrees of consistency are obtained. These are mixtures of various complex aromatic hydrocarbons, cresols, amines, and similar substances. In general, coal tar, coal-tar pitch, anthracene oil, and other such fractions are skin irritants and may contain carcinogenic agents. Thus skin cancer is a definite hazard to workers in contact with coal-tar pitch, soot, etc.

Although there has been some incidence of skin cancer from coal tar in this country, much more has been reported from England. Chimney sweeps especially, in that country, have frequently been afflicted. It seems probable that English coal tar may be more carcinogenic than that produced from American coal. It is possible, however, that differences in technical processes or racial susceptibility may be responsible.

When coal-tar products are handled, skin contact should be avoided as far as possible. Regular medical examinations will detect skin cancers at an early stage, when they can usually be removed before permanent damage is done.

PETROLEUM AND ITS PRODUCTS

The volatile components of petroleum consist mainly of aliphatic hydrocarbons, which have been discussed in Chapter VII. In addition to paraffins and olefins, some petroleums contain relatively high percentages of naphthenes and aromatic hydrocarbons.

The less-volatile fractions of petroleum are used as fuels, lubricants, and construction materials (asphalt). These substances are somewhat irritating to the skin, and some are even carcinogenic, but much less so than coal-tar products. Asphalt, for instance, causes much less skin trouble than coal-tar pitch, although both have similar applications. Mule spinner's cancer, however, is caused by the oil used for lubricating certain machines (mules) in the process of spinning cotton. This disease, too, is reported primarily from England and is rare, if not unknown, in this country.

Cutting Oils

Although the lubricating oils for motors and machinery are not notorious skin irritants, the similar oils applied as lubricants and coolers for the machining and cutting of metals are responsible for one of the most widespread of all occupational diseases, cutting-oil dermatitis. This condition is probably known in the majority of machine shops and is prevalent in many. It is caused primarily by the insoluble cutting oils, and not by the water emulsions frequently used as cooling agents.

Because cutting-oil dermatitis appears and disappears without apparent reason, many persons have looked for causative factors other than the oil itself. Bacteria have frequently been blamed, and disinfecting agents are sometimes added to the oil, or it may be periodically sterilized with heat. Some authorities deny that the trouble is of infectious origin, however.[220] The presence of small metal particles in the oil, which inflict small cuts in the skin, are believed by some to be important, and their removal by filtration is recommended. In other situations, changing from one oil to another may bring relief, and the presence of a specific irritant not present in all oils is then inferred.

It is probable that some incidence of dermatitis is to be expected wherever there is widespread, continued contact with any cutting oil. The most effective preventive measures are based on promoting prompt removal of excess oil from the skin and preventing unnecessary contact with the oil. Morris recommends a mixture of sawdust and liquid soap as a cleaner for removal of cutting oil.[220]*

* Also recommended—equal parts of cornmeal and the following cleansing solution: sulfonated oil (corn, castor, neat's foot, etc.), 45%; light liquid petrolatum, 45%; gelatin, 25% aqueous solution, 10%.

WOODS AND PLANTS

Skin irritation from poison ivy and poison sumac or oak is an occupational hazard for many outdoor workers. The United States Public Health Service has recommended protective creams to be applied to exposed areas *before* workers enter infested areas.[221] One successful formula consists of stearic acid, 20 grams; potassium hydroxide, 1.4 grams; water, 80 ml; and 90% alcohol, 4 ml.; after these are mixed, 10 grams of sodium perborate are added. The sodium perborate oxidizes urushiol, the active ingredient in poison ivy, forming a non-irritant product.

In addition to these widely distributed irritant plants, certain weeds, especially in the southwestern part of the United States, are also skin irritants.

Cashew-nut oil is highly irritant, and severe dermatitis has resulted from handling cashew nuts.[222] Roasting the nut is said to destroy the irritating ingredient of the oil.

Sawmill operators, woodworkers, and carpenters are, of course, exposed to the dust of various kinds of woods. Certain of the rarer woods, including cocobolo, cytisus, acacia, yew, juniper, satinwood, black ebony, boxwood, mahogany, redwood, rosewood, teak, tagayasan, sabicu, roko, iroko, Gambala, and cedar have been classed as toxic.[223, 224, 225] In addition, there is some evidence that maple and birch sawdusts contain some chemical irritants.[226]

For the most part, however, wood dust has little toxic effect but may cause some irritation of eyes and upper respiratory passages.

ANIMAL AND VEGETABLE FIBERS

There is considerable controversy over the effects of inhalation of many of the vegetable fibers, such as cotton, jute, and hemp. A mild febrile condition, known as cotton or jute mill fever, is fairly prevalent in plants where the dusts of these fibers are found. This is apparently similar to metal fume fever; it seldom causes lost time, and immunity is acquired after a few days' exposure.

