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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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xi

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CONTENTS

I. Notes on History.....	1
<i>Fred J. Wampler, M.D.</i>	
II. Methods Employed in the Appraisal and Control of Industrial Health Hazards.....	7
<i>J. J. Bloomfield</i>	
III. Industrial Accidents: Their Cause and Prevention.....	23
<i>Donald E. Cumming, B.S.</i>	
IV. Medical Services in Industry and the Industrial Physician.....	35
<i>Fred J. Wampler, M.D.</i>	
V. The Lay-Out of the Medical Department.....	43
<i>Fred J. Wampler, M.D.</i>	
VI. Governmental Agencies in Industrial Hygiene.....	48
<i>J. J. Bloomfield</i>	
VII. Industrial Health Program of the American Medical Association....	65
<i>C. M. Peterson, M.D.</i>	
VIII. The Effects of Temperature and Humidity on Industrial Workers....	69
<i>Anna M. Baetjer, Sc.D.</i>	
IX. The Effects of Abnormal Atmospheric Pressures.....	91
<i>Anna M. Baetjer, Sc.D.</i>	
X. Light, Lighting and Seeing.....	101
<i>Matthew Luckeish, M.D., and Frank K. Moss, E.E.</i>	
XI. Fatigue.....	133
<i>Lorin A. Thompson, Ph.D.</i>	
XII. The Physical Examination.....	144
<i>Fred J. Wampler, M.D.</i>	
XIII. Medical Control of Industrial Exposure to Toxic Chemicals.....	150
<i>John H. Foulger, M.D.</i>	
XIV. Survey of Substances which Cause Occupational Poisoning.....	180
<i>Willard Machle, M.D., and Francis F. Heyroth, M.D.</i>	
XV. Industrial Lead Exposure and Lead Poisoning.....	202
<i>Robert A. Kehoe, M.D.</i>	
XVI. Carbon Disulfide.....	241
<i>Ralph W. McKee, Ph.D., and J. A. Calhoun, M.D.</i>	
XVII. The Toxicity of Certain Organic Solvents in Industry.....	256
<i>J. M. Carlisle, M.D.</i>	
XVIII. Carbon Monoxide Poisoning.....	269
<i>Frank S. Rossiter, M.D.</i>	
XIX. Some of the Poisonous Gases.....	278
<i>Frank S. Rossiter, M.D.</i>	

XX. Electricity.....	288
<i>Charles F. Engel, M.D.</i>	
XXI. Occupational Diseases of the Skin.....	296
<i>Louis Schwartz, M.D.</i>	
XXII. Pneumoconiosis (Dust Diseases of the Lung).....	345
<i>George Zur Williams, M.D.</i>	
XXIII. Occupation and Tuberculosis.....	375
<i>Sarah I. Morris, M.D.</i>	
XXIV. Venereal Disease Control in Industry.....	394
<i>Otis L. Anderson, M.D.</i>	
XXV. What Industry Can Do to Improve Nutrition.....	409
<i>William A. Sawyer, M.D.</i>	
XXVI. Eyes in Industry: Care and Prevention of Injury.....	421
<i>George H. Cross, M.D.</i>	
XXVII. Traumatic Shock and Burns.....	435
<i>J. M. Carlisle, M.D., Augustus Gibson, M.D., Albert Hemming, M.B., Ch.B., and D. P. Robertson, M.D.</i>	
XXVIII. The Industrial Back.....	458
<i>Rutherford T. Johnstone, M.D.</i>	
XXIX. The Nurse in Industry.....	483
<i>Joanna Johnson, R.N.</i>	
XXX. Compensation.....	498
<i>B. E. Kuechle, A.B.</i>	
XXXI. Vocational and Industrial Rehabilitation.....	507
<i>Richard N. Anderson, M.A.</i>	
XXXII. Industrial Medical Service for the Smaller Plant.....	518
<i>Fred J. Wampler, M.D.</i>	
XXXIII. Women in Industry.....	528
<i>Milton H. Kronenberg, M.D., and Kenneth Mores, M.S.</i>	

ST0853724

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

ST0853726

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.
- 1923—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.

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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 HAZARDS¹*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

ST0853732

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.

ST0853734

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, DAMPNES, 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-

Name of Plant..... Page..... of.....
 Department..... Industry Code and No.....
 Informant's name..... D/W..... S/D.....
 Surveyed by..... Date.....
 Workroom No.....

[illegible]

ST0853736

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, 1934-38*

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 temperature	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.

ST0853738

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.

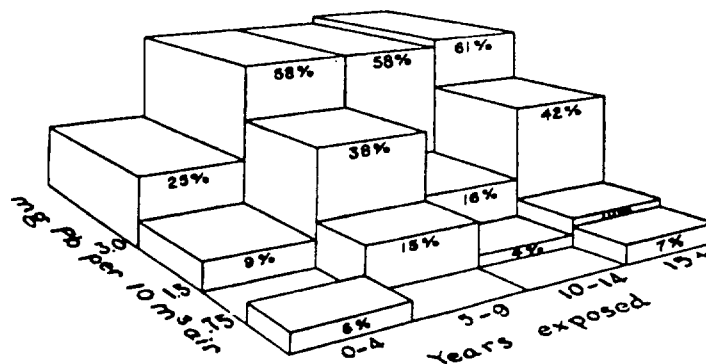


FIG. 1. Percentage of workers with early plumbism. The percentages of storage battery workers in each of 16 exposure groups diagnosed as cases of early plumbism are represented by the height of blocks. Thus, of the workers exposed more than 15 years to atmospheric lead concentrations in excess of 3 mg. 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

