The Principles and Practice of INDUSTRIAL MEDICINE

Edited by

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FOREWORD

Advancement in the knowledge of industrial medicine presupposes a far wider dissemination of instruction than that which has obtained to this time. The instructional effort in this field must increase tremendously, both in its purely didactic and in its clinical aspects before the student in medicine may be considered as having received adequate general training. Not only for the student, but for those already engaged in the art of healing is such instruction eminently useful.

Concern for the welfare of the industrial worker is no longer a matter in the realm of mere sentiment. It has become a vital, concrete necessity even when completely divorced from its humanitarian implications. The day has actually come when the absence of well-conducted medical departments in industry is suggestive of medievalism when measured by the criterion of social progress and of financial obtuseness when measured by the more tangible criterion of profits and losses.

Scientific discovery, in its ever accelerating pace, has brought before us new concepts of the relationship of the environment to physical well-being. It is inevitable that industry take cognizance of these facts and be guided by them in providing protection for its workers. The great strides in scientific knowledge have widened the scope of industrial medicine; but our contribution to the worker's health will not be commensurate to the possibilities unless we are, as physicians, aware of the broadened horizon.

We have noted, with the utmost gratification, that of recent years many of our medical schools have included training in industrial medicine in their curricula. We have noted, moreover, that the new offering is being regarded less and less as usurping a place in the roster of courses and more and more as an integral part of medical training. This augurs well for industrial medicine and will assure for it an increasing receptivity both from the worker and from management.

Warm commendation is justly forthcoming to the editor of this book and to the individual contributors. They have made a signal contribution to the student of medicine whether he be still in school or in the active practice of his profession. Their contribution is, indeed, a particularly timely and loyal service to the nation at a moment when a dire need for this service exists.

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PREFACE

The purpose of this book is twofold. In the first place, many practicing physicians are being asked to take on the medical care of industrial workers when they never have had experience in industrial medicine before. This book should give them a general idea of the principles of industrial medicine. Secondly, medical schools should be placing more emphasis on the teaching of industrial medicine to medical students so that the oncoming physicians will know how to step in and do a real job of health protection for the man who works.

Industrial medicine differs from regular practice principally in the viewpoint. Here the physician is working with well people trying to keep them well. This, then, is a new discipline. Industrial medicine thus becomes the practice of adult health. New techniques have been and are being developed which help the industrial physician better to do the job of keeping the worker well. Some of these techniques are brought out in this book. Starting with these, the alert physician will develop other methods of his own.

I want to record here my appreciation of the willingness with which the contributors undertook their jobs in a time of unusual stress and when there were many demands upon their time. This was done in spite of the fact that some of the men had military and governmental responsibilities thrown upon them that took much of their time after they had promised to contribute to this book.

I wish to express my appreciation to J. Clarence Funk, Sc.D., who read much of this material and made valuable suggestions as to clarity of statement, and to Prof. Harvey B. Haag, my colleague, for reviewing the chapters in the toxicological field. I should also here record my appreciation for the work that Mrs. Wampler put on this book. She has done most of the secretarial work in my office in connection with this publication.

The thirty-three contributors to the book are now joined together in a little family of workers in a common cause. It has been a pleasure to work with scientific men and women like these. We are sorry to have to record that Professor Don Cumming, one of the youngest, ablest and most genial of the group, lost his life in an airplane accident a short time after he had sent in his manuscript.

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CONTENTS

Chapter I

NOTES ON HISTORY

Fred J. Wampler, M.D.

Industrial medicine is largely a development of the last hundred and forty-three years—the machine age—but occupational diseases were known from before the Christian era. The more important stepping stones in the development of our knowledge of occupational diseases will be outlined in this chapter.

In the early days labor was performed largely by slaves. The nearest approach to our modern industrial practice was when an individual's slaves would become proficient in some type of work. This slave owner would then contract for this particular type of service. Ax grinding is known to have been done in this way and mining and other types of work could easily have followed this line of development.

Hippocrates (about 460–370 B.C.) described a disease that might well have been hookworm; those suffering from it ate stones and earth, had great intestinal disturbances and were jaundiced. Insanitation of mines up until modern times made them ideal places for the spread of hookworm disease.

Other diseases described by Hippocrates are those involving metallurgists, fullers, tailors, horsemen, farm hands and fishermen. He describes the metal worker as being pale and livid, with difficult respiration, a distended, hard abdomen, large spleen and a swollen right hypochondrium.

Pliny the Elder (23–79) described a protective mask for miners, which consisted of a bladder tied over the mouth to prevent inhalation of poisonous dusts and vapors. Pliny recommended to wine dealers the use of cabbage and bitter almonds as a protection against alcoholic intoxication. He described poisoning with zinc and sulphur.

Galen (131–201), the founder of experimental physiology, has many references to occupational diseases. He visited a copper mine on the island of Cyprus and was nearly overcome with fumes. He writes that the workmen who carried out a vitriolic liquid ran with all speed from the mine to avoid death in the midst of their labors. Galen also said students and scholars are exposed to a hazard from the fumes of the tallow candles they were forced to use. Other occupational diseases mentioned by Galen concerned runners, farmers, wrestlers, wet nurses, gypsum workers and those who used their voices excessively.

As times advanced others made important contributions in the field of occupational diseases, such as Agricola, Ulrich Ellenbog and Paracelsus. Georg. Agricola (1494–1555), in his treatise on mining and metallurgy, refers to dangers and diseases to which miners and refiners are exposed. Ulrich Ellenbog of Swabia, Germany, described in 1473 the symptoms of mercury and lead poisoning (headache, visual disturbances, paralysis and unconsciousness) and gave advice concerning their avoidance. This was the first work written expressly on the subject of industrial metal poisoning. Paracelsus (1493–1541), a Swiss, wrote a treatise dealing exclusively with occupational diseases. This was not published until 1567, twenty-seven years after the author's death. He wrote on metals and minerals known at that time and gave the symptoms caused by each and the therapeutic procedures to be used in case of poisoning. It is supposed that Paracelsus himself was a victim of his enthusiasm in the investigation of the noxious influences found in mines and that he died as a result of heavy metal poisoning.

We now come down to Ramazzini (1633–1714), known as the Third Hippocrates and the Father of Industrial Medicine, who wrote an excellent work on the diseases of tradesmen, the "De morbis artificum diatriba." His work was published in Padua in 1700 and was written so well that it was more than one hundred years before it was improved upon. He made an exhaustive review of the literature and his book contained most of the information in this chapter that precedes this paragraph. He described about a hundred different occupations and the special hazards involved in each. His descriptions were based on his own clinical observations. The book was written for practicing physicians that they might be acquainted with the various trades and to advise the proper treatment and prevention of occupational diseases.

Even a brief history of industrial medicine would not be complete without mention of the influence that the craft guilds had upon the lives of skilled workmen in the fourteenth century and afterwards. The guilds were formed primarily for the protection and the regulation of certain trades; these were generally organized ultimately into monopolies. The guild controlled the admission of apprentices, controlled the hours of labor and regulated wages and the selling price of finished products. The standards of the trades were upheld by frequent inspections. It can be seen from this that they completely controlled the personnel and the quality of the finished products of the trades. The physical and social welfare of the individual member of these guilds was considered quite important and this was a step in the advancement of better working conditions in the production of goods. Some guilds of this general type still

exist in China and India although they have long since died out in countries where machinery has taken the place of hand labor. The famous Bidar ware produced in Hyderabad, a native state in India, is a good example of such a guild at the present time. Here the guild owns the secret process for making this product. Diamond cutting and some of the Swiss watch making are still done by secret techniques handed down from father to son by the old craft guild system.

The introduction of machinery in the production of goods changed the whole industrial picture. In England the factory system developed rapidly in the latter part of the eighteenth century. While workers were generally well paid, the physical welfare of the employee was neglected. Long hours were the rule and machinery was unguarded. Lighting and ventilation were bad. Under such conditions the accident rate was high and industrial diseases prevalent. The mortality rate of workers was higher than that of farmers or other non-factory workers. Child labor was the general rule and oftentimes the child had to work twelve hours in twenty-four. The Health and Morals Act to regulate the labor of bound children in cotton factories was passed by Parliament in 1802.

The great prison reformer, John Howard (1762–90) was one of the leaders in laying the foundations of modern hygiene. He visited most of the known prisons of England and Europe and wrote accounts of his findings. He thus gave publicity to the inhuman and often corrupt administrations which he found. He might be said to have established the value of inspection and reporting, which are strong arms of modern hygiene.

Edwin Chadwick (1800–90), a barrister who became interested in life insurance, studied the effect of environment on the length of life. Chadwick in 1832 was appointed to investigate the workings of the Poor Laws. When a factory commission was set up by the Factory Act of 1833, Chadwick was the secretary and executive officer of the commission which was composed of three appointed members. Professional inspectors were appointed by this commission and they were a very potent force in the proper conduct of factories. (See Newsholme.)

Labor legislation in the United States has lagged considerably behind that of England (1897) and of the more progressive (Germany 1882) countries on the continent. The year 1911 marked the beginning of effective legislation in the United States. In that year California and New York made compulsory the reporting of occupational diseases and New Jersey made certain occupational diseases compensable. Now (1943) all the States, except one, have laws requiring compensation for industrial accidents. About half of them have laws requiring compensation for at least some of the industrial diseases.

High lights in the history of the development of industrial hygiene in America are given in the following outline:

1837—Committee appointed by Pennsylvania to investigate child labor.
1875—First federal statute relating to labor was passed.
1877—Massachusetts gave inspectors the right to enter into factories.
1884—Federal Bureau of Labor was created.
1895—The first nurses were employed by an industrial organization.
1906—National Committee on Child Labor and the American Association of Labor Legislation were organized.
1910—The first clinic for occupational diseases was established, first American Congress on Industrial Diseases was held, and the United States Bureau of Mines was created.
1911—As noted in paragraphs above, the first American laws for compulsory reporting of occupational diseases were passed by California and New York, and certain occupational diseases were made compensable by New Jersey.
1912—The use of white phosphorus matches was abolished by the establishment of a prohibitive tax, the Children's Bureau in the Department of Labor was created, and the congress authorized the appointment of a Commission on Industrial Relations.
1913—The National Safety Council was organized. The Public Health Service and the Bureau of Mines did the first study of the health of workers in dusty trades.
1914—In the Public Health Service the office of Industrial Hygiene and Sanitation was organized as was the Section on Industrial Hygiene of the American Public Health Association.
1915—The first symposium on Industrial Hygiene and Medicine was held at the meeting of the American Medical Association.
1916—The American Association of Industrial Physicians and Surgeons was organized.
1919—The Journal of Industrial Hygiene was established and Wisconsin passed the first clear cut law making occupational diseases compensable.
1920—National Safety Code for protection of heads and eyes of industrial workers was published.
1921—The Safety Code for Foundries was approved and the reporting of morbidity among industrial workers was begun by the U.S.P.H.S.
1922—Committee on Benzol was established by National Safety Council.
1926—Committee on Industrial Medicine and Traumatic Surgery was appointed by the American College of Surgeons and this same year the College formulated and adopted the Minimum Standard for Medical Service in Industry.
1929—Safe limits of dust concentration in granite cutting plants were established.
1932—The publication of the magazine of Industrial Medicine was begun.
1934—Adoption of Safety Code for Sanitation of Factories; Federal Conference on Labor Legislation; U. S. joined International Labor Office.
1937—A.M.A. established Council on Industrial Health.
1939—First Congress on Industrial Health was held by A.M.A.'s Council on Industrial Health.

1940—President Roosevelt created Office of Defense, Health and Welfare Service, which appointed Committee on Health and Medicine with Sub-Committee on Industrial Health and Medicine. Dept. of Labor appointed National Committee for the Conservation of Manpower in Defense Industries.

1942—American Association of Industrial Nurses was organized.

All of the above have had their place in the development of the modern industrial medical and health hygiene programs. Some of the actions and developments, however, were much more important than others. In this important group would be the Federal and State legislation requiring employers to pay for accidents and occupational diseases, which did more through bureaus and divisions of government set up by this legislation than other factors toward improving working conditions and reducing industrial sicknesses. Much, however, was accomplished by a number of voluntary organizations and scientific and professional associations. Foremost in this group promoting industrial health have been the National Safety Council, the American Association of Industrial Physicians and Surgeons and the American Industrial Hygiene Association. These last two groups carried the banner for industrial health when many doubters and opposers stood on the side lines. The high standards of industrial medicine and hygiene were made possible by the character and professional strength of the men making up these two organizations.

More recently several professional organizations have had a leading hand in carrying forward this program. One of these is The American College of Surgeons which organized its Committee on Industrial Medicine and Traumatic Surgery in 1926. This committee has done excellent work in setting minimum standards for medical services in industry, in surveying the medical departments of industrial plants and accrediting those that measure up to their standards. Another is the Council on Industrial Health formed by the American Medical Association in 1937. This Council has had a strong influence on the continued development in industrial medicine through its Annual Congress on Industrial Health, through its exhibits, and through influencing the state and local medical societies to appoint committees and helping them develop programs on industrial medicine and health. Post-graduate and under-graduate industrial medical education are also being stressed by this Council. In the hands of these associations and their councils and committees, industrial medicine should make rapid strides forward in the immediate future.