More serious and lasting effects (i.e., byssinosis), resulting in rather widespread involvement of the respiratory system, have been attributed to these fibers, but their etiology is not entirely clear.

Bagassosis, another serious lung disease, occurs among workers with bagasse, the residue from sugar cane.

Wool in itself is not physiologically active, but raw wool from certain areas of the world may contain anthrax spores.

Furs are also not ordinarily harmful in themselves, but the dyes and finishes employed are frequently irritant or toxic.

LEATHER

The handling of green hides from certain parts of the world may involve contact with anthrax spores. The process of tanning destroys or removes these spores effectively, however.

Many toxic and irritant chemicals are required in tanning leather, but the finished product is ordinarily harmless, for practical purposes. In a few cases, however, workers handling leather have contracted a dermatitis unquestionably due to some ingredient in the leather. Although the chromium in chrome-tanned leather is believed to be completely in the tervalent state, which is non-irritating, it is claimed that chrome-sensitized individuals may contract dermatitis from chrome-tanned leather. Other ingredients which have been suspected of causing "leather itch" have been formaldehyde and certain fillers. Finishes that contain irritant ingredients may also be encountered occasionally on leathers.

RUBBER

Natural rubber itself is non-toxic and non-irritant. Similarly, the synthetic rubbers, after polymerization, are apparently physiologically inert, although the raw materials involved in their synthesis include such toxic substances as acrylonitrile and chloroprene (neoprene).

Natural rubber latex contains ammonia, which may present a problem when the latex is heated. Neoprene latex under some conditions contains small amounts of impurities, probably partly polymerized chloroprene, which give off a strong odor, and this situation has occasionally led to complaints of ill effects. These were probably largely psychological, as the quantity of volatile substance in some of these products was found to be negligible.

Finished rubber contains fillers, plasticizers, accelerators, antioxidants, retarders, vulcanizing agents, and possibly pigments, in addition to the rubber itself. Most of these are inert or innocuous, but a few require consideration from the toxicologist.

Lead oxide was formerly used extensively as an accelerator for

natural rubber, mainly in the process of making hard rubber. The reprocessing, grinding, or cutting of such rubber might produce lead poisoning, but limited data suggest that this hazard is slight.

Antimony sulfide, employed as a pigment in red rubber, has apparently caused no difficulties, owing undoubtedly in large measure to its insolubility.

Of the organic accelerators, hexamethylene tetramine (see Chapter X) is by far the worst offender. Its propensity for causing dermatitis among workers handling partly processed rubber has been responsible for its abandonment in most rubber formulas. Other accelerators, retarders, and antioxidants have irritant properties but have caused only occasional difficulty. Davis discussed these substances in detail, but many new ingredients have been introduced since his work was done.[227] Later reports have covered some of these.[231]

Selenium compounds appear to some extent in synthetic rubber formulations, but to date no selenium hazard has been demonstrated in rubber processing.

Spolyar reports a highly toxic fume, tetramethyl succinonitrile, given off when Porofor N, used to make sponge rubber, is heated.[228]

PLASTICS

The compounds of high molecular weight which are the main components of plastic materials are inert and non-toxic. Frequently some unreacted material is present and may cause trouble. Formaldehyde, in urea-formaldehyde and phenol-formaldehyde plastics, is one of the commonest offenders in this respect, especially in the uncured plastics. Phenol-formaldehyde varnishes have been particularly bad producers of dermatitis, under certain circumstances.

In addition, certain plasticizers, such as tricresyl phosphate and camphor, which are sometimes present in plastics, are toxic and may be volatilized if the material is heated. The more common plasticizers, such as castor oil and various esters of phthallic acid, have caused little trouble, however.

Nitrocellulose plastics, such as Celluloid, present a fire hazard since, in addition to being very flammable, they give off quantities of nitrogen dioxide when they burn. Other nitrogen-bearing plastics, such as Melamine, have been said to give off hydrogen cyanide

when heated, but the quantities are apparently not sufficient to produce real danger.

Vinyl carbazole, used in the preparation of dielectrics, caused a number of dermatitis cases in a Massachusetts plant.[229]

ADHESIVES

Water-soluble glues present little or no problem to the industrial toxicologist. Phenol-formaldehyde cements may cause dermatitis. Most cements made from plastics or rubbers contain volatile solvents which may produce a vapor hazard.

Pyroxylin cements may contain benzene but more often are dissolved in acetone or other ketones, esters, and alcohols. Natural rubber is usually dissolved in naphtha, but benzene may be used, and occasionally carbon tetrachloride or carbon disulfide. The solvents commonly found with synthetic rubbers, according to Greenburg and Moskowitz, are as follows:[230]

Buna S and butyl rubber: naphtha.
Thiokol: ethylene dichloride; methyl ethyl ketone.
Buna N: ethylene dichloride; propylene dichloride.
Neoprene: benzene; toluene; carbon tetrachloride; trichloroethylene.