ST0853740

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

ST0853742

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	1800 FPM		Reduced concentration to safe level
Grain elevators.....	All tools		1800	Reduced concentration to safe level
	Elevator boot and head, garner		500	Visual test
Paint spraying.....	Spraying booth		50-200	Reduced concentration to safe level
Sand pulverizing.....	Bagging machine	400 FPM		Reduced concentration to safe level
	Horizontal drilling with Kelley trap	60*		Reduced concentration to safe level
Quarrying and mining.....	Vertical drilling with Kelley trap	200*		Reduced concentration to safe level
	Chromium plating	50**	1800	Reduced concentration to safe level
Electroplating.....	Steam and acid tanks	75-100		Reduced concentration to safe level
	Brushing	200*		Usual practice
Hatters' fur.....	Cutting machines	380*		Usual practice
Electric welding.....	Blowers	2000*		Usual practice
	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-

teative 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
	Un- controlled	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

ST 0853744

affected. 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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ST0853746

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:

ST 0853748

- 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.

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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 pneumoconioses 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 pneumoconiosis 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

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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 $\frac{1}{2}$ 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 $\frac{1}{2}$ 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-

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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 or 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

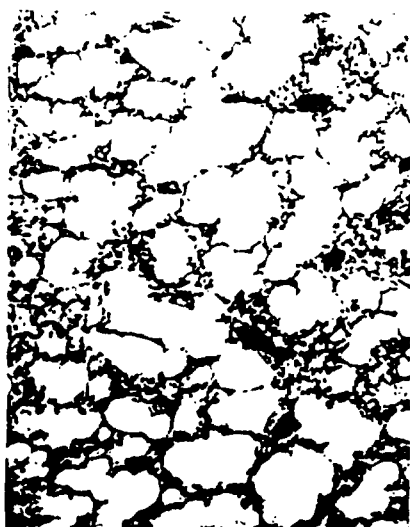


FIG. 1A

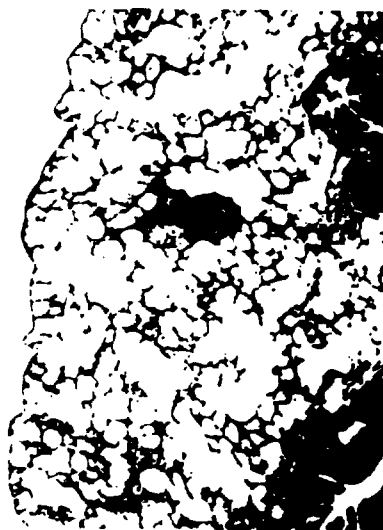


FIG. 1B

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

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

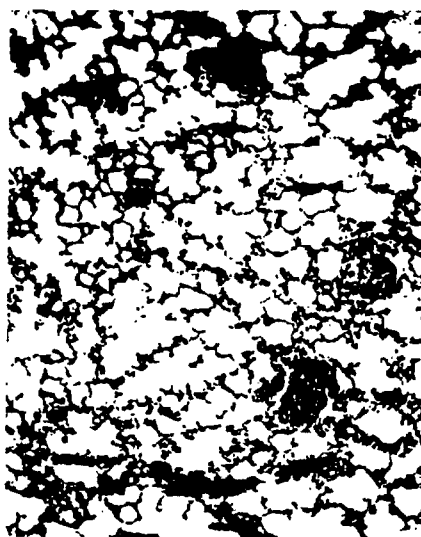


FIG. 1C. ($\times 50$) Guinea pig lung after mild induced respiratory infection. Note proliferating lymphoid nodules in parenchyma

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that following pneumonia or bronchitis, this change occurs by the enlargement of microscopic 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

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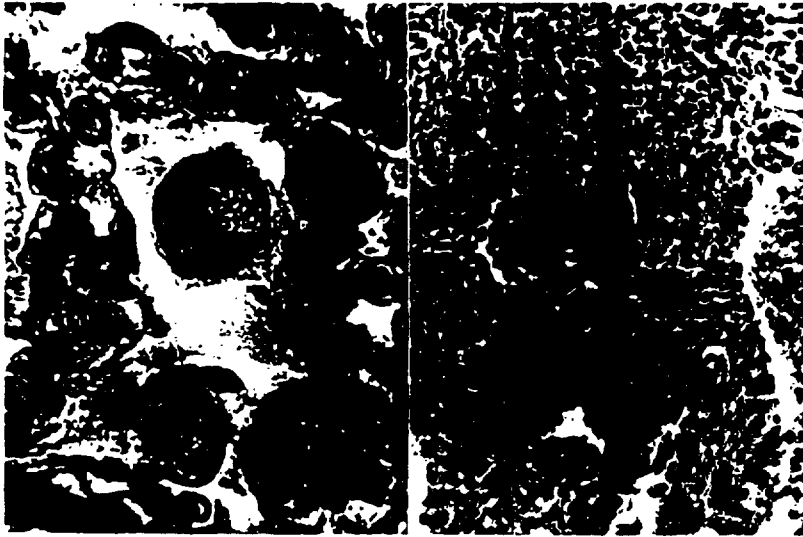