More about organized medicine's part in this program is given in Chapter VII. Of the governmental bureaus, the Division of Industrial Hygiene, National Institute of Health, Bethesda, Maryland, has at the present time the most widely diversified program and has a large group of

specially qualified personnel working on the problems of modern industrial medicine and hygiene. The work of this group is described more fully in Chapter VI.

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CHAPTER II

METHODS EMPLOYED IN THE APPRAISAL AND CONTROL OF INDUSTRIAL HEALTH HAZARDS[1]

J. J. Bloomfield

It is generally conceded that in order to obtain practical results in industrial hygiene, we need the combined efforts of the physician, the engineer, the chemist, and the nurse. Basically, we can divide our mode of attack on industrial hygiene problems into two parts: First, we must attack those problems concerned with the hygiene of the individual, and second, those dealing with the environment in which the individual works and lives. The first function comes within the scope of the medical sciences and has been treated adequately in the other chapters of this book. The second function deals with engineering practices and forms the basis for the present discussion.

Under medical sciences would come the allied professions—nursing and the various medical specialties, such as pathology and physiology. Engineering services would include, in addition to engineering, the work of other public health personnel, such as chemists, bacteriologists, and biometricians.

In other words, the functions coming within the province of the medical department are concerned with the individual, while the functions with which the engineer concerns himself deal with the environment.

Insofar as the working environment is concerned, it is within the province of the medical department to determine the existence of such diseases as may be due to the working environment, while on the basis of the physician's findings the engineer is in a position to learn what unhealthful conditions should be investigated and where control measures need to be initiated. It is essential therefore that the various professions clearly understand the functions of each, and approach the solution of the industrial hygiene problem as co-workers in a joint effort, cooperating with each other to the fullest extent.

Ill health and premature death have been associated with the nature of man's livelihood from time immemorial. Studies in recent years have clearly indicated that the health of workers engaged in industry can be affected by the conditions, materials, and processes of work. For these

[1] Much of the material in this chapter has appeared in the Industrial Medicine Number of the July 1942 issue of the Medical Clinics of North America. Credit is due to W. B. Saunders Company for permission to reprint this material.

reasons, important functions in industrial hygiene are the study of the workroom environment and its effect on health, and the subsequent development of methods for the control of environmental hazards.

It is realized, of course, that in ordinary times the worker spends only approximately one-third of his day at his place of employment. His health can therefore also be influenced by his home and recreational environment. These latter conditions may also bear an important relationship to the so-called nonoccupational diseases, which are a major cause of time lost from work. For this reason, attention must also be given to factors outside the workroom which might have a bearing on a worker's health and efficiency. The present chapter will be limited to a discussion of the working environment only.

If we examine the literature on industrial hygiene, we are confronted with the fact that methods employed in the study of the effect of industrial health hazards differ but little from those used in investigations of other phases of public health. This is not strange, since in the studies of industrial diseases it is essential, as in communicable disease investigations, to determine the etiology, pathology, symptomatology, and the application of measures to prevent or control the disease. One may therefore employ the term "industrial epidemiology" when referring to the methods used for the investigation of diseases occurring in industry or among industrial workers, and the subsequent determination of methods of prevention or control of such diseases.

It is the purpose of the present discussion to treat some of the methods which are employed in those phases of industrial epidemiology dealing with the workroom environment.

THE STUDY OF THE WORKROOM ENVIRONMENT

The Reconnaissance Survey

One of the functions of an engineer in the field of industrial hygiene is the study of the workroom environment in an effort to determine any relationship between that environment and its effect on the health of the worker. In all such investigations there are certain preliminary steps of fundamental importance which must be undertaken in order to serve as a guide in the more detailed studies which may be indicated. These preliminary steps are the basis for the reconnaissance survey and consist of the sanitary appraisal and the occupational analysis of the workroom and its inhabitants (1).

The sanitary survey of the workroom consists in noting items of a general sanitary and hygienic nature, such as provisions for ventilation, illumination, fire protection, accident protection, exposure to specific poisons, such as dusts, fumes, vapors, and gases, fatigue, and so on. In other

words, sanitary survey yields information concerning the presence of various health hazards and serves as a guide in determining which hazards require further detailed study in the nature of actual quantitative determinations. One should look upon this type of survey as a listing of the facilities afforded the worker while in the industrial environment and may be likened to the inventory of materials in stock which a business establishment usually undergoes periodically.

The occupational analysis, which is also a part of this inventory or reconnaissance survey, permits one to learn of the activities involved, the particular hazards associated with each occupation, and the number of persons in each occupation.

In order to assist the engineer in the conduct of such reconnaissance surveys, certain forms are recommended. The forms illustrated have been developed as the result of experience during the past two decades in numerous investigations of industrial establishments throughout the United States. It has been found that in nearly all instances the filling out of these forms has proved a valuable guide and starting point in the subsequent studies of the workroom environment.

The first form lists data of a general nature. It covers items of a sanitary character in order to provide information on housekeeping; provision of certain personal services, such as cloak room, locker rooms, showers, and toilets; and information on various control measures which may be in use, such as ventilation and safety devices. Space is also provided for the listing of exposures to certain hazards arising from fumes, gases, dusts, and specific poisons. The second form deals primarily with the individual occupation, providing space for the designation of the occupation, the number of persons involved, the nature of the work itself, and the raw materials and by-products associated with each occupation. Space is also provided for noting control measures associated with each exposure and individual occupation.

In practice, the reconnaissance survey consists of carefully filling out the two inspection forms and jotting down any additional notes on items which may not be provided for in the forms. Under certain conditions, such as may exist in a coal mine or a cement mill, some of the items listed on these forms may obviously be omitted. After filling out survey forms for each workroom in an entire plant, a detailed analysis of the data contained in the forms is then in order. It is such an analysis that enables one to furnish a complete picture of the hygienic conditions in each of the workrooms studied and in the plant as a whole. Perhaps one typical illustration from actual experience will demonstrate the value of a reconnaissance survey of an industrial establishment.

The Division of Industrial Hygiene of the National Institute of Health

[FORM 1]

INDUSTRIAL HYGIENE SURVEY
PLANT SURVEY

Page No..... Date.....
Name of plant..... Industry Code and No.....

1. GENERAL
Department (room)..... Type of Building.....
Building No..... Location.....
Operations conducted in room.....
..... Shifts worked.....
General impression as to crowding.....
.....

2. SANITARY DATA

Dimensional		Cleaning services	
Floor area	ft.²	Times per day	
Net floor space	%	Sweeping dry	
Height of room	ft.	Sweeping wet	
Area windows	ft.²	Vacuum	
Ratio windows to floor area		Washing	
Ft.²/cap		Refuse disposal	
		Cuspidor service	

Personal Services

	M	F
Cloak rooms		
Lockers provided		
No. clothing changes provided per week		
Washing facilities: Type		
No. workers per wash faucet		
No. workers per shower		
Drinking water: Type		

Toilet facilities
Type and No..... Illumination Vent. Cond.
Male
Female

3. VENTILATION
Natural..... Type.....
Artificial..... Type & No.....
.....

4. ILLUMINATION
Natural..... General impression.....
Condition of windows.....
Artificial..... General impression.....
Type & No..... Condition.....
Shadow or glare.....

[FORM 1—Continued]

5. SAFETY HAZARDS
.....
.....
.....

6. FUMES AND GASES
.....
.....
.....

7. DUSTY PROCESSES
.....
.....
.....

8. SPECIFIC POISONS
.....
.....
.....

9. EXPOSURES TO ABNORMAL TEMPERATURES, DAMPNESS, RADIATION, NOISE, ETC.
.....
.....
.....

has been receiving reports from industrial sick-benefit organizations since 1922 (2). A study of these reports from the steel industry showed consistently higher incidence rates for pneumonia in all forms than employees of other industries. For the period 1922 to 1928, inclusive, the pneumonia case rate in the steel industry was nearly 70 per cent above the rate in the reporting public utilities, and nearly 50 per cent higher than all other reporting industries as a group. A five-year inquiry into the causes of high pneumonia rates among iron and steel workers in a representative mill disclosed the fact that the largest number of cases occurred in certain departments, such as in the blast furnace and open-hearth steel mills. When one realizes, however, that these departments contain anywhere from 60 to 100 different occupations, the task of a preventive program is almost a hopeless one, unless definite information is obtained concerning such important items as (*a*) the number of persons in each occupation, (*b*) the activities associated with each occupation, (*c*) the health hazards associated with each occupation, and (*d*) the incidence of pneumonia for each occupa-

[Form 2]

INDUSTRIAL HYGIENE SURVEY
WORKROOM SURVEY DATA

Page.....of.....

Name of Plant..... Industry Code and No.....

Department..... D/W..... S/D..... Workroom No.....

Informant's name..... Surveyed by..... Date.....

Occupation	Number of persons			Nature of job	Raw materials and byproducts	Control Measures										Remarks
	M	F	T			Pos. Ven	Neg. Ven	Loc. Exh.	Enc. Proc.	Wet Meth.	Gas Mask	Respirat.	Pres. Hel.	Pro. Clo.	Other	
Total brought forward																
Total																

tion. Such information may be readily obtained from a reconnaissance survey.

For example, in the reconnaissance survey made in this plant, it was found that the most important exposures associated with the various occupations were heat with wide changes in temperature, gases (sulfur

TABLE 1.—*Frequency of Pneumonia according to Occupation and in Relation to the Nature of Industrial Exposure Involved in the Blast-Furnace Department, 1924–28*

Sections and occupations	Nature and extent of industrial exposure[1]					Annual number of cases of pneumonia per 1,000 men	Number of cases of pneumonia		Approximate number of years of life observed
	Heat with wide changes in temperatue	Strenuous work	Outdoor work in all kinds of weather	Gases and smoke	Dust		Actual	Expected[2]	
All sections						14.0	36	10	2,578
Stacks and stoves (casting section)						27.2	17	2	624
Keeper	**	**	*	**	*	8.3	1	0	120
First and second helpers	**	**	*	**	*	41.5	12	1	289
Blowers	*	0	*	*	0	0	0	0	52
Hot-blast men	*	*	**	**	*	16.7	2	1	120
Stove cleaners	*	*	**	**	**	46.5	2	0	43
General labor section and car-dumper laborers	0	0	**	0	0	30.7	15	2	489
All other sections[3]	*	*	*	*	*	2.7	4	6	1,465

[1] Symbols for extent of exposure are as follows: 0, no exposure; *, slight or occasional exposure; **, heavy exposure.

[2] Number expected from the rate per 1,000 men in "All other departments."

[3] 8 per cent of the men heavily exposed, 23 per cent slightly or occasionally exposed to heat with wide temperature changes. To strenuous work no one was heavily exposed, and only 5 per cent had occasionally to work strenuously. To outdoor work in all kinds of weather about 8 per cent of the men were heavily exposed, 80 per cent slightly or occasionally exposed. To gases and smoke about 5 per cent were heavily and 25 per cent slightly or occasionally exposed. About 1 per cent of the men were heavily exposed to dust, and about 55 per cent slightly or occasionally exposed.

dioxide, hydrogen sulfide, and carbon monoxide), siliceous dusts, strenuous work, and outdoor labor in all kinds of weather. The survey enabled one to note these exposures for those occupations in which they occurred. Table 1 presents the frequency of pneumonia according to occupation in the blast furnace department, in relation to the nature of the exposure,

during the period of 1924–1928. It is obvious that the highest pneumonia rates occurred among those occupations exposed to one or more of the potential hazards cited. The actual number of cases for those occupations not associated with these five exposures (all other sections) were found to be even less than the expected cases of pneumonia for such workers. The reconnaissance survey indicated that, in the blast furnace department, attention should be centered on the occupations in the casting and general labor sections, in an effort to determine the degree of exposure to gases, dusts, extreme temperature changes, and so on. Such studies are carried out by the engineer and chemist, whose task it is to determine the extent of the occupational exposure to the materials and conditions enumerated. Thus, the engineer is concentrating on the important factors and omitting the unimportant. In this particular study, it was necessary to evaluate the working environment for but a few occupations of the more than 60 in the blast furnace department. It was these few occupations that accounted for the high pneumonia incidence for the entire department and once the occupational exposure factor had been evaluated, the engineer was in a position to initiate control measures for the minimization or the elimination of the hazards demonstrated to be deleterious to health.

The Detailed Survey

In determining a worker's exposure to materials or conditions incident to his employment, it is necessary to have precise data on such exposures. In some cases the difference between a hazardous and a non-hazardous condition may depend entirely on whether the worker is exposed continuously to concentrations of materials bordering on the threshold limit. For example, in the case of exposure to certain lead compounds, for which the threshold limit has been set as 1.5 milligrams per 10 cubic meters of air, it is very important to know whether the worker is inhaling lead dust approximately above or below this limit. Quite often a difference of a milligram or so may spell the difference between a safe or an unsafe condition.

Detailed appraisals of exposure may be said to serve a three-fold purpose. First, they enable one to determine the extent of a hazard. This is accomplished by obtaining occupational exposures, by precise methods, to the toxic materials or conditions under consideration. Today, the engineer and chemist have at their disposal delicate instruments and methods of analysis unprecedented in the history of industrial hygiene and our knowledge concerning such matters is increasing from day to day. The guesswork as to actual exposures has been fairly well taken out of industrial hygiene.

A second purpose served by the detailed study is the fact that if clinical

investigations are made concurrently with environmental studies, the findings on occupational exposure may indicate the permissible amounts of the toxic materials which may be tolerated with safety.