The naphthenes, especially cyclohexane, serve to some extent as rubber solvents.

PAINTS

Paints ordinarily consist of oil-dispersed inorganic pigments, mixed with a thinner which is usually a hydrocarbon, such as turpentine, hydrogenated naphthalene, or relatively non-volatile petroleum fractions. Owing to the low volatility of the solvents, a vapor hazard does not commonly occur in painting except in confined spaces.

The most important toxic pigments used in paints are the carbonates, sulfate and oxide of lead. The best outside paints usually contain a white lead pigment, whereas red lead paints are used for priming coats. All such paints present a lead-poisoning hazard when sprayed or sanded. When metal coated with a lead paint is welded or cut with a gas or electric torch, toxic lead fumes are given off.

Lead chromate is a common ingredient of yellow and green

paints. Cadmium compounds are sometimes used in yellow and red pigments. Mercury compounds are used in non-fouling paints for ship bottoms. Zinc chromate is employed in certain priming paints. All these present some hazard if the paint is sprayed or handled carelessly.

Paint ingredients which are considered inert include zinc oxide, barium sulfate, and titanium dioxide.

VARNISHES

Varnishes usually consist of oils and resins dissolved in a hydrocarbon solvent. Although highly toxic solvents are rarely employed, the less-volatile aromatics (xylol and coal tar naphtha) are common, and a solvent vapor hazard is more probable than with paints. Occasionally varnishes contain phenol-formaldehyde resins which are skin irritants.

ENAMELS

Enamels are essentially pigmented varnishes, but pigments are used chiefly for coloring. Lead chromate, and cadmium sulfide, and cadmium selenide are the compounds of toxic elements most frequently encountered.

Vitreous enamels, on the other hand, are water dispersions of silicates and may contain lead.

LACQUERS

Lacquers are solutions of plastics, most commonly nitrocellulose. Alcohols and esters, or ketones, are usually present in the solvent, which may also contain a hydrocarbon. Benzene is the most toxic solvent ordinarily found in lacquers, and it is now rather uncommon. Butanol is very common, and methyl isobutyl ketone is also widely used. Some special lacquers contain nitroparaffins.

In colored lacquers the pigments are essentially the same as those used in enamels.

DYES

As a group, dyes are apparently of low toxicity, in spite of the prevalence of nitro and amino groups which, in simpler compounds, are associated with harmful action to the body. Many dyes are quite irritating when inhaled, however, although evidence of systemic action is absent.

Fur and hair dyes, in comparison with textile dyes, have a bad reputation, largely because *p*-phenylenediamine, a powerful skin irritant, is one of the best black dyes for furs. Dermatitis from furs and fur felt dyed with this compound is not uncommon. On the other hand, some beauty shops have used *p*-phenylenediamine as a hair dye, apparently without serious results.

In spite of the absence of proved occupational disease, the handling of dry dyes involves a disagreeable and possibly deleterious exposure to dust, which should be controlled by suitable ventilation and enclosure of the dusty processes. In Massachusetts dye-blending plants, dust concentrations up to 190 mg per cubic meter and averaging about 30 mg were found. Concentrations above 25 to 50 mg are considered excessive and unnecessary, if proper control measures are taken.

INSECTICIDES

The use of insecticides has increased greatly in the last few years, and with it the potential hazard to the health of workers engaged in manufacturing and applying these substances.

Formerly various arsenic compounds were mainly relied upon for the control of insect pests. The trend recently has been toward organic compounds, of which there are four main types: natural substance extracts, such as rotenone and pyrethrum; chlorinated compounds; phosphorus compounds; and thiocyanates.

Rotenone and pyrethrum have been used for some time without notable evidence of toxicity to humans.

Of the organic chloride insecticides, DDT and benzene hexachloride have already been discussed. Others include chlorinated camphene ($C_{10}H_{10}Cl_8$); Chlordane ($C_{10}H_6Cl_8$), a derivative of indane; and various derivatives of DDT, such as dichlorodiphenyldichloroethane, and dimethoxydiphenyltrichloroethane. Of these, the first two are of a somewhat greater toxicity than DDT, whereas the last two are materially less toxic.[232]

The phosphorus compounds used as insecticides include tetraethylpyrophosphate, hexaethyltetraphosphate, and Parathion (diethyl *p*-nitrophenylthiophosphate). The acute toxicities of these compounds are considerably higher than that of DDT.[232]

The organic thiocyanates, sold commercially as Lethane or Thanite, apparently have a toxicity of the same order as DDT.

INDEX

(Italic numbers indicate pages on which illustrations appear.)