FIG. 2A

FIG. 2B

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

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

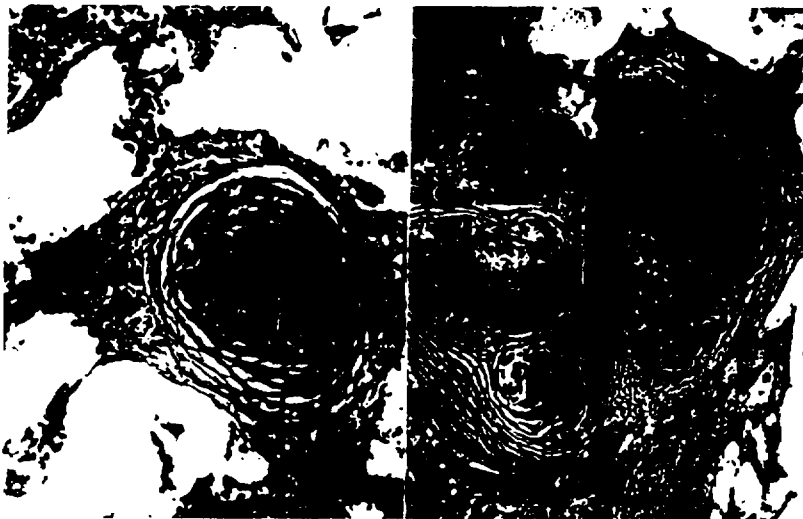


FIG. 2C

FIG. 2D

FIG. 2C. ($\times 50$) Early characteristic silicotic nodule—human lung

FIG. 2D. ($\times 10$) 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

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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 pneumoconiosis 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,

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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

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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

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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 auscultatory 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

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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

ST0853766

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

ST0853767

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

ST0853768

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 cardiovascular 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

ST0853769

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

ST0853770

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, bizarre masses of infected gray foul

ST0853771

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

ST0853772

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.	20 days; may be extended to 60 days.
Kentucky	Workmen's Compensation Board in Department of 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 \$324 a year.

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State	Workmen's Compensation Bureau.	No board; Bureau may appoint special examiner.	General; compulsory.	Compensated.	Unlimited.
North Dakota	Industrial Commission.	Commission shall appoint 3 silicosis referees; findings advisory.	Schedule; compulsory.	Not compensated.	\$200; may be increased in unusual cases.
Ohio	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.
Pennsylvania	Department of Labor and Industries.	No Board; Department may require examinations.	Schedule; compulsory.	Compensated.	May be limited to compensable period for certain disabilities.
Washington	Workmen's Compensation Commissioner.	Board of 3; findings advisory, and reviewable by Commissioner.	Special fund; elective.	Compensated.	\$300; may be increased to \$1,000.
West Virginia	Industrial Commission.	Commission may appoint from panel, or may appoint special examiners; all findings advisory.	General; compulsory.	Compensated.	Limited to compensable period. ¹
Wisconsin					

¹ Constructed 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.

ST0853775

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

ST0853776

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.

ST0853771

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

ST0853778

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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ST0853779

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S10853780

INDEX

- A**
- Abnormal atmospheric pressure, 91-100
 below sea level, 91, 92
 caisson's disease, 93-97
 prevention, 94-96
 symptoms, 93-94
 treatment, 96-97
 effects of, 91-100
 high pressure, mechanical effects of, 92, 93
- Abortion, in benzene poison, 259
- Absenteeism, 537-549
 control of health, safety and welfare facilities, medical care, 540
 married versus single women, 540
 home responsibilities, 540
 national health survey, 537-538
 tables on, 539
 non-occupational conditions, 543
 dysmenorrhea, 544
 fatigue, 545
 menopause, 544
 menstruation, 544
 noise, 547
 nervous manifestations, 548
 occupational illness
 dermatitis, 541
 lead, 543
 organic solvents 543
 radium poisoning, 542
 synovitis of arm, hand, wrist, 542
 women versus men, 537-540
 Boston Edison Co., 537-538
- Absorption of solvent vapors, 249-250
- Acetone, 265, 266
 careful selection of workers, 266
- Accidents, 23-34, 424, 484, 505
 causes of, 26-27
 control, 30
 compensation laws, 484, 505
 costs, 30, 424, 484, 505
 direct, 30, 484
 indirect, 30, 484
 occupational, 23
- Accident proneness, 27-29
 Newholds table for determining, 29
- Acid, 192, 195, 281-283, 430-431
 burns of eye, 430-431
 hydrocyanic, 281-283
 nitric, 283
 phosphoric, 192
 sulphuric, 195
- Actinic causes of cancer, 326
- Acute pneumoconiosis, 356-357
- Acute silicosis, 354-355
- Acute transient inflammatory reaction, 78
- Age, 24, 303
 as factor in accidents, 24
 as factor in skin diseases, 303
- Agricola, 2, 296
- Air conditioning, 87
- Air hose accident to eye, 429-430
- Alameda Medical Society, 521
 in medical education, 521
 in medical service to small plants, 521
- Alcohol methyl, see methanol
- Allergy, 304-306
 factor in skin disease, 304-306
- Alkali burns of eye, 430-431
- Allen, T. D., 425
 on protecting eyes, 425
- Alpers, B. J., 248
 on pathology in CS₂ poison, 248
- Aluminum, 188, 189
- American Association of Industrial Physicians and Surgeons, 4, 5, 66, 518
 committee on education, 66
 committee on nutrition, 409
 results of survey, 409-413
- American Association of Labor Legislation, 4
- American Association of Public Health Nursing, 5, 496
- American College of Surgeons, 4, 5
 committee on industrial medical and traumatic surgery, 4
 minimum standards for medical service in industry, 4
- American Dietetic Association, 414
 training of dietitians, 414
- American Industrial Hygiene Association, 4, 5
- American Medical Association
 Council on Industrial Health, 4, 5, 65-68, 518, 521
 first Congress on Industrial Health, 4
 medical education
 post-graduate, 66
 under graduate, 65, 66, 518
 first symposium Industrial Hygiene and Medicine, 4
 Section on Industrial Hygiene, 4