And, finally, the third purpose served by this type of study deals with the control of the hazard. In other words, one is in a position to determine the efficiency of any device which may have been introduced for the minimization or elimination of the hazard. Some examples illustrating each of the three purposes served by the detailed study follow.

Extent of Hazard.—In a study of lead poisoning among storage battery workers, it was important to determine the relationship between the amount of lead dust inhaled by the men and the incidence and severity of plumbism. Such a study is valuable in that it may indicate the maximum amount of lead which may be inhaled with impunity.

Table 2 shows the fundamental correlation between the lead dust in the air and the rate of plumbism in the major departments of the plant in which a study was made (3). It is evident that a close correlation exists between

TABLE 2.—*Lead Exposure and Maximum Monthly Rate of Initial Compensation Cases for Plumbism*

Department	Milligrams of lead per 10 cubic meters of air	Maximum monthly rate (per 100)
Mixing	120	44
Pasting	50	12
Burning	5.7	4.4
Casting	1.2	.18

the lead exposure of workers in different departments and the risk of developing a case of lead poisoning.

Correlation with Clinical Data.—Figure 1 illustrates the relationship between lead dust exposure, years of exposure, and the percentage of workers diagnosed as having early plumbism (4). It is evident that 1.5 milligrams of lead dust per 10 cubic meters of air, except for very prolonged exposure, is the limit of safety under the conditions encountered in these studies. This important finding is of great value to the engineer, since it gives him a basis upon which to develop protective devices in the way of exhaust ventilation, respiratory protection, good housekeeping, and so on.

Efficacy of Control.—The detailed survey as employed in studying the efficiency of various methods employed for the control of a hazard will be treated in more detail in the next section, which deals with the control of industrial health hazards. It is desired, however, in closing this portion of the discussion, to point out that the engineering problems in industrial hygiene are constantly increasing in number, especially now when many

new chemicals and processes are being introduced into our industries. Our knowledge of these substances, as to their action on the body, is constantly being augmented by the work of toxicologists and by field studies of the type described herein. It is the engineer's task, once this knowledge is available, to devise ways and means for controlling these injurious materials and conditions, a discussion of which follows.

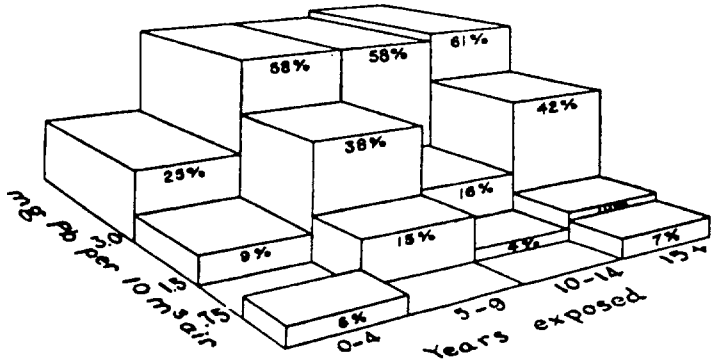

FIG. 1. Percentage of workers with early plumbism. The percentages of storage battery workers in each of 16 exposure groups diagnosed as cases of early plumbism are represented by the height of blocks. Thus, of the workers exposed more than 15 years to atmospheric lead concentrations in excess of 3 mg. Pb per 10 m³ of air, 61 per cent were found to have early plumbism. Data taken from table.

THE CONTROL OF INDUSTRIAL HEALTH HAZARDS

The control of industrial health hazards is also a function of the medical and engineering departments. The physician and his co-workers recognize the existence of diseases due to the workroom environment and exercise medical supervision and initiate studies designed to eradicate the dangerous conditions. The engineer and his co-workers determine the extent of the hazard, and, armed with a knowledge of the toxicity of the material involved, are in a position to consider methods and equipment for the control of the hazard.

No set rules may be established for the mechanical protection to be instituted in an attempt to control an industrial health hazard. Specific conditions encountered in a plant will determine the type of protection to be employed. In general, however, there are five methods which may be attempted in the minimization of an industrial exposure. These are: (*a*) Substitution of a non-toxic material for the toxic one, (*b*) isolation of the harmful process, (*c*) wet methods in the case of some dusty operations, (*d*) local exhaust ventilation, and (*e*) respiratory protection. In many instances it may be necessary to employ a combination of the above methods in the control of a single exposure.

Substitution.—The protection of workers against certain dusts known

to be toxic may at times be accomplished by the substitution of a non-toxic material for the toxic one. One example of such a procedure is the use of a metallic or other type of artificial abrasive for sand in the sandblasting process in those operations in which it is not necessary to use sand, a substance high in quartz content. The data shown in table 3 indicate clearly the lowering in dust concentration when a steel abrasive is employed instead of sand in a sandblasting room (5).

TABLE 3.—*Showing Reduction in Concentration and Quartz Content of Dust in Sandblast Rooms with the Substitution of Steel for Sand Abrasive*

Type of abrasive	Average dust concentration in millions of particles per cu. ft.	Percentage of quartz
Sand	969	42–98
Steel	155	3

Another example of substitution is one recently employed in the hatting industry. For more than 100 years mercury had been employed in the carroting solution which was applied to rabbit fur utilized for the production of felt hats. Certain operations in the preparation of the hatters' fur and in the subsequent manufacture of the hat itself entailed exposure to mercury vapor and dust with accompanying mercurialism among the workers. Recently the hatting industry itself developed a non-mercurial carroting solution which was adopted by the industry, so that mercurialism in the future will be completely eradicated in this industry (6).

Isolation.—The mechanical enclosure or isolation of certain operations also serves to protect the worker. In the case of dust exposure, we have an excellent illustration of this type of protection by the use of the modern sandblast barrel used in the cleaning of small objects. Table 4 illustrates the practical elimination of dust exposure in certain sandblasting operations in which well enclosed and isolated operations are contrasted with those not offering this sort of protection.

TABLE 4.—*Dust Concentrations for Sandblast Installations Regarded by Manufacturers as "Ideal" in Comparison with Other Equipment*

	Average dust count in millions of particles per cubic foot		
	Barrels	Tables	Cabinets
Ideal	1.7	0.2	1.9
Other	29.0	22.0	38.0

Wet Methods.—In the case of dust, it is possible to protect workers by the substitution of wet for dry processes. This method is illustrated by the results shown in table 5.

TABLE 5.—*Contrasting Wet and Dry Methods of Rock Drilling and Loading*

Processes	Number of samples	Average dust count in millions of particles per cubic foot	
		Dry	Wet
Drilling	23	568	33
Loading	10	636	32

More recent studies of rock drilling operations have shown that wet methods employed in conjunction with general exhaust ventilation may often result in even lower dust concentrations than those shown in this table (7).

Local Exhaust Ventilation.—Perhaps the most universally used method of control of certain workroom conditions which are harmful to health is local exhaust ventilation. This method aims to remove dusts, fumes, vapors, and gases at their source. So much depends upon the correct design and construction of hoods and exhaust systems that they should be laid out and maintained with great care. The past few years have witnessed the gradual accumulation of a tremendous amount of basic engineering data on this type of control. Data pertaining to quantity of air necessary at hoods to control hazards or nuisances are rapidly accumulating.

For example, it has been shown that spray painting booths require velocities of 100 to 200 feet per minute at the openings to reduce effectively the presence of vapors to safe limits. For laterally exhausted chromium plating tanks, specific air velocities and air volumes have been developed and are now embodied in a national standard recently promulgated by the American Standards Association (8). In connection with granite-cutting, special hoods have been designed for the control of the hazard in this industry (9). In certain instances, down-draft ventilation has been found to be most effective. Such practice materially reduces the air velocities required for control and utilizes a natural tendency for heavy dust to settle (10).

The scope of the present article does not permit adequate treatment of this particular method of control, although when properly considered it is an effective one. Table 6 presents some data concerning required air volumes and other pertinent information regarding local exhaust ventilation.

Respiratory Protection.—It is generally agreed that in the control of exposures to air-borne toxic materials, primary consideration should be given to procedures for preventing excessive contamination of the air in

the breathing zone. However, there will always be situations where these procedures will be inapplicable, impracticable, or at times not effective. For these situations, personal respiratory protection will be required, either as a primary means of protection or as an adjunct to other preventive procedures. In recent years, great strides have been made in the improvement of respiratory protective devices and at present the United States Bureau of Mines is approving such devices after rigid testing (11). Such an approval system has stimulated further research on the part of the manufacturers handling these devices, which has resulted in a better prod-

TABLE 6.—*Minimum Air Velocities Required to Capture Certain Industrial Dusts and Vapors*

Industry	Process	Required air velocity		Criterion
		At point of origin	At face of hood	
Granite cutting	Hand pneumatic tool	200 FPM		Reduced concentration to safe level
	Surfacing machine	1500 FPM		Reduced concentration to safe level
	All tools		1500	Reduced concentration to safe level
Grain elevators	Elevator boot and head, garner		500	Visual test
Paint spraying	Spraying booth		50–200	Reduced concentration to safe level
Sand pulverising	Bagging machine	400 FPM		Reduced concentration to safe level
Quarrying and mining	Horizontal drilling with Kelley trap	60*		Reduced concentration to safe level
	Vertical drilling with Kelley trap	200*		Reduced concentration to safe level
Electroplating	Chromium plating	50**	1500	Reduced concentration to safe level
	Steam and acid tanks	75–100		Reduced concentration to safe level
Hatters' fur	Brushing	200*		Usual practice
	Cutting machines	380*		Usual practice
	Blowers	2000*		Usual practice
Electric welding	Welding	200		Visual test
Metal spraying***	Lead	200		Effective removal of all fumes
	Zinc	125		

* Cubic feet per minute.
** Cubic feet per minute per foot of slot (common practice).
*** At opening of booth.

uct. The reader is referred to an excellent treatise on respiratory protective devices which was prepared by the Subcommittee on Personal Respiratory Protective Devices of the Committee on the Prevention of Silicosis through Engineering Control of the National Silicosis Conference (12).

It is also not possible to include in the present discussion the important subject of protective clothing, such as goggles, aprons, boots, rubber gloves, and especially the newer type of protective clothing made from plastics, nor is it practicable to go into a discussion of the subject of pro-

tective ointments. The reader is referred to the excellent chapter in this book (Chapter XXI) on this subject by Dr. Louis Schwartz.

SUMMARY

Today we are witnessing vigorous action on the part of many official organizations in the development of codes and other regulations dealing with the removal of noxious air-borne materials and other environmental hazards in industry. Nonofficial agencies, such as the American Standards Association, and various medical and engineering organizations are collaborating with State and Federal agencies and industry toward the development of such codes on a scientific basis. One of the great needs today to aid in the development of such codes is factual data based on studies of existing installations.

TABLE 7.—*Exposure of Hatters' Fur Workers to Mercury Vapor and Treated Fur Dust under Controlled and Uncontrolled Conditions*

Occupation	Total mercury exposure in milligrams per 10 cubic meters		Average air flow, C.F.M.	Method of control
	Uncontrolled	Controlled		
Drummers	2.5	0.6		Segregation
Clippers	1.5	0.7		Segregation
Brushers	3.1	1.2	300	Local exhaust ventilation
Cutters	4.0	1.8	383	Local exhaust ventilation
Sorters*	3.8	1.7	383	Local exhaust ventilation
Blowers	4.6	0.7	2,000	Local exhaust ventilation
Pilers	5.4	Trace		Good natural ventilation
Storage workers and shippers	7.2	Trace		Good natural ventilation

* Depend on exhausted cutters.

It is well-known that in many industries health hazards are being effectively controlled. The engineer and the clinician should systematically evaluate the efficiency of the various methods employed so that the data would be available and useful in the preparation of good practice codes.

Perhaps it would be proper at this point to give an example of the usefulness of a study of existing installations. In a survey of the mercury hazard in the hatters' fur cutting industry, the engineers were in a position to evaluate various control measures in vogue in various representative plants in the industry (13). Table 7 indicates the exposure to mercury dust and vapor of some of the workers in this industry under controlled and uncontrolled working conditions. It is apparent that where some measure of control is practiced by such methods as segregation or local exhaust ventilation, a material reduction in the exposure to mercury is

effected. Although no one plant was found to have all of the control measures in effect, as shown in the table, methods were found in representative plants of the industry showing that the mercury exposure for different occupations could be controlled by existing practices. The engineer, in conducting a study of this type in representative plants, is in a position to present to the industry as a whole information showing how certain hazards can be and are being effectively controlled.

With further reference to basic standards for the control of hazards in industry, two approaches may be employed. Standards can be based on clinical data, that is, on definite knowledge which shows that exposure to more than a certain amount of a toxic material will involve injury to health. Such knowledge then can be used in engineering design in order to reduce the exposure within the so-called threshold limit. Although our knowledge concerning the toxicity of materials is rapidly becoming more and more abundant, there are still many substances for which we have no basic information at this time concerning their toxicological properties. In such cases it is suggested that standards could be employed which are based on results which can be obtained by good engineering practices. Quite often these engineering practices are found to yield conditions far better than those which might be based on our present knowledge of the toxicity of a material. One must bear in mind that our present ideas concerning the toxic limits of certain substances are subject to change as our methods of diagnosis and analysis improve. Chapter XIII describes in detail recently developed techniques for the early discovery of indications of toxic exposure.

In closing, it is desired to emphasize that studies of the industrial environment necessitate numerous laboratory examinations of a clinical, physical, and chemical nature. These call for highly trained chemists and biochemists. Today, with the added use of chemicals in many of our processes, there is a greater need for data concerning the toxic effects of these substances. The proper procedure is a study of these new materials or processes on a small scale prior to their widespread use in industry. This practice, which is now being employed in some of our more progressive industries, calls for a close collaboration between the production and development departments in the plant, and the industrial health maintenance service.

Once the problem in industry has been evaluated and everything possible has been done to control unhealthful conditions, it will still be essential for the medical, nursing, engineering, and chemical personnel to maintain close vigilance, in order that safe conditions may be maintained and improved upon. Constant study of the workroom environment is essential to ascertain whether certain measures are really effective. Such practice

in cooperation with the work of the physician and the nurse appears to afford the best means of controlling industrial health hazards, and their attendant loss of valuable time from work.

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CHAPTER VII

THE INDUSTRIAL HEALTH PROGRAM OF THE AMERICAN MEDICAL ASSOCIATION

C. M. Peterson, M.D.

Many large corporations in this country and elsewhere have learned by experience through extended periods of time that medical supervision over workers, processes and environment is good business. Many physicians have been associated with such activity in manufacturing operations of every description. They have amply demonstrated that an industrial medical service can be administered to the very great advantage of worker and employer alike, and at the same time in conformity with existing ethical and scientific standards. Concisely expressed, the objectives of a properly organized industrial medical service have proved to be:

1. The prevention of disease or injury in industry by the establishment of proper control over working conditions.
2. The restoration to health and earning capacity as promptly as possible after industrial injury or disease.
3. The conservation of the health of workmen through physical supervision and education.

It has been apparent for some time that smaller industrial organizations have been denied the advantages of industrial hygiene, preventive industrial medicine and health conservation mainly through lack of properly trained professional personnel and the absence of a plan of industrial medical service which could receive wide acceptance by small employers. In recognition of these two material facts, the industrial health program of the American Medical Association is intended to:

1. Acquaint physicians with the special character of industrial medicine, surgery and hygiene and the necessity for the enforcement of standards defining scope and professional competence.
2. Convince employer and employee alike that medical service in industry merits support if soundly organized, economically, scientifically and ethically and that to produce lasting benefits it must be satisfactory both to those who provide and to those who receive the service.

In accordance with this view, improved standards of medical accomplishment in industry must stem directly from better industrial medical education and efforts at self-improvement and self-discipline administered by the physician's own organizations. To accomplish this purpose the Coun-

cil on Industrial Health of the American Medical Association was established.

It was not long until the Council realized that no plan of industrial health training would be successful without full cooperation from

1. Medical and professional schools.
2. State and county medical societies.
3. Medical specialty groups.

The Council has on several occasions communicated with the administrative officers of medical and public health schools with a view towards stimulating better organization of industrial health teaching, both before and after graduation. A program for improved organization of teaching has been prepared in conjunction with the Committee on Education of the American Association of Industrial Physicians and Surgeons. Improvement has been gradual, but in a number of medical centers very substantial contributions are being made in the field of industrial health teaching and it seems safe to predict that much greater effort will occur in other institutions located in highly concentrated and important industrial areas.

At the invitation of the Council, 45 states and the District of Columbia have organized committees on industrial health. Each of these agencies acts independently in accordance with local situations and requirements. However, in the interests of concerted and uniform action, the following broad-scale program has been recommended:

1. To train industry and labor to the value of industrial health conservation.
2. To develop a clear understanding of the proper scope and functions of industrial medicine and to clarify relationships between private and industrial practice.
3. To keep the medical profession informed about all accepted methods for reducing the frequency and severity of industrially induced disability.
4. To evaluate medical relations under workmen's compensation.
5. To scrutinize all legislation affecting the health of industrial workers.
6. To improve relationships between medicine and insurance.
7. To establish working relationships with all agencies in the state interested in industrial health.
8. To arrange for the adoption of similar activities through cooperating committees in the medical societies of the industrial counties.

In recognition of the major medical interests involved, it has been felt that each state committee on industrial health should contain representation from:

1. Private practice.
2. Industrial medical practice.
3. Medical representation if such exists from each of the following:

a. State bureau of industrial hygiene.
b. State workmen's compensation agency.
c. Medical faculties in the state.
d. Casualty insurance company.

A bulletin has been developed by the Council on Industrial Health to promote inter-change of ideas between committees of the state societies and as a means of estimating progress. In much the same way, representatives of the state committees attend the Annual Congress on Industrial Health in Chicago each January to report developments and advances.

Successful organization of cooperating agencies in state medical societies has suggested that similar objectives and methods be introduced into the county medical societies as a means of mobilizing community resources for general or special industrial health activity. Such committees can establish dependable information about local or regional health exposures in industry and use this knowledge as a basis for early recognition and control and to promote in other ways the physical welfare of workers. There should be many opportunities to provide better professional training in medical society meetings and symposia. The community at large can be instructed in the objectives of industrial health and made aware of the favorable effect of industrial health procedure. Certainly, a county medical society committee enables the special technics of industrial practice to be integrated into the general pattern of community medical service and it has already been demonstrated that helpful working relationships can be promptly established with such allied professional groups as the industrial hygienists, the safety engineers and the industrial nurses.

Following the example set originally by the Section on Dermatology and Syphilology, most of the Sections in the Scientific Assembly of the American Medical Association representing all major specialty groups affected by industry have formed committees to initiate programs which will be of benefit in industrial relationships encountered by specialists and to act in an advisory capacity to the Council on Industrial Health. Already these committees have contributed in a very substantial way to our knowledge of occupational medicine and will, it is confidently believed, steadily improve methods of attack on specific problems related to improved case management, disability evaluation and rehabilitation.

In addition to improved training and better national, state and local organization for proper consideration of industrial health relations, the Council has inaugurated and maintained other services calculated to advance standards and improve results. Work is going forward in the standardization and classification of nomenclature in industrial health, in the determination of better clinical and preventive methods, in medical ad-

ministration under workmen's compensation, in improved vital statistics relating to occupational morbidity and mortality, in clearing house service and in the general field of socio-economic development.

All these activities indicate that the problems of industrial health, whether in hygiene, medicine, surgery or whatnot are beginning to receive genuine consideration throughout the profession. There is good reason to believe that as industrial health continues to develop, the responsibilities of the medical profession will be well recognized and ably represented.

Chapter XXII

PNEUMOCONIOSIS (DUST DISEASES OF THE LUNG)

George Zur Williams, M.D.

I. THE PROBLEM

Early History

It is not the purpose of this chapter to discuss the early history of pneumoconiosis at length nor to consider in detail the many disputed hypotheses and results of experimental investigations concerning the theories of production and prevention of pneumoconiosis. It will be the purpose of these paragraphs to set forth briefly and as concisely as possible the various practical and proven concepts concerning the etiology, pathogenesis, pathology, clinical course, and control of the various dust diseases, particularly that due to silica (silicosis).

Dust disease of the lungs is a long recognized entity, but long before distinction from other diseases of the lungs Hippocrates mentioned symptoms of breathlessness in certain metal workers who "degenerated rapidly." Although the disease was differentiated and known to affect miners before 1899 on the Transvaal, the first commission was not appointed until 1902 to study the effects of dust on miners' lungs. In 1919 the first technical commission was appointed in Australia to use x-ray in the diagnosis of dust diseases of the lungs. Silicosis was first reported in a western state of this country in 1894 and a thorough x-ray study was made and reported by Lanza and Childs in 1917. Many reviews of the literature on the effects of dust in the lungs are complete and should be consulted by the reader who is particularly interested in the details of early work.

Dust in the Lungs

All normal lungs contain a certain amount of dust, accumulation beginning soon after birth. Many different dusts are accumulated by the lungs and stored in varying amounts in the small pulmonary lymph centers and hilar lymph nodes. In localities with large amounts of smoke in the atmosphere, children begin to show blackening of the lungs early in life due to accumulation of coal dust. Silica is found in all lungs of adults in small amounts regardless of the occupation of the person. In persons under 48 years of age 50 milligrams per cent to 200 milligrams per cent of dry weight of the lungs is found to consist of silica. The hilar lymph nodes contain from 1,000 to 3,000 milligrams per cent in individuals of from 48 to over 52 years of age. This amount of silica does not produce pathological changes

in normal persons. The silica can be found only by histo-incineration or by chemical analysis.

Definition

The pneumonoconioses are conveniently divided into various types depending upon the character of dust accumulated in the lungs. In almost all instances a varying amount of coal dust is present and, therefore, the prefix "anthraco" may usually be applied. The following definitions are taken from the National Silicosis Conference Report on Medical Control, United States Department of Labor, Bulletin 21, Part 1: "The committee on pneumonoconiosis of the industrial hygiene section of the American Public Health Association recently defined silicosis as: 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."

As will be pointed out later, considerable importance may be attached to the difference between the clinical disease, silicosis, and the discovery of moderate nodulation of the lungs caused by inhalation of silica and found incidentally upon roentgenological examination. This difference is of some importance in the disposition of individual cases with relation to control and industrial compensation.

Classification

There are pulmonary diseases due to inhalation of organic dusts which do not produce nodulation and which are infectious in nature and, therefore, are not properly included under this term. Silicosis is the one true uncomplicated nodular fibrosis which has been proven clinically and experimentally to be caused by the inhalation of dust containing high concentrations of free silica. No other dusts than those containing silica will produce true nodular fibrosis. A more diffuse fibrosis and mixtures of nodular and diffuse fibrosis are caused by other dusts, such as asbestos, coal dust, and iron ore dust. Usually, with the possible exception of asbestos, these dusts have a rather high concentration of silica and, therefore, there is yet a question concerning the true etiology of anthracosis and siderosis. Silicosis is the most important of this group of pneumoconioses, particularly from the point of view of industrial medicine and will be discussed in more detail.

Occupational Incidence

Although the incidence of disabling silicosis is not as high or grave as many reports and the news publicity in recent years would indicate, silica

dust occurs in hazardous concentrations in the air of many industrial mines and plants. Silica dust hazard occupations are listed in Occupational Disease Leaflet #9, 1936, Division of Labor Standards, United States Department of Labor, as follows:

Abrasive-powder makers	Metal grinders
Abrasive-soap makers	Pottery makers
Blasters (sand)	Pneumatic rock drillers
Brickmakers	Polishers
Coal miners	Sanders
Core makers	Stone finishers
Foundry workers	Slate quarriers
Glassmakers	Slate finishers
Glass mixers	Sand pulverizers
Granite quarriers	Tunnelers
Hard-rock miners	Vitreous enamelers

Control Problem

Of great importance to society, and particularly to industry, is the problem of the control of silicosis. In some states silicosis is now a compensable disease and due to this fact, insurance companies and compensation commissions have stimulated a widespread interest in the prevention of this disease and some control by legislation in certain states provides for prevention and compensation of this disease. Further details concerning this general problem are discussed in Chapter II.

II. THE PATHOGENESIS

Causative Agent

It has been proven beyond doubt, both clinically and experimentally, that silica (SiO_2-silicon dioxide) is the primary etiological agent in the production of a nodular fibrosis of the lungs. There is no definite evidence that non-siliceous dusts produce this characteristic fibrosis and when no history of exposure to silica dust can be obtained from a patient suspected of suffering from silicosis, careful investigation of his historical background should be made to determine exposure to any other dust which may have contained dangerous concentrations of silica without the patient's knowledge. The combined forms are termed silicates and these are of no importance in the pathogenesis of disease with the one exception of magnesium silicate (asbestos).

Silica is the most abundant constituent of the earth. It occurs commonly in both free and combined forms. The free silica occurs in the form SiO_2 and is commonly known as quartz, being the chief constituent of granite, schist, and other rocks, such as sandstone and quartzite. Many ores are deposited in veins which are composed almost entirely of quartz. Another form in which silica occurs commonly is the amorphous hydrated

type of colloidal origin abundant in diatomaceous earths. Other forms of free silica occurring in nature, but much more rare, are tridymite, cristobalite, and siliceous glass.

Action of Silica

Silica is very commonly believed to be insoluble because of its resistance to chemical reagents with the exception of hydrofluoric acid. However, it has been proven by well controlled chemical experiments that silica is relatively soluble in many agents, including water, and particularly slightly alkaline solutions of salt, when exposed to the solvent in very finely divided particles measuring only a few microns in diameter. Therefore, size of the silica dust particles is important in determining their action upon tissues of the lungs. A second factor determining the action of silica on tissue is the dose (the concentration of the dust in the inhaled air). A third factor is the effect of other dusts mixed with the silica.

It has been shown by both chemical and animal experiments that silica particles of very small size, i.e. measuring ½ micron or less in diameter, are relatively innocuous if aspirated into the lungs because they are so rapidly dissolved that most, if not all, of the silica is excreted through the kidneys in dissolved form. Recent experiments show that unusually high concentrations of silica of this small size may produce extremely toxic effects, but no nodular fibrosis is produced. Particles of silica from 10 microns in size to larger are also more or less innocuous when introduced into the lungs in high concentrations. This probably is due to the fact that these particles are too large for phagocytosis by the alveolar cells and also that their rate of solution is so slow there is no stimulation of the phagocytic activities of these cells. As a result they are swept back out into the trachea and the pharynx by the ciliary activity of bronchial and tracheal mucosal cells.