- American Public Health Association, 4, 346
 section on industrial hygiene formed, 4
 American Red Cross, 33
 first aid training, 33
 American Standards Association, 186, 197, 244
 allowable concentration of CS_2 in air, 244
 on harmfulness of cadmium fumes, 186
 on safe concentration of manganese dust, 197
 Anderson, Otis L., 394
 Anderson, Richard N., 507
 Anemia, 272, 273
 in carbon monoxide poisoning, 272, 273
 Anemia, aplastic, in benzene poison, 259
 Anthracosilicosis, 357-363
 Anthracosis, 373
 Antimony, 190, 193-194
 Aprons, 32, 322
 Appraisal of visual defects in industry, 423
 Argon, 181
 Army, 379
 4 x 5 x-ray film, 379
 Arsenic, 190, 192-193, 329
 as cause of cancer, 329
 as impurity in metals, 192
 symptoms of poisoning, 192-193
 dermatological, 193
 Asbestosis, 372
 Asphyxiation, from electrical shock, 292-294
 theories, 292
 treatment, 292-294
 Association of American Medical Colleges, Industrial Medical Education, 518
 Atmospheric pressure, 91
 Austria, 3, 498
 labor legislation, 3, 498
 Auten, H. L., 427
 care of eye injuries, 427
 Aviation medicine, 172, 179
 Journal of, 171
- B
- Baetjer, Anna M., 89, 91
 Barium, 182, 183
 pneumoconiosis in Italian mill workers, 183
 Baron, 188, 189
 Barth, E., 221
 on accumulation of lead in bones, 221
 Baths, supervised, 322
 as protection against skin irritants, 322
 Beazley, Ada, 487
 1st nursing visit to industrial policy holder, 487
 Bechterew's sign, 476
 Bends, 99
 in caisson workers,
 Benzene (benzol), 259
 acute poisoning
 death from, 259
 signs and symptoms, 259
 chronic form, 259
 abortion in, 259
 signs and symptoms, 259
 cutaneous dryness and cracking, 260
 diagnosis, 259
 effect on girls and pregnant women, 259
 prophylaxis
 blood count weekly, 260
 education of employees, 260
 protective ointments, 260
 synthetic rubber gloves, 260
 ventilation, 260
 urinalysis for sulfates, 260
 treatment
 remove from exposure, 260
 special treatment as necessary, 260
 Benzine, 261
 see gasoline
 Benzol, 259
 see benzene
 Berkeley plan, 521
 in medical education, 521
 in medical service to small plants, 521
 Beryllium, 182
 Bills, Arthur G., 134
 factors influencing mental output, 134
 Bismuth, 190, 194
 Blindness, 264
 wood alcohol poisoning, 264
 Blood in treatment of traumatic shock and burns, 435
 Blood pressure, 155-176
 effect of continued abnormal, 176
 in exposure to toxic chemicals, 155-179
 measurement of, 157
 scoring, 158-160
 registration of score, 160-162
 things affecting, 169-175
 useful levels, 157-158
 Bloomfield, J. J., 7, 48
 Blumer, F. M. R., 193
 on arsenic in hair of normal and exposed persons, 193

ST0853782

- Brieger, H., 248
hematological survey in CS₂ poisoning, 248
- Bristol, Leverett D., 494
on public health nurse, 494
- British X-ray and Radium Protective Committee, 185
on safe exposure to x-rays, 185
- Bromine, 197, 199
- Brown, A. L., 431
on epidermal or mucous membrane grafts following chemical burns of eye, 431
- Bulson, Jr., A. E., 428
on foreign bodies in eye, 428
- Bureau of Labor Statistics, 50, 395
on employment, 395
- Bureau of Mines, 4, 33, 50, 56
functions, 56
first-aid course, 33
- Bureau of Standards, 425
on colored eye protective glasses, 425
- Burns, 441-457
acid, 430
alkali, 430
Berkow's method for estimation of burned surface, 457
electrical, 288
eyeball, 430
infection, 452
thermal burns in major catastrophe, 441-446
anticipation of, 445
- Burn shock, 438-447
- Burns, treatment of, 455
general
amino-acid, 449
barbituates, 448
blood transfusions, 438, 449
chemotherapy
penicillin, 444, 455
sulfa group, 455
morphine, 447-448
oxygen, 448
plasma transfusions, 438, 449
saline, 438, 448
water, immersion in cold, 448
- local
boric acid ointment gauze and pressure dressing, 451
cleansing of surface, 450
envelope method, 454
miscellaneous methods, 454
Pickrell's translucent pliable film, 452
saline bath, 454
- sulfa drugs, 451-452, 455
tanning-tannic acid, silver nitrate, Dymixal, 451
- Burns, treatment orders, 456
emergency, 456
laboratory, 456
routine, 456
vitamin requirements, 456
- C
- Cadmium, 185, 186
symptoms of poisoning, 186
toxicity of fumes, 186
- Cafeterias in industrial plants, 413-417
dietitian in, 414
importance of, 413
operation of, 414-416
- Caisson's disease, 93-97
prevention, 94-96
helium-oxygen mixture, 96
hours of work, 95
inhalation of pure oxygen, 95-96
nitrogen-oxygen mixture, 96
proper selection of personnel, 96
rate of decompression, 94-95
treatment, 96, 97
oxygen-helium mixtures, 97
oxygen-nitrogen mixtures, 97
recompression, 96
- Calcium, 182, 183
- Calcium fluoride, 198
- California, 4, 57, 486, 526
Industrial Accident Commission, 57
law on work for women, 526
nursing consultant, 486
passed early law on reporting occupational diseases, 4
- Calhoun, J. A., 241
- Calvery, H. O., 193
public health aspects of arsenic sprays, 193
- Candle power, 103
- Carbon, 98, 99, 189, 275, 279, 281, 287, 294
carbon dioxide, 189
effects of at high pressures, 98, 99
with oxygen, 275, 279, 281, 287, 294
solid carbon dioxide, 189
solid, freezes and destroys tissue, 189
- Carbon disulphide, 241-255, 264
absorption, 249
clinical manifestations
local, 245
general, 245-246
differential diagnosis, 247
excretion, 249
exposures, 244

ST0853783

- Carbon disulphide—continued
 manufacture, 242
 mechanism of toxicity, 251
 method of determinations, 243-244
 pathological changes, 248
 poisoning, 246
 prevention, 252
 therapy, 253
 properties, 241
 reactions, 241
 use, 242
- Carbon monoxide, 174, 189, 269-277, 281
 absorption, 269
 chronic poisoning, 273
 factors to consider in diagnosing, 274
 elimination, 269
 fatigue and, 174
 prevention, 275
 prognosis, 271
 reacts with metals, 189
 symptoms, 271
 tests for, in blood, 276-277
 tests for, in air, 276
 toxicity at high altitudes, 174
 treatment, 275
- Carcinoma, 184, 327
 in radium workers, 184, 327
 in x-ray operators, 327
- Carlisle, J. M., 256, 435
- Carman, H. F., 425
 on protecting eyes in industry, 425
- Case, E. M., 99
 toxicity of gases at high pressure, 99
- Case finding in syphilis, 401
- Case finding in tuberculosis, 388
- Cellulose xanthate, 241
- Central nervous system, 196, 246, 260, 271
 progressive depression in toluene poisoning, 260
 carbon disulphide poisoning, 246, 247, 248, 265
 carbon monoxide poisoning, 271
 manganese poisoning, 196
- Cerium, 189
- Cesium, 181
- Chadwick, Edwin, 3
 investigated working of poor laws, 3
 secretary of factory commission, 3
- Chemical irritants, 307-308, 383
 in dermatitis, 307-308
 in tuberculosis, 383
- Chemical poisoning, 150-179
 acute
 result of accident, 152
 result of carelessness, 152
 temporary inadequacy of personnel, 152
 chronic, 152-154
 physical examinations in control of, 150-179
- Chilblains, 78
- Children's Bureau, 4, 50
- Chimney sweep's cancer, 296
- China, 326
 kang cancer, 326
- Chlorine, 197, 199, 278-280
 action, 278
 prognosis, 279
 symptoms, 278
 treatment, 280-281 277
- Chromium, 194-195
- Cleanliness, 212-213, 258, 263, 322-323, 552
 personal 258, 263, 322-323
 plant, 212-213, 322, 552
- Cleveland Railway Co., 28
 study of accident proneness, 28
- Clothing, 80, 88, 320-322, 531-532
 change of, 80, 257, 264, 320-321
 protective, 263, 320-322
 work, 320-321, 531-532
 frequent laundering, 320-321
- Coagulation therapy of burns, 453
 disadvantages of, 453
 constriction of blood vessels, 453
 difficulty in detection of infection beneath coagulum, 453
 liability to produce rigid scar, 453
- Cobalt, 197
- Colorado, 526
 law on work for women, 526
- Columbium, 190
- Commission on Industrial Relations,
 Congress authorized appointment, 4
- Committee on Education, American Association of Industrial Physicians and Surgeons, 66
- Committee on Nutrition, 409-413
 American Association of Industrial Physicians and Surgeons, 409
 results of survey, 409-413
- Commonwealth of the Philippines, 391
 all occupational diseases compensable, 391
- Compensation, 498-506
 accident and disease prevention, 505
 compensation laws, 505
 physical examinations, 505
 administration and review, 503
 industrial commissions, 504
 labor departments, 504