Silica particles of the sizes between ½ micron and 10 microns, and particularly those of the size from 3 to 5 microns, are the sizes found to be most injurious in producing experimental nodular fibrosis of the lungs. These sizes permit some solution with resulting "toxic" paralysis of mucosal cilia. Large numbers of experiments have been completed to determine the dust concentration required to produce this nodular fibrosis. It is generally found that dust concentrations between 5,000,000 and 10,000,000 particles per cubic foot are capable of producing nodular fibrosis in only a very small number of exposed animals or individuals, but that dust concentrations above 10,000,000 particles per cubic foot are definitely hazardous and produce a nodular fibrosis in the majority of exposed individuals. Dust concentrations below 3,000,000 particles per cubic foot are very rarely hazardous.

The third factor is that of a mixture of various other dusts. Early ex-

periments led to the conclusion that carbon dusts in the form of coal dust in hard coal miners protected the miners from silicosis because it was found that apparently hard coal miners developed less silicosis and of less severity and in fewer numbers than did other hard rock miners. However, this contention has not been proven and in other studies of hard coal miners such results are not always found. It has been found that alkaline dust when mixed with silica dust apparently renders it innocuous. Experimental evidence has been obtained explaining this effect as due to the fact that the silica dust particle carries a negative static charge. The alkaline dust particle carries a positive static charge. When negative dusts are combined with statically positive dusts, agglutination of neutralization occurs and the dusts precipitate into large clumps and fall out of the air. The concentration of silica in the dust is greatly lowered by precipitation due to this electrical agglutination and the particles of agglutinated dust become so large that solubility is decreased in the bronchial secretions and the cilia sweep them outwardly before phagocytic action can occur. Experimental proof is lacking to show that iron dust, aluminum dust, and other metallic dusts have any effect whatsoever on increasing or decreasing the injurious action of silica on lung tissues.

Related Anatomy

There are certain aspects of the anatomy of the human and animal lungs which become important factors in predisposing to, or protecting the lungs from, the action of silica. Animals possess two types of pulmonary lymph drainage systems. These are termed the "open" and the "closed" types of lymphatic drainage. The human lung, the guinea-pig lung (fig. 1a), and the pig lung have the "open" type. The rabbit, dog, rat (fig. 1b), and mouse lung are of the "closed" type. Microscopic examination of these lungs reveals large numbers of small lymphocytic nodules scattered throughout the lungs extending into the peripheral portions in the closed type. Special studies have demonstrated that lymph drainage occurs by collection in small lymphatics which traverse the lung along the vessel courses and drain through the numerous chains of lymphocytic nodules into the hilar lymph nodes at the root of the lung. On the other hand, in the open type of lymphatic system, there are no such lymphocytic filter centers in the course of lymph drainage from the periphery of the lung to the hilus. In this type foreign particles, fragments, and organisms picked up by the lymphatic system in the periphery of the lung are transported directly to the first filtering nodes which occur at the hilum.

In animals and man possessing the open type of lymphatic drainage, there occurs a significant change to the *closed* type as the result of many acute and chronic respiratory infections. In the guinea-pig it will be found

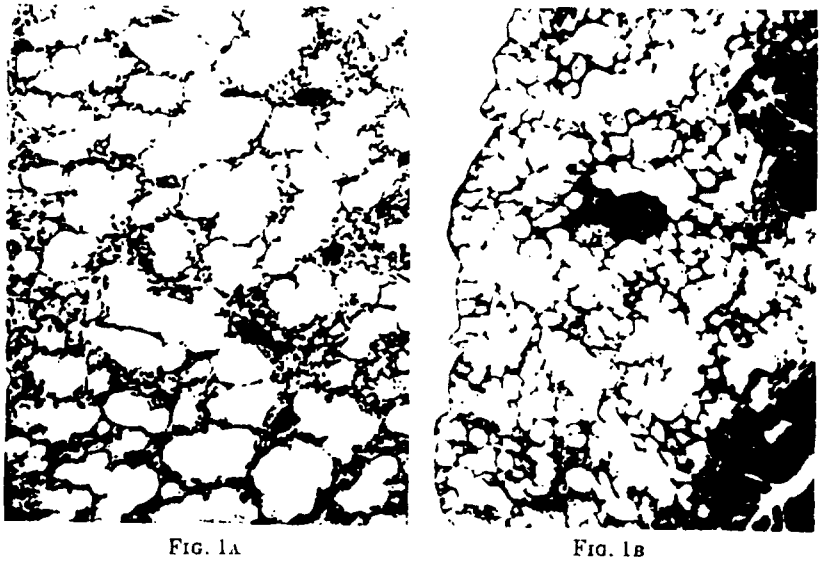

FIG. 1A FIG. 1B

FIG. 1A. (× 50) Normal guinea pig lung—"open" type lymphatic system. Note absence of lymphoid nodules in parenchyma

FIG. 1B. (× 50) Normal rat lung—"closed" type lymphatic system. Note dark lymphoid nodules adjacent to peripheral bronchioles

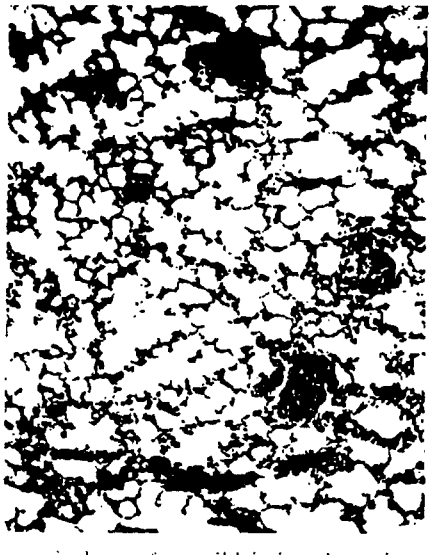

FIG. 1C. (× 50) Guinea pig lung after mild induced respiratory infection. Note proliferating lymphoid nodules in parenchyma

that following pneumonia or bronchitis, this change occurs by the enlargement of microsocpic collections of a few lymphocytes into nodules of many lymphocytes which accumulate during the infection and persist for long periods thereafter (fig. 1c). These nodules may become very large and are frequently found peripherally as far as the pleura. They occur usually at the bifurcation of the blood vessels and are so constructed that the lymph filters through the nodule. It is important for the reader to keep in mind these two types of lymphatic system and to remember the basic fact that chronic infection and inflammation will change the open lymphatic system of the human lung to the closed type when considering predisposing factors to silicosis.

Predisposing Factors

Many experimental findings have partially explained numerous puzzling problems disclosed by study of large numbers of silicosis cases. Some of the problems consist of exceptions to the general rule; for instance, individuals who were exposed for long periods of years to high concentrations of silica dust without developing silicosis, and on the other hand, patients who were exposed to lower concentrations of dust for short periods and yet have developed a rapidly progressing typical silicosis with nodular fibrosis and complicating infections. The clinical experiments and observations on large numbers of mine workers, sand blasters, etc., as well as thorough and well-controlled animal experiments have shown that there are several important predisposing factors which cause considerable variation in the type of silicosis produced and in the length and dose of exposure required to produce a disabling fibrosis. Acute and chronic pulmonary and upper respiratory infections are known to produce many changes in the lungs which may be found at post mortem examination. Chronic sinusitis with a persistent post-nasal drip, frequent colds with complicating bronchitis, pneumonia and its residual changes, bronchiectasis, and tuberculosis are among the chief contributory conditions. These inflammatory conditions are known to act in two ways. In the acute types of pulmonary infection, there is produced a focal lymphocytic accumulation in the peripheral lung fields thereby closing portions of, or all of the lymphatic system which was previously an open type. All inflammatory scars which produce extensive fibrosis along lymphatic channels will cause obstruction and thereby further modify the lymph drainage. Secondly, certain acute and more especially the chronic respiratory infections, such as low grade tuberculosis, bronchiectasis, and repeated attacks of bronchitis, will cause a loss of the ciliated epithelium of the bronchi and replacement by non-ciliated squamous cells. This loss of cilia permits the accumulation and stagnation of

secretions in the bronchi and bronchioli which in turn promotes accumulation of inhaled dusts. Certain gaseous irritants in mine gases and explosive gases have been found experimentally to paralyze cilia of the tracheal and bronchial mucous membranes. Certain experiments apparently demonstrate that dissolved silica from very small particles of dust will have a slowing or paralyzing effect on ciliary activity. Thus, the normal protective mechanism of the ciliary sweep of secretions to the pharynx is destroyed. Stagnation of dust and secretion allows the phagocytes longer periods to engorge themselves with silica particles and to traverse the alveolar walls into the lymph spaces.

Aside from the general anatomical changes and the ciliary paralysis and destruction, there is apparently a particular increased susceptibility of tuberculous patients to silicosis. This phase will be discussed more fully below.

Progressive Development

Silicosis is not a complicated disease from the point of view of pathogenesis. Although predisposing factors in various degrees of severity produce a large number of variations, the actual pathogenic cycle of inhalation of dusts with the subsequent production of fibrosis appears to be a rather clear-cut process. Inhaled silica of a size from 1 to 10 microns in diameter is phagocytosed by the alveolar cells. These accumulate in large groups in the alveoli and gradually pass through the alveolar walls into the lymph spaces. They are collected by the draining lymph and carried into the lymphatics and thence to the hilar lymph nodes. Thus, the primary accumulation of silica occurs in the hilar nodes in cases of open lymphatic system. During this transportation, the phagocytes which are engorged with large numbers of small particles ($\frac{1}{2}$ to 3 microns in diameter) seem to become "mummified" and inactive or are killed by the toxic action of the dissolving silica. These cells lose their mobility and, therefore, accumulate in groups obstructing the lymphatics. The cells disintegrate and the liberated particles of silica are again phagocytosed by fresh cells (histiocytes) which are apparently attracted to this focus. These continue migration until they in turn are also destroyed by the toxic action of the dissolving silica. This process is repeated many times until eventually the dust may reach the hilar lymph nodes.

It is obvious that in the instance of lungs in which previous inflammation has "closed" the lymphatic system, these masses of dust-laden phagocytes (dust cells) will accumulate and remain in the peripheral foci (fig. 2a). These groups stimulate the production of early fibrotic nodules (fig. 2b). In each of these foci the gradual dissolution of silica from the disintegrating cells seems to produce a toxic or stimulating factor which causes further

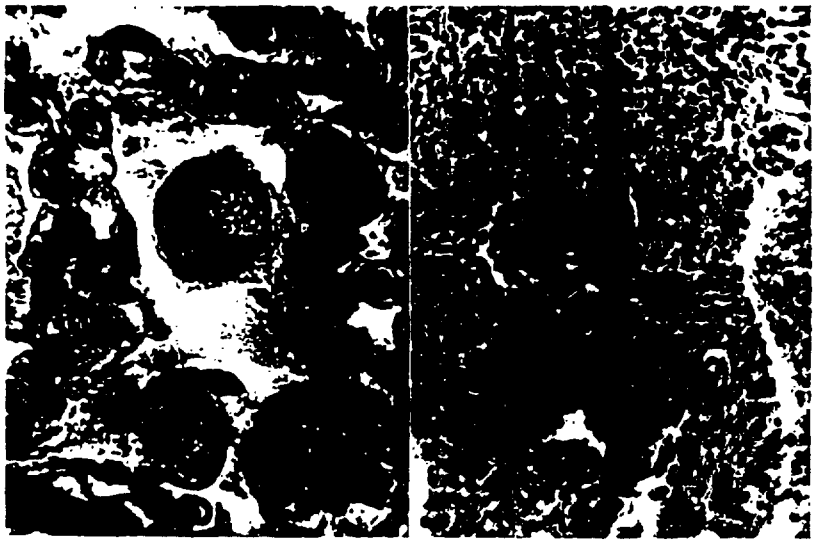

FIG. 2A FIG. 2B

FIG. 2A. (× 800) "Dust cells" collected in alveolus of human lung. Note refractile silica granules in cytoplasm

FIG. 2B. (× 120) Early silicotic fibrosis—human lung. The alveolar walls are thickened causing collapse of alveolus around dust cells

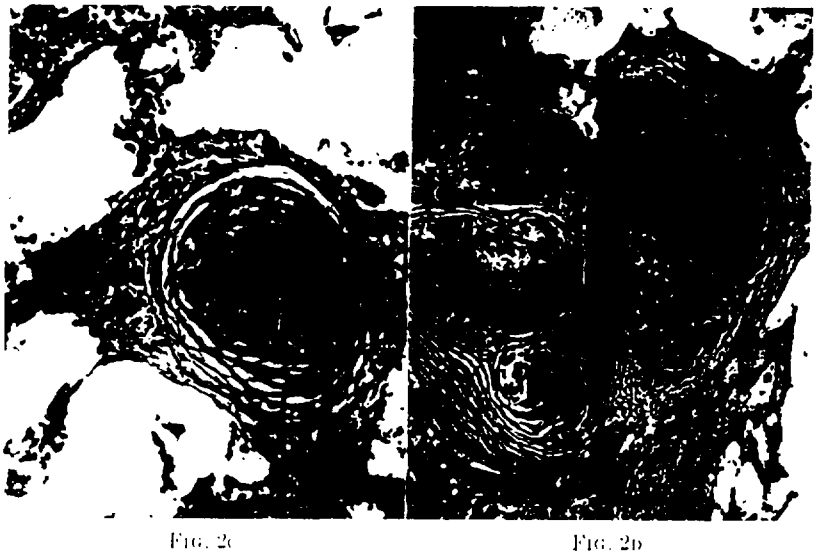

FIG. 2C FIG. 2D

FIG. 2C. (× 50) Early characteristic silicotic nodule—human lung

FIG. 2D. (× 10) Confluent silicotic nodules in massive fibrosis—human lung

fibroblastic reaction and collagen deposition. Some may interpret this as a protective action on the part of the tissues to wall-off the accumulation of dust cells. As the fibrosis progresses the "mummified" dust cells disintegrate and the silica lies free in the centers of these fibrotic nodules. The nodules grow by peripheral concentric accumulation of layer upon layer of dust cells eventually producing a whorl-like collagenous structure (fig. 2c). Much of this collagen appears to be deposited without the usual number and activity of fibroblasts found microscopically in other types of fibrosis. This acellular type of fibrosis seems to be peculiar to silicosis. As these fibrotic nodules progress in size they grow closer together and finally coalesce into large masses of collagen with numerous centers of accumulated and trapped dust (fig. 2d). The dissolution of the dust does not stop with the walling-off by fibrous tissue, but continues as small but constant amounts of tissue fluid diffuse into and out of these nodules. Therefore, in the young and small silicotic nodule there is less fibrosis and a large amount of entrapped silica dust, while in the old nodule (particularly in the individual who has been removed from dust exposure for a period of years), there may be very marked fibrosis, but much less silica dust.

This is the picture of the development and progress of so-called *simple silicosis*. A further complicating histological finding, especially in hard rock and coal miners, is coal dust in the form of carbon particles which are found mixed with the silica dust in all the nodules. Add to this the complicating picture of scarring and chronic inflammation due to infection of the lungs and the pathology of complicated silicosis is complete.

Nodular fibrosis is not the only type which occurs in silicosis. Even in the simple form there is always a second type which may be termed *linear fibrosis*: a thickening of the walls of the lymphatics often causing complete obstruction of the lymphatic channel. This is probably caused by constant deposition of collagen in the region of scattered dust cells along the walls of the lymphatics. Many studies have shown that although progressing more slowly than the fibrosis of the nodules in the lymph nodes and lymphoid centers, this fibrosis is definite and eventually obliterates the lymphatics. This results in stagnation of peripheral lymph in the lung and thereby increased accumulation of the dust cells and subsequent fibrosis peripheral to the obstructed lymphatic channels. Therefore, in lungs without previous or concomitant chronic or acute inflammatory changes, continuous exposure to hazardous concentrations to silica dust will eventually result in a fibrosis of the entire lung from hilum to pleura. It is obvious that with complicating infection, this extensive fibrosis will be much more rapid.

Acute Silicosis

The pathogenesis of so-called "acute silicosis" is different from that described above according to reports of several investigators. Cole and Cole

have published their studies of lungs from patients suffering with acute silicosis (especially sandblasters in whom the duration of the disease was in terms of months rather than years). By special stains (Masson trichrome) for collagen, fibrous tissue, and blood vessels, they have found that in the instances of exposure of workers to enormous concentrations of very finely divided particles (which are rapidly soluble in slightly alkaline tissue fluid), there occurs a thickening of the pulmonary capillary walls. They have produced evidence that this is true collagen deposition. They report focal capillary obstruction which causes marked respiratory embarrassment without accompanying severe fibrosis. In later stages there is actual collagen deposition and fibrosis of the capillary walls and the lymphatics resulting in characteristic linear peribronchial fibrosis revealed by roentgenological examination. Although this hypothesis of capillary obstruction due to the toxic action of rapidly dissolving silica in the acute cases has not been confirmed by other workers, it is at present the only plausible explanation. The reader is referred to the monograph of Cole and Cole on pneumonoconiosis for further details concerning this aspect of silicosis.

III. THE DISEASE

Roentgenological examination of various portions of the body reveals abnormal tissue conditions and, therefore, constitutes a method for the study of pathological processes. Consequently, the x-ray appearance or roentgenopathology, histopathology, and gross anatomical changes will be described together. For purpose of convenience the silicotic changes in the lungs will be described in several stages: the early non-clinical stage found only by roentgenological examination or accidentally on post-mortem examination when death is caused by intercurrent disease or accident, advanced simple silicosis, and complicated silicosis, particularly silico-tuberculosis.

The clinical manifestations of silicosis and its complications are best divided into several phases for clarity of discussion. In all cases of dust exposure, silicosis should be suspected and in all individuals with pulmonary symptoms, a careful history should be obtained to determine exposure to silica dust.

History

Complete occupational history always should be obtained and this must include detailed information concerning all occupations and industries in which the patient has been employed throughout his working life. It is often advisable to record this history in chronological order and if any data indicates probable or definite exposure, this particular portion of the history must be obtained in detail including description of any symptoms during this period of exposure, the exact length of exposure, the industry,

and details concerning the patient's exact duties, hours of work per day, precautions taken, if any, etc. All facts of ill health or upper respiratory infection immediately before or during the time of exposure to the dusty occupation should be carefully noted. Past medical history, particularly in relation to upper and lower respiratory infections, is an important aid to diagnosis and the date of onset and duration and severity of any such illnesses are to be recorded. History of pneumonia, "flu," and bronchitis must not be overlooked because these diagnoses not infrequently inadvertently cover tuberculosis of which fact the patient and his attending physician remain ignorant.

Acute or Rapidly Developing Silicosis

Diagnosis.—The patient always complains of sudden onset and rapid development of shortness of breath, cough, and sometimes chest pain. There will be a history of progressive weakness, loss of weight, digestive disturbances, and occasionally, the patient will complain of palpitation and pain over the heart. Examination reveals objective symptoms of pulmonary and cardiac embarrassment and these findings may cause confusion of this disease with such conditions as pulmonary edema, lobar pneumonia, bronchial asthma, cardiac decompensation with pulmonary congestion, or advanced tuberculosis. The cough is usually dry, but may be productive. Hemoptysis is rare, but if there is a superimposed pneumonitis, this may occur. Chest expansion is definitely decreased and the expiratory movement is prolonged over the inspiratory movement. Percussion elicits moderate hyperresonance over emphysematous areas when such have developed. Increased dullness over much or all of the lung field is the more common finding. Breath sounds may be increased over these areas of congestion and partial alveolar collapse while beginning focal pulmonary exudation and transudation may produce sibilant or musical rales. There are no findings of consequence in the examination of the peripheral blood. Urinary examination reveals nothing except by special chemical tests which will demonstrate the presence of excessive dissolved silica in the urine according to King. Especially in cases exposed to high concentrations of silica, relatively large amounts are reported to be eliminated in the urine. This test is not practical in routine diagnosis of industrial cases and is of only experimental use at the present writing. Sputum examination for silica is considered of some diagnostic value. Examination of fresh sputum under the microscope by dark field reveals large characteristic refractile silica particles in the alveolar phagocytes. By the experienced worker, this dust can sometimes be seen with bright field illumination. Smears, which have been incinerated, will reveal large concentrations of silica dust in the phagocytes of the sputum. Chemical

tests for silica can be used with larger quantities of sputum, if obtainable, but the tests are rather complicated and will not be found practical in routine industrial investigations.

Prognosis.—The prognosis of acute silicosis is usually very grave. The great amount of dust already inhaled before the patient is discovered will produce a progressive fibrosis in spite of removal of the patient from the dusty occupation. Fatal complications are most frequently superimposed respiratory infections, but instances of cardio-vascular decompensation have been reported.

Simple Silicosis

The fibrosis produced by inhalation of silica dust only is rarely seen except in experimental animals. Occasionally, it is seen in rapidly advancing silicosis of young workers such as sandblasters and drillers employed in tunneling through sand under river beds. In the early stages of this disease, the x-ray findings are identical with those described below (Stage 1).

In the later stages of simple silicosis, the histopathological picture remains free from inflammatory changes and pigmentation by coal dust. Otherwise the clinical and pathological picture is identical with that of anthracosilicosis.

Anthracosilicosis

This is the most common form of silicosis occurring in patients and discovered incidentally upon routine examination of miners and workers in dusty trades.

Early (Stage 1)

Roentgenopathology.—The earliest stages of silicosis and anthracosilicosis are always asymptomatic and produce no physical signs. This stage is discovered only by routine x-ray examination. The roentgenogram of the lungs reveals typical discrete miliary nodulation of both fields, particularly in the mid portions, with characteristically clear bases (fig. 3a). The miliary nodules are seen as uniformly distributed small densities. In some instances there are also increased linear peribronchial markings due to peribronchial lymphatic fibrosis in an early phase.

Histopathology.—In the affected areas of lung tissue, many patches of alveoli contain accumulation of alveolar phagocytes which are engorged with minute refractile granules which by special processes may be identified as silica dust and larger particles of carbon. There is moderate thickening of the inter-alveolar tissue by accumulation of dust cells and beginning deposition of collagen. A bit later young fibroblasts will be

seen. In older areas uniformly small nodules occur. These are made up of collagenous tissue with a center of hyaline matrix in which there may or may not be multinucleated giant cells loaded with silica dust. Around the collar of collagen there is a zone of degenerating dust cells and round cells. In the regions of early fibrosis there is also found a hyaline thickening of the walls of the lymphatics due to deposition of collagen. Fibroblasts may or may not be present. In carefully prepared sections and particularly in experimental animals, there are frequently seen one or more layers of dust cells coating the lymphatic walls. Frequently, flat, plaque-like, thickened, hyaline apical scars immediately beneath the pleura are found to contain large amounts of anthracotic dust. These scars are densely fibrous and lack typical silicotic or tuberculous nodulation, but have been found to contain abnormal quantities of silica and probably result from areas of poor aeration and stagnation. They have not been proved tuberculous as long claimed.

Intermediate Stage (Stage 2)

Symptoms.—With the onset of symptoms the progress of silicosis may be stated to have entered the intermediate stage. The onset is insidious and the patient first notes that upon excessive exertion shortness of breath is more marked than he has experienced before. This shortness of breath gradually becomes more noticeable and later there may be added a slight dry cough. The patient is normal in all other respects having lost no weight, frequently being robust and healthy in appearance. Chest pain is rare, but may occur in this stage, particularly in the more rapidly advancing type of silicosis. When present it is described as being a sense of painful compression of the chest. The dyspnea is unusual and characteristic in that there is no orthopnea and the patient will complain that he obtains no relief from sitting up in bed. Hemoptysis is rare.

Signs.—Definite signs are not always present at the beginning of the second stage. However, as this disease progresses and respiratory embarrassment becomes evident, chest expansion will be decreased; the expiratory phase of respiration becomes prolonged; dyspnea following exercise becomes more marked; there may be a slight increase in tactile fremitus of uniform distribution over the entire chest and to percussion there may be found decreased diaphragmatic excursion and some slight impairment in resonance over all the lung areas except those involved by emphysema. Upon osculatory examination decrease in breath sounds may be general, particularly as the condition progresses. A few subcrepitant rales are heard in occasional cases of silicosis and these clear up following cough, but only upon the development of extensive emphysema are wheezing musical rales commonly heard. Decreased chest expansion is not a reliable sign because there is much variation between chest expansion of

various individuals and only upon repeated examination of the same patient can a progressive decrease in the amount of expansion be determined and considered significant.

As pulmonary fibrosis progresses, signs of disability may appear or be elicited by exercise tests. Increasing dyspnea is the outstanding symptom and repeated examinations over a period of several years in the average case will disclose progressive lengthening of the recovery period for the respiratory rate to return to normal after a given amount of exercise.

Diagnosis.—Although anthracosilicosis is a very clear-cut disease with a single well-proven etiological agent and a definite progressive course, the pathologic changes cause symptoms which are not particularly characteristic because they are often associated with many other cardiac and respiratory diseases.

Roentgenopathology.—Although more reliable, the roentgenological findings are not absolutely characteristic. Considerable experience and thorough familiarity with all aspects of this disease are required to qualify a physician as competent in the differential diagnosis of silicosis especially when complicated by infection. The roentgenologist must examine the patient carefully, obtain his occupational and past history, and be informed concerning all aspects of the case before he can properly interpret the roentgenological findings. Otherwise, the roentgenologist should phrase the report in descriptive terms for interpretation by the physician who *is* thoroughly familiar with the patient. This correlation of all facts and findings for accurate diagnosis and the legal aspects of the problem, which place important responsibility upon the medical profession, are especially emphasized in the National Silicosis Conference Report on Medical Control (1938).

The shadows seen on roentgenological examination are larger conglomerate patches of densities due to coalescence of the smaller nodules with increasing fibrosis. The large patches are still separated by areas of well aerated lung, but beginning emphysema is found. Although areas of clear normal lung in the bases and extreme apices may persist, the apices are usually found involved in this stage. The domes of the diaphragm may be moderately depressed and there is always considerable intensification of the trunk shadows. Enlarged hilar nodes are extremely dense, but may not be visualized due to the large overlying patches of conglomerate nodules (fig. 3b).

Pathology.—The lungs are grossly filled except in apices and bases by minute uniform hard nodules which are dull greenish-black or black and which are prominent on the anterior surface of the lung specimen. More involvement is manifested by scattered larger areas of coalescent nodules and intervening patches of crepitant emphysema.