ST0853784

- benefits, 501
 - disability, 501
 - death, 502
 - permanent partial, 501
 - permanent total, 501
 - coverage, 499
 - groups not covered, 500
 - maritime and interstate commerce, 500
 - injuries, 500
 - historical
 - Europe, 498
 - United States, 498
 - Wisconsin, 498
 - insurance and security requirements, 504
 - private, 504
 - self-, 504
 - state, 504
 - medical and rehabilitation, 502
 - eye injuries, 424, 433
 - limited in time and cost, 502
 - selection of physician, 502-503
 - rehabilitation, 503
 - objectives, 499
 - Public Health Administrators, 58-59
 - silicosis, 367-371
 - table on, 368-369
 - voluntary election on part of employer, not covered, 500
 - Compressed air illness, 93-97
 - prevention, 94-96
 - helium-oxygen mixture, 96
 - hours of work, 96
 - inhalation of pure oxygen, 95-96
 - nitrogen-oxygen mixture, 96
 - proper selection of personnel, 96
 - rate of decompression, 94-95
 - treatment, 96, 97
 - oxygen-helium mixtures, 97
 - oxygen-nitrogen mixtures, 97
 - recompression, 96
 - Congress, 4, 500
 - authorized appointment of Commission on Industrial Relations, 4
 - jurisdiction over maritime and interstate commerce employment, 500
 - Connecticut, 486, 496, 526
 - law on work for women, 526
 - nursing consultant, 486
 - program, man power conservation, 496
 - Contact finding, 389, 401
 - in syphilis, 401
 - in tuberculosis, 389
 - Control of accidents, 30-34
 - Control of diseases, 211-225, 319-323
 - Converse, J. M., 430
 - on war injuries to eye, 430
 - Copper, 181-182
 - Cost of hiring and dismissal, 421
 - Costs of compensable accidents, 30, 424, 484
 - Congenital defects, 465-467
 - in industrial back conditions, 465-467
 - correction of, 487
 - Craft guilds, 2-3
 - Cross, George H., 421, 428
 - Cumming, Donald E., 23
 - Curriculum Guide for Public Health Nursing, 496
 - Cyanogen, 281-283
 - chloride, 281-283
- D
- Dean, H. T., 199
 - on mottling of teeth, 199
 - Defense, Health and Welfare, Office of, 5
 - Committee on Health and Medicine, 5
 - Subcommittee on Industrial Health and Medicine, 5
 - Demianoff's sign, 476
 - De morbis artificum diatriba*, 2, 296
 - Delaware law on work for women, 526
 - Denny, J. J., 188
 - on exposure to aluminum dust, 188
 - Department of Agriculture, 531
 - booklet on work clothing, 531
 - Department of Interior, 4, 33, 50, 56
 - Bureau of Mines, 4, 33, 50, 56
 - Department of Labor, 50, 56, 395, 433, 504
 - see U. S. Department of Labor
 - Dermatitis, 541-542
 - in industries using organic solvents, 257-258
 - in women, 541-542
 - Dermatoses investigation, methods of, 330-341
 - contact and explain to superintendent, 330
 - examine medical records, 331
 - inspect plant, 331
 - inspect workers, 331
 - make card record, 332
 - perform patch test, 332-341
 - de Vries, W. M., 328
 - scrotal cancer in briquette workers, 328
 - Diastolic blood pressure in exposure to toxic chemicals, 155-179
 - difference between two arms, 158
 - normal limits, 158

- Diet, 302, 262
 - as factor in dermatitis, 302
 - high protein, high carbohydrate in tetrachlorethane poison, 262
 - Dietary supplements, 171, 233-234, 418-419
 - in lead industry, 233-234
 - vitamins in, 418-419
 - in exposure to toxic chemicals, 171
 - Diethylene oxide, 266
 - Dimethyl ketone, 265-266
 - Dioxan, 266
 - Direct costs of accidents, 30, 484
 - Direct lighting, 104
 - Disability benefits, 501
 - death, 502
 - permanent partial, 501
 - permanent total, 501
 - Disease, toxic exposure and, 174
 - Diseases of skin. See occupational diseases of skin, 296-341
 - District of Columbia, 391
 - all occupational diseases compensable, 391
 - Division of Industrial Hygiene, 9 61, 62
 - four major fields in war effort, 61
 - Division of Labor Standards, 50, 56
 - Drinker, C. K., 274
 - CO poisoning in metropolitan New York, 274
 - Dry ice, 189
 - Dublin, L. I., 23, 376
 - death rates from accidents in America vs. Canada and England, 23
 - Dunn, Mary J., 496
 - curriculum guide for public health nursing, 496
 - Dust concentration
 - safe limits in granite cutting, 4
 - Dust diseases, 345-374
 - Dysmenorrhea, 544
- E
- Eastman Kodak cafeterias, 414
 - Economic importance of visual disability, 424
 - Education of ophthalmologists, 422
 - Education program
 - nutrition, 419-420
 - prevention of industrial backache, 478
 - tuberculosis control, 389
 - venereal disease control, 402
 - Effects of abnormal atmospheric pressures, 91-99
 - increase below water, 91-92
 - mechanical effects of high pressure, 92-93
 - Effects of temperature and humidity on workers, 69-88
 - high temperatures
 - effects on health, 79-81
 - acute respiratory diseases, 80-81
 - tuberculosis, 383
 - effects on work and accidents, 81-83
 - endurance table, 86
 - recommended for industry, 83-88
 - air conditioning, 87
 - air movement, 85
 - toxic exposure and extremes of low temperatures, 173-174
 - acute transient inflammatory reaction, 78
 - chilblains, 78
 - frost bite, 78, 79
 - occurrence, 78
 - treatment, 79
 - Electricity, 288-295
 - injuries produced by, 288, 432
 - Elimination of solvent vapors, 250
 - Ellenbog, Ulrich, 2
 - Ely's sign, 476
 - Emotional factor, 148, 536, 549
 - consider in physical examination, 148
 - in accidents, 536
 - in fatigue, 549
 - women vs. men, 536
 - Employees of Federal government, 500
 - compensation for, 500
 - Employer resistance to using disabled, 512
 - Employers Mutual of Wisconsin, 487-488
 - program within industry, 488
 - services outside industry, 488
 - Employment policy and venereal disease control, 404
 - Engel, Charles F., 288
 - England, labor legislation, 3, 498
 - Everts, Glenn S., 518
 - medical service to small plants, 518
 - Exposure to toxic chemicals, early effects of, 154-156
 - Eye accidents, 421-433
 - air hose, 429-430
 - burns
 - acid, 430-431
 - alkali, 430-431
 - compensation, 424, 433
 - concussion, 429
 - contusion, 429
 - costs, 424

factors in, 425
 foreign bodies, 429
 in cornea, 426
 intra-ocular, 427
 tolerance to, 428
 prevention of, 424
 the problem, 421-422, 424
 Eyes, care and prevention of injury,
 421-433

F

Factors in accident production (eye), 425
 Factory Act (England), 3
 Fair Labor Standards Act, 527
 on employment of women, 527
 Family physician, 37, 145, 518
 Fatigue, 133-143, 154, 155, 174, 254, 382,
 425, 547-548
 avoid in CS₂ exposure, 254
 definition, 133
 factor in accident production, 138, 425
 length of work week and work day,
 136-138
 measures against
 change of pace, 133
 favorable environment, 139
 food, 133
 lighting, 139
 rest pauses, 133
 production curves, 138
 psychological factors, 133, 135
 boredom, 141
 foremen, like or dislike, 139
 loyalty to company, 139
 monotony, 141
 noise, 135, 547-548
 reaction to rules, etc., 139
 wage incentive, 140
 tuberculosis and, 382
 unnecessary toxic exposure and, 174
 women, 547-548
 Federal Employer's Liability Act, 500
 compensation in maritime and inter-
 state commerce, 500
 Federal Government, 500
 administers compensation acts:
 Federal Employer's Liability Act,
 500
 Longshoremen's and Harbor
 Workers' Compensation Law,
 500
 Merchant Seamen's Act, 500
 employees, of, 500
 First aid personnel training, 33
 for eye protection, 423

First aid training, 33, 423
 American Red Cross, 33
 Bureau of Mines, 33
 for eye accidents, 423
 First nursing visit to industrial insurance
 policy holder, 487
 Fluorine, 197-199
 Footcandle, 103
 recommendations for more difficult
 tasks, 124
 table of recommendations, 123
 Footcandle scale of effectiveness, 120-122
 Footlambert, 103
 Foreign bodies in eye, 427
 locating by electro-magnet, 427
 Foulger, John H., 74, 150
 and Weaver, W. L., 74
 vitamins in control of heat cramps and
 heat prostration, 74
 France, 498
 Frequency rate (industrial accidents),
 24-25
 Frostbite, 78, 79
 treatment of, 79
 Fungi in skin diseases, 312-313, 320
 active treatment in organic solvent
 plants, 258
 infections of foot, prevention, 320

G

Galen, 1
 Gallium, 189
 Gasoline, 261-262, 273
 anaesthetic effect, 261
 dermatitis, 261
 prophylaxis, 261
 protective clothing, 261
 ventilation, 261
 treatment, 262
 Gehrman, G. H., 328
 on papillomata and carcinoma of
 bladder, 328
 General plus lighting, 104
 General practitioner, 37
 relation to industrial physician, 37
 Georgia, 486
 nursing consultant, 486
 Germanium, 190
 Germany, labor legislation, 3, 498
 Gibson, Augustus, 435
 Glare, 103, 125-129
 Gloves, 32, 322
 Gloyne, S. R., 372
 roentgenological appearances of as-
 bestosis, 372
 Goenslen's sign, 476

- Goggles, 32, 435
in protection against eye accident and injury, 32, 435
- Gold, 181, 182
- Gordy, S. T., 246, 247
symptomatology of chronic CS₂ poisoning, 246, 247
- Governmental agencies in industrial hygiene, 48-64
state and local health departments, 52-60
functions, 53-54
chemical, 54
engineering, 54
medical, 54
nursing, 54
integrated service, 54-56
interdepartmental relationship, 56-58
- Greenburg, L., 424
economic importance of visual disability in industry, 424
- Greenwood, D. A., 199
on mottled teeth, 199
- H
- Hafnium, 190
- Hair, long, 532
hazard to women workers, 532
- Haggard, H. W., 249
on absorption of toxic vapors, 249
- Hamilton, Alice, 244, 246, 540
on CS₂ exposure, 244, 246
on susceptibility of young women to poisons affecting nervous system, 540
- Haldane, J. B. S., 99, 272
effects of gases under high pressures, 99
on oxygen consumption, 272
- Harmon, F. L., 135
- Harrower, J. R., 244
procedure for determining CS₂ in blood, 244
- Health and Morals Act, 3
- Health facilities in control of absenteeism, 540
- Heim, J. W., 174
toxicity of CO at high altitudes, 174
- Helium, 181
caisson work, 93, 96
oxygen mixture, 96
- Hemming, Albert, 435
- Henderson, V., 249
on absorption of toxic vapors, 249
- Heyroth, F. F., 180
- Hippocrates, 1
- Hoffman, Frederick L., 204
occupational distribution of cases of plumbism, 204
- Holland tunnels, 274
CO concentration, 274
- Home responsibilities of married women, 540
- Houghten, F. C., 87
on body temperatures, 87
- Hours of work, 136, 535
fatigue and, 535
production and, 136, 535
- Housekeeping, 9, 212, 322, 308, 552
necessity for good, 322, 552
plant construction and, 212
- Homard, John, 3
inspection and reporting, 3
- Humidity, high, effect on industrial worker, 86-87
- Hunter, A. W., 244
procedure for determining CS₂ in blood, 244
- Hydrocyanic acid, 281-283
action, 282
after effects, 283
post-mortem findings, 283
prevention, 283
treatment, 283
- Hydrogen fluoride, 197-198
acute poisoning, 197-198
symptoms, 197-198
chronic poisoning, 198
roentgenological findings, 198
safe limits, 198
- Hydrogen sulphide, 285-287
prevention of poisoning, 286
symptoms sub-acute poisoning, 286
gas eyes, 286
symptoms acute poisoning, 286
paralysis of sense of smell, 286
toxicity, 285
treatment, 287
- I
- Idaho, 57-58, 486
law forming Bureau of Industrial Hygiene, 57-58
nursing consultant, 486
- Illinois, 486
- Illumination, 103, 110, 111, 112, 122, 425
poor, and accidents, 425
precision and production, 112
recommended levels of, 122
speed of vision and, 111
type size and visibility, 111
visual acuity and, 110