As would be expected the microscopic picture is that of coalescence of

the numerous medium-sized and small silicotic nodules containing both silica and carbon dust. The central portion of the nodules shows a

FIG. 3A. Silicosis Stage I

FIG. 3B. Silicosis Stage II

FIG. 3C. Silicosis Stage III

granular degeneration. There are fewer giant cells and the periphery of the nodules consist of active fibroblastic proliferation and intermixed

accumulation of dust cells. Trapped between many of the coalescing nodules are areas of atelectatic lung tissue and small gland-like structures of bronchiolar cuboidal epithelial cells.

Laboratory findings are of little help in establishing the diagnosis except in those instances where the patient is being exposed to high concentrations of silica dust in the occupational atmosphere. In such cases examination of the sputum will often reveal large numbers of dust cells and the silicotic dust content may be identified by histo-incineration, dark field examination, or, if available, by careful chemical analysis.

Differential diagnosis of stage 2 silicosis is frequently difficult due to the many pulmonary diseases, such as early bronchiectasis, disseminated tuberculosis, and pulmonary lymphatic extension of metastatic carcinoma or primary carcinoma of the lung, which may mimic the pathological and roentgenological findings in this stage. Experienced roentgenologists can differentiate the characteristic findings of silicosis from other pulmonary diseases by x-ray examination and the above described laboratory findings are diagnostic when positive. However, from clinical history and physical examination alone, a diagnosis can never be conclusively established, merely suspected. It must be remembered that many patients who have been exposed to silica dust at some previous time, or are under constant exposure to moderate concentrations of silica dust may contract lung diseases other than silicosis and these should not be ignored in making a differential diagnosis. Before disability develops, definite diagnosis can be established only by the most cautious and complete studies with exclusion of all other diseases which produce similar roentgenographic, laboratory, and physical manifestations.

Progress with Disability.—As stage 2 of this disease approaches the more advanced type, disability may become apparent and is usually manifested by shortness of breath upon exertion and rarely by so-called asthmatic paroxysms. Weakness is not a common complaint even with marked dyspnea. The chief complaint is usually inability to continue work because of shortness of breath upon normal exertion required by the occupation. This symptom becomes worse until it advances to the profound dyspnea of stage 3.

Advanced Stage (Stage 3)

Symptoms.—The term, stage 3, is only a convenient designation for the most advanced type of silicosis in which disability is marked. Disability is due to severe shortness of breath, inability to work, and in some cases, cardio-vascular complications caused by the abnormal load upon the heart. It is questionable whether or not a normal heart, which has not previously been injured by some other disease, will undergo hypertrophy, fibrosis, or

other pathological change in response to decreased pulmonary vascular bed alone. It is claimed that in the most severe cases and in so-called acute silicosis, rapid reduction of the vascular bed may produce cardiac dilatation even in the previously uninjured heart.

Signs.—As would be expected, the signs of advanced fibrosis of the lung include widespread dullness over both lung fields anteriorly and posteriorly; decreased or absent breath sounds due to emphysema and fibrosis; and frequently, patches of coarse rales heard over localized areas of bronchiectasis and pneumonic infiltration.

Diagnosis.—Laboratory diagnostic procedures are practically useless because usually the patient has been long removed from exposure to dust and no silica can be found in the alveolar cells of the slight sputum produced. Coughing may become persistent, but is only productive when there is a complicating infection. Dyspnea may advance to the stage of cyanosis. In long standing cases with cardio-vascular involvement, club fingers and cyanotic nails are occasionally seen. Differential diagnosis in this stage is not difficult because of characteristic roentgenological signs of massive conglomerate and complete opacity of the lungs.

Roentgenopathology.—The x-ray picture of this late stage reveals a mass of large dense shadows leaving little or no normal aerated lung (fig. 3c). Emphysema is marked and trunk shadows are completely obliterated by the large conglomerate shadows. The apices and bases have become diffusely involved. The domes of the diaphragm are depressed and frequently there is exaggerated posterior bowing of the spine. If the emphysema is extreme, there will be increased widening of the intercostal spaces.

Pathology.—Grossly the lung is a voluminous "petrified" specimen, stony throughout with only small irregular areas of softer, paler tissue. The cut surface is granite-like, but possesses blacker mottling. Microscopic sections from lungs of the third stage reveal massive areas of fibrosis obscuring the individual nodules. These masses of hyaline tissue appear to be closely packed together and the narrow intervening and peripheral spaces of emphysematous lung are seen in conspicuous contrast.

Prognosis.—Occasionally, termination is due to cardio-vascular failure with pulmonary edema and acute dilatation of the heart, but more frequently complicating pulmonary infection in the form of a nonspecific pneumonia or tuberculous pneumonia is the fatal end result. Prognosis of simple silicosis in the earlier stages may be said to be good for life and disability if the patient is removed from exposure to hazardous concentrations of silica dust. Fibrosis will progress even though the patient is removed from such surroundings, but will gradually reach a maximum and from that point become static and no further involvement of the lung

tissue will take place. The prognosis of advanced silicosis is definitely grave because of the extreme susceptibility to complicating nonspecific or specific infection of the lungs leading to a fatal pneumonia. Questionably, in cases with normal heart, and certainly in instances where the heart has previously been damaged by some other disease or by arteriosclerosis, advanced silicosis will produce marked overwork of the cardio-vascular system and not infrequently may cause fatal cardio-vascular decompensation.

IV. COMPLICATED SILICOSIS (SILICOSIS WITH INFECTION, SILICO-TUBERCULOSIS)

Very few proven cases of simple silicosis are on record. Partly due to the common and widespread incidence of respiratory infection of all types, but particularly because of increased susceptibility of tuberculous patients to silicosis and of silicotic patients to tuberculosis, mixed infection and silicosis is the usual picture found at necropsy. Chronic bronchitis, pneumonia, active tuberculosis, and other infections are frequently associated with silicosis and, therefore, complicate the pathologic picture and the clinical differential diagnosis. The industrial physician will rarely see uncomplicated simple silicosis and he will be confronted with vexing problems concerning the source of the disability and its proper compensation.

Bronchopneumonia in Silicosis

Whenever silicosis is complicated by bronchopneumonia, the pathological picture is that of the advanced silicotic fibrosis superimposed by infiltration of monocytes and polymorphonuclear leukocytes into the emphysematous or partially collapsed alveoli. The inflammation may be caused by any one of the many microorganisms which affect the lung.

Silico-tuberculosis

From the earliest recognition of silicosis students of this disease have noted that silicotic patients are apparently much more susceptible to pulmonary tuberculosis than other individuals. A great amount of laboratory and clinical research has been done to explain this apparent increased susceptibility. It is impossible in this brief consideration to include an exhaustive discussion and review of the theories and findings resulting from this work. However, it can be stated definitely that the incidence of tuberculosis among sufferers from silicotic fibrosis of the lungs is definitely much higher than that in the general population.

Pathogenesis.—Many laboratory studies, including animal experiments and test tube cultures by workers at the Saranac Laboratory and the University of Toronto and others, apparently indicate that the presence

of silica in culture media or in the animal lung or other animal tissue definitely favors the growth of tubercle bacilli. This may explain the apparent increased susceptibility of silicotic patients to complicating tuberculous infection. Another factor which must not be overlooked is the decrease of aeration in the lungs due to marked silicotic fibrosis. The numerous stagnant pockets formed by scarred areas containing imprisoned bits of bronchi which later dilate and accumulate secretion and the decreased circulation through the fibrotic areas and their vicinity undoubtedly are significant physiological and physical factors favoring infection and inflammation. Therefore, any type of infection may become seated in these areas, but because of the general susceptibility of the man to tuberculosis, this infection more frequently obtains a foot-hold in the less resistant lung. It is conceivable that if enough cases of non-silicotic types of long standing chronic advanced fibrosis of the lung could be collected and thoroughly studied, a high incidence of complicating tuberculous infection might be found due to the same physical and physiological circumstances outlined above. Too few cases of severe x-ray fibrosis of the lung have yet been accumulated to justify conclusions from study of this type of fibrosis.

Reports thus far published to support the claim that silicotic fibrosis of the lung breaks down old calcified or fibrous tuberculous scars and reactivates an old tuberculous infection are not conclusive or convincing. It is much more probable that because of the well known wide distribution of tubercle bacilli, a reinfection tuberculosis occurs in the silicotic individual. It is certain that in mines and many other industries there is sufficient opportunity for the silicotic employees to be exposed to other individuals who are open cases of tuberculosis. This fact and the low incidence of tuberculous complication in workers (including silicotics) reported from certain isolated or selected industries and mines may be accounted for by the varying incidence of tuberculosis in these communities.

Symptoms and Signs.—The manifestations of silico-tuberculosis are those of a fibrosis as described above under simple silicosis plus those of tuberculous infection. The roentgenological findings are definitely suggestive and sometimes almost conclusive to the experienced roentgenologist. The signs are those of bronchial and pulmonary infection at first in localized areas and later in large areas denoting pneumonic involvement. Tuberculous pneumonia is not uncommon as a terminal process in the advanced silico-tuberculous patient. In fact, frequently the typical findings and symptoms of tuberculosis may overshadow those of silicosis to the extent that the latter is not diagnosed until the necropsy.

Diagnosis.—Undoubtedly, many cases of so-called silico-tuberculosis which have been reported in the literature are not true tuberculous infections since no definite proof by culture or guinea-pig inoculation has been

produced to show the presence of tubercle bacilli. Roentgenological findings are frequently inconclusive, but there are certain manifestations of complicating infection which can be properly interpreted and evaluated by the experienced roentgenologist, particularly if he has available the complete history, laboratory findings, and data concerning exposure to silica dust. It is the author's opinion that the diagnosis of silico-tuberculosis should never be made in any case until tubercle bacilli are demonstrated in the sputum or a positive guinea-pig test completed. Otherwise, even though definite findings of infection are seen on roentgenograms and are apparently confirmed by physical findings (rales, increased breath sounds, areas of pneumonic dullness, and productive cough) the diagnosis of *"silicosis with infection"* is preferable. Differential diagnosis is established only by the finding of tubercle bacilli in the sputum. All the laboratory studies, such as tuberculin test, sedimentation time, complement fixation test, leukopenia with predominating monocytes, guinea-pig inoculation with the production of tuberculosis in the animal, will, of course, aid in establishing a diagnosis.

Progress.—Clinical pictures of progressive silico-tuberculosis are as protean as those of tuberculosis. The numerous variable controlling factors include the degree of silicotic fibrosis, the dose and virility of the tubercle bacilli causing the reinfection, the so-called natural resistance or biological resistance of the particular patient to this disease, the physical and atmospheric surroundings, continuation or cessation of exposure to the reinfecting source of tuberculosis, the degree of allergic response of the particular patient to the tuberculous infection, the continuance or cessation of exposure to silica dust, the general working conditions, and the physical condition of the patient. These factors combine in various forms and degrees of inter-relation to produce innumerable manifestations of tuberculosis complicating silicosis.

Roentgenopathology.—In silico-tuberculosis the roentgenologic findings are much more confusing and complicated than in either anthracosilicosis or uncomplicated tuberculosis. There is mottling characterized by shadows which are ill-defined with "fuzzy" borders and vary in size and distribution. These shadows usually denote inflammatory infiltration. In more advanced cases the "soft" nodulation acquires flocculent irregular and indistinct borders larger but of irregular size, varying density, and distribution. Massive shadows of homogeneous density not of inflammatory origin with asymmetry of distribution denote the presence of silicotic fibrosis. Diffuse, dense opacity of entire lobes usually indicates tuberculous pneumonia superimposed upon the silicotic nodular fibrosis. Outlines of normal structures, such as the vascular bed and bronchial tree, will be partially or completely obliterated.

Pathology.—The lungs are stony, bizzare masses of infected gray foul

tissue and granite blackened by carbon dust and silica. Gross cavitation with exudate of secondary infection is common. Normal anatomic structures are often not recognizable. There is much disagreement in the literature concerning the origin of many of the pathological lesions found in so-called silico-tuberculosis. The Saranac group are of the opinion that the early caseation of tuberculous nodules cannot be distinguished histologically from the acute necrosis of silicotic nodules which also may produce an acute monocytic inflammatory reaction in the peripheral zone. Many other students of silicosis disagree with this statement and claim ability to differentiate the characteristic histologic features of silicotic and tuberculous nodules. For the purpose of the student of industrial medicine, it suffices to emphasize that in this disease, regardless of pathological controversies, the diagnosis can be established only by characteristic roentgenological findings and the demonstration of tubercle bacilli in the sputum or (and) a febrile reaction to the Mantoux, patch or percutaneous tuberculin test. In almost all cases of silico-tuberculosis, there will be demonstrated either an old silicotic fibrosis with superimposed inflammatory tuberculous giant cell nodules, caseation necrosis with or without cavitation, or the opposite picture of old fibro-caseous tuberculosis or arrested quiescent tuberculosis with calcification of hilar nodules and peripheral scars and a more recent uniformly distributed miliary non-necrotic silicotic nodular fibrosis. Large areas of caseous pneumonia with the characteristic accumulation of masses of monocytes and caseous necrotic debris leaves no doubt about the tuberculous nature of the pneumonitic reaction. Cavitation is frequent, but may be bordered by walls containing silicotic nodules. In specimens examined under the dark field after histo-incineration, there will be found very little or no silicotic dust in the central portion of tuberculous scars whereas the peripheral zones will contain varying amounts of silica depending on length of exposure to silica. The reverse pattern of much silica in the predominating silicotic fibrous tissue in contrast to much less in the superimposed inflammatory tissue is revealed by histo-incineration.

As will be pointed out in the paragraph on control, it is needless to caution that the open case of silico-tuberculosis is as great a hazard to his fellow workers and members of his immediate family and to society as is any case of active tuberculosis. It is also obvious that in cases of active silico-tuberculosis, all treatment should be focused on the control of the tuberculosis since this complication will sooner or later completely incapacitate the individual so far as industry is concerned. In occasional instances when the diagnosis of tuberculous complication is made in the middle stage of silicosis or early in the tuberculous phase of the disease, properly instituted treatment including rest, diet, fresh air, removal from exposure to source of infection, etc., may arrest the disease and eventually permit

the patient to return to moderate physical activity. However, almost all cases are discovered in a later stage when the tuberculous infection is advanced. Progress of the infection in these cases is always rather rapid and ever increasing involvement of the lungs due to the widespread silicotic scars and poor aeration and circulation may be expected. The extension of the tuberculous infection is usually by aspiration due to the many areas of poorly functioning bronchi with accumulated debris and secretion and also by contiguous tissue due to the poorly vascularized silicotic scars. Aerated lung may be infiltrated by characteristically monocytic cells and large areas of caseation necrosis as seen at necropsy. In some instances this process may progress rapidly into all the uninvolved portions of the lungs and result in a rapid fatal termination.

Prognosis.—The prognosis is obviously very grave. However, prognosis must be guided by consideration of the variable factors outlined above, and in the more mild infections of low virulence with manifestly better resistance, patients may survive for considerable periods of time and occasionally are able to earn a living at some minor trade for a period of years. Unfortunately, the greater proportion of cases do not follow this course. If diagnosis could be established earlier and effective treatment for the tuberculous infection instituted, prognosis would certainly improve.

The question concerning the increased susceptibility of old cases of pulmonary tuberculosis to fibrosis produced by silicotic dust in the lungs will be mentioned only to point out that it is the author's opinion that there is a definite increased susceptibility of tuberculous patients to silicotic fibrosis, but that this is due to the closed lymphatic circulation produced by the tuberculous infection and in no way is related to any specific affinity for silicosis or silicotic scarring to tuberculous areas. It is the author's belief that any other type of widely distributed scarring or necrosis produced by an infection and resulting in closure of the pulmonary lymphatic system would produce a similar degree of so-called increased susceptibility to silicotic fibrosis.

Silicosis of Other Organs

Silicotic nodulation of the liver and spleen is described in experimental work. Occasional silicotic nodules have been discovered in post-mortem examination of individuals exposed to high concentrations of silica dust. No clinical manifestation or significant injury has been demonstrated to arise from such accumulations of silica in the viscera.

V. COMPENSATION OF SILICOSIS

In order to adequately cover the involved subject of industrial compensation in silicosis, an entire chapter would be required. Only a few pages can be devoted to the problems peculiar to silicosis.

TABLE 1.—*Workmen's Compensation Provisions for Silicosis*
(Summary of Coverage Provisions for Silicosis as of January 1, 1938)

State	(1)* Administrative agency	(2) Medical boards	(4) Coverage	(9) Partial disability	(10) Medical care
California	Industrial Accident Commission.	No board; Commission may appoint special examiners.	General; compulsory.	Compensated.	Unlimited.
Connecticut	One Commissioner for each of 5 districts.	No provision.	General; elective.	Compensated.	Unlimited.
Illinois	Industrial Commission in Department of Labor.	No board; Commission may appoint special examiners.	General; elective.	Compensated.	6 months.
Indiana	Industrial Board in Division of Labor.	No board; Industrial Board may appoint examiners or other experts.	General; elective.	Compensated.	30 days; may be extended to 60 days.
Kentucky	Workmen's Compensation Board in Department Industrial Relations.	No board; examiners shall be appointed, on application, by Workmen's Compensation Board.	Voluntary; by joint election.	Compensated.	90 days and $200; may be increased to $400.
Massachusetts	Department of Industrial Accidents.	3 Medical referees whose diagnosis is binding.	General; elective.	Compensated.	2 weeks; may be extended in certain cases. 90 days.
Michigan	Department of Labor and Industry.	Commission must appoint 3 impartial physicians in disputed cases; majority decision final.	For mining only; elective.	Not compensated.	90 days.
Missouri	Workmen's Compensation Commission.	No board; Commission may order examinations.	General; elective.	Compensated.	90 days and $750; may be increased.
New York	Industrial Board with Department of Labor.	Board of 3 for all dust diseases; findings advisory.	General; compulsory.	Not compensated.	90 days; may be extended to 180 days.
North Carolina	Industrial Commission.	Medical Board; findings advisory; supervises periodical examinations.	Schedule; elective.	Compensated.	3 years; not exceeding $334 a year.

North Dakota	Workmen's Compensation Bureau.	No board; Bureau may appoint special examiner.	General; compulsory.	Compensated.	Unlimited.
Ohio	Industrial Commission.	Commission shall appoint 2 silicosis referees; findings advisory.	Schedule; compulsory.	Not compensated.	$200; may be increased in unusual cases.
Pennsylvania	Workmen's Compensation Board in Department of Labor and Industry.	Medical Advisory Board appointed from panel by Workmen's Compensation Board.	Schedule: elective.	Not compensated.	3 months and $200; may be increased.
Washington	Department of Labor and Industries.	No Board; Department may require examinations.	Schedule; compulsory.	Compensated.	May be limited to compensable period for certain disabilities.
West Virginia	Workmen's Compensation Commissioner.	Board of 3; findings advisory, and reviewable by Commissioner.	Special fund; elective.	Compensated.	$800; may be increased to $1,600.
Wisconsin	Industrial Commission.	Commission may appoint from panel, or may appoint special examiners; all findings advisory.	General; compulsory.	Compensated.	Limited to compensable period.[1]

[1] Construed by Industrial Commission as "unlimited."
Division of Labor Standards, U. S. Department of Labor.
* Due to limitation of space, columns 3, 5, 6, 7, and 8 of the original table have been omitted.

Disability and Compensation

Although it is well proven that silicosis definitely produces disability, the problem of compensation for disability has caused much legal and medico-legal confusion and has resulted in many law suits involving large damage claims against industries and mining companies. The term, disability, is defined in the National Silicosis Conference Report as "a decrease in capacity to do work required of an individual in the course of his usual occupation." It may vary from partial to complete and may persist as partial or, in the case of progressive fibrosis due to large amounts of previously inhaled silica or continuous exposure to dangerous levels of silica dust in the industry, may progress to serious and permanent total disability.

In many states disability due to silicosis is still not compensable, but the various states which have passed compensation laws including this disease are given in table 1.

As a result of the large amount of legal action and legislation concerning silicosis disability in many states, it has become the general precaution of employers in industries involving silica dust hazards, to reject for employment any persons who have already developed silicotic fibrosis or other types of fibrosis of the lungs demonstrable by x-ray examination. This causes much hardship upon employees who have learned their trade well and become experienced over a period of years in occupations involving the silica dust hazard. It is the opinion of many medical experts that whenever pre-employment examination discloses fibrosis of the lungs in a person who has been exposed in a dusty industry for a period of years and that whenever this fibrosis is not disabling and is not complicated by infection, this person should be recommended for employment as a good risk, furthermore, provided that the silica dust hazard has been eliminated and the concentration of dust in the new job is below the hazard level. There are several obvious advantages to such a procedure, the chief advantage being that such a person has already been tested, so to speak, concerning his ability to withstand silicotic fibrosis without suffering complicating respiratory infection, particularly tuberculosis. Also, such a person has developed a certain skill in that particular occupation which is of value both to himself and his employer. He is a much smaller risk than a younger man who is inexperienced, untrained, and whose reaction to silica dust exposure is unknown even though examination may fail to disclose any respiratory infection.

Tests for Disability.—Much research, both clinical and laboratory, has been reported concerning practical tests for measuring the degree of disability produced by silicotic fibrosis of the lungs. Most of these tests are based upon methods of measuring lung function. Many of these tests are

impractical because the necessary equipment and time for their performance are not generally available. The practical test which is suggested by the National Silicosis Conference Report on Medical Control is as follows:

> The individual places one foot upon a chair or firm stand, 18 inches in height, and raises his body to an erect position 25 times in 30 seconds. Those with marked respiratory or cardiac disturbances cannot be subjected to such a test, but in the course of examining persons already employed or applying for work few of such individuals will present themselves. The pulse and respiration are taken with the examinee at rest, immediately following the exercise test, and after a 2-minute rest period. It is evident that such a test is not one whose results are affected by respiratory conditions only, but one which is influenced by various factors, such as weight, heart condition, age, general physical condition, and numerous metabolic differences. It does serve as an indication of decreased capacity for work. When we consider all factors, definite information concerning respiratory capacity is available. Regardless of the presence of slight cardiac defects or changes due to age, etc., the individual with appreciable pulmonary fibrosis will not only exhibit shortness of breath following the exercise, but the increased respiratory rate will persist well beyond the 2-minute rest period, and the altered respiratory rhythm is characteristic. The expiratory phase is markedly prolonged often to such an extent that the rate of respiration is much less than in the case of a person with pulmonary infection or cardiac disturbances. Due to loss of elasticity of the lung, the individual cannot empty his lungs rapidly enough to allow for a great increase in respiratory rate. In fact, the rhythm approaches that of the asthmatic before exercise. The condition may be differentiated from bronchial asthma by the absence of the rales and other clinical data. In cases of well-established silicosis, prolonged expiration may be elicited by careful observation during the course of physical examination, prior to any exercise test.

VI. CONTROL OF SILICOSIS

Because silicosis is caused only by silica dust, but is complicated by infection and because of the apparent predisposing action of healed or latent tuberculosis and other chronic infections of the lungs, all methods of control must take cognizance of both exposure to silica dust and the health of the workers before employment in a dusty occupation.

Thus, the control of silicosis may be divided into two general types: First, the *medical* control through physical examination before employment, periodic health examination during employment, and education of the employees and employers concerning general hygienic measures, and maintenance of good health with reduction of upper respiratory and pulmonary disease; and secondly, *mechanical* control whereby the actual concentration of silica dust in the air to which the workers are exposed is reduced to a level less than the hazardous concentration and maintained by continuous efficient operation of the mechanical devices. These methods of control are discussed in detail in Chapters II, XIII and XV.

VII. OTHER PNEUMOCONIOSES

There is considerable disagreement in the medical literature concerning the exact etiology of certain types of pulmonary fibrosis due to dust inhalation other than silica dust. Asbestosis, siderosis, and anthracosis are the most important of these fibroses.

Asbestosis

It has been well proven that a peculiar type of linear pulmonary fibrosis is caused by minute asbestos fibers inhaled by workers in industries utilizing asbestos for the manufacture of fire resisting material, insulation, certain fabrics and ropes, paints, roofing, tile, etc., as well as the manufacture of asbestos itself.

Asbestos is magnesium silicate and is the only silicate which has experimentally been proven to produce a pulmonary fibrosis without exposure to silica.

The onset and clinical course of asbestosis is similar to that of slowly progressing silicosis and the symptoms in the later stages are identical. Fatal termination is usually by complicating pneumonitis, cardio-vascular decompensation, or tuberculosis. The roentgenopathology is typical and not difficult to differentiate from silicosis. Gloyne and Merewether state that the roentgenological appearances of asbestosis are revealed typically as a general lack of translucency with a fine pinhead mottling. This is the characteristic "ground glass" appearance. The film should always be compared with a film of a normal patient and a film of a silicotic patient taken with the same exposure.

The diagnosis depends upon a definite history of exposure to asbestos dust including sufficient period of exposure, roentgenological appearance, decreased pulmonary capacity, and the finding of asbestos bodies in the sputum of patients not yet removed from exposure to the dust. These asbestos bodies are well described and usually illustrated in most text books of clinical pathology. The pathology may be summarized in the single statement that it consists of a persistent, progressive, diffuse perialveolar fibrosis of the lungs. The fibers may be seen on histological examination to lie embedded in the fibrous walls of alveoli and bronchioli and each fiber appears to be encrusted by an iron-containing yellow pigment. As would be expected, in the later stages the fibrosis becomes confluent and massive.

Siderosis

Siderosis is chiefly a red pigmentation of the lung tissue due to inhalation of iron-containing dust frequently found in hematite miners. Experimentally, fibrosis has not been produced by pure iron or iron oxide dust

and only when mixed with hazardous concentrations of silica dust does fibrosis result. Therefore, unless the patient is also affected with silicosis siderosis by itself is asymptomatic and produces no roentgenological or clinical findings.

Anthracosis

Black pigmentation of the lungs due to inhalation of smoke and other types of carbon particles is common particularly in residents of large industrial cities. Most marked blackening is produced in coal miners. There is some controversy concerning the question as to whether or not carbon dust of itself can produce a pulmonary fibrosis. There is no conclusive experimental evidence that pure carbon dust will produce fibrosis. It is the author's opinion that all instances of fibrosis in anthracotic lungs are due to an admixture of silica dust in the inhaled air or an inflammatory fibrosis due to respiratory disease. Anthracosis is asymptomatic and produces no clinical findings or disability.

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