- Indiana, 486, 526, 529
 accidents to women, 529
 law on work for women, 526
 nursing consultant, 486
 Indium, 189
 Indirect costs of accidents, 30, 484
 Indirect lighting, 104
 Industrial accidents, their cause and prevention, 23-34, 136, 421-434
 causes of
 accident proneness, 27
 environmental factors, 28
 individual factors, 27
 length of work day, 136
 control, 30-34
 costs, 30, 484
 emergency care, 33
 eye, 421-434
 factors influencing, 23
 rates by industries, 24, 25
 records, 29
 safety committee, 32
 safety supervisor, 32
 Industrial back, 458-482
 backache:
 causes of, 463
 congenital defects:
 articular facets, 467
 horizontal sacrum, 467
 failure of fusion, 465
 sacroization, 465
 spondylolisthesis, 466
 spondylolysis, 466
 herniated nucleus pulposus, 469
 infection, 473
 sacroiliac slip, 469
 visceral disease, 464
 examination for, 473
 history, 474
 incidence, 458
 physical examination, 475
 signs
 Bechterew's, 476
 Demianoff's, 476
 Ely's, 476
 Gaenslen's, 476
 Lasague's, 476
 Linder's, 476
 Neri's, 477
 Ober's, 476
 Soto-Hall's, 477
 x-ray, 477
 other laboratory, 478
 treatment
 educational campaign in prophylaxis, 478
 specific treatment, 479
 correction of congenital defects, 487
 eucipin in oil, 480
 exercise, 479
 manipulation, 480
 medicolegal aspects, 482
 operative, 487
 physiotherapy, 479
 Industrial cleansers, 323
 Industrial Commissions, 504, 507
 Industrial diseases, 4, 296-341, 367-371, 500-501
 compensation, 367-371, 500-501
 dermatoses, 296-341
 first American Congress, 4
 Industrial engineer, 21
 function, 7, 8, 35, 54
 in state and local bureaus, 54
 Industrial Health, 65-68
 program of the American Medical Association, 65-68
 purpose of, 65
 Industrial health clinics, 519
 smaller industries, 519
 Industrial health hazards, methods employed in appraisal and control, 7-22
 appraisal of
 study of workroom environment, 8
 detailed survey, 14-16
 reconnaissance survey, 8
 accident protection, 8
 exposure to specific poisons, 8
 fire protection, 8
 housekeeping, 9
 illumination, 8
 individual occupation, 9
 ventilation, 8, 9
 control of
 isolation of hazardous process, 17, 212
 local exhaust ventilation, 17
 respiratory protection, 18
 substitution, 16
 wet method, 17
 Industrial housekeeping, 9, 212, 552
 Industrial hygiene:
 development of, in U. S., 49-51
 governmental agencies in, 48-64
 organization, 51
 problem, 48, 49
 control of health hazards, 16-20
 survey, forms of, 10, 11, 12
 survey, reconnaissance, 8-14
 survey, detailed, 14-16
 war effort and, 61-63

- Industrial hygiene divisions, 9, 52-54, 62, 387, 402
 - National Institute of Health, 9, 62
 - state and local health departments, 52-54, 387, 402
 - portable x-ray, 387
 - tuberculosis control, 387
 - venereal disease control, 402
 - Industrial medicine, 1-6, 49-51, 296, 399, 448, 498-506
 - cost of, 399
 - financial saving, 399
 - history, 1-6, 49-51, 296
 - Bureau of Mines, 50
 - Craft Guilds, 2
 - Department of Labor, 50
 - Division of Industrial Hygiene, 5, 49-50
 - federal and state legislation, 5
 - labor legislation, 3, 448, 498-506
 - non-official agencies, 50-51
 - organized medicine, 5, 65-68
 - magazine Industrial Medicine began publication, 4
 - Industrial medical education, 5, 65-66, 422, 518, 520-522
 - better, 65
 - ophthalmologists, 422
 - postgraduate, 5, 35, 519
 - Council on Industrial Health, 5, 66
 - American Association of Industrial Physicians and Surgeons, 65
 - undergraduate, 5, 35, 518
 - Council on Industrial Health, 5, 65-66
 - American Association of Industrial Physicians and Surgeons, 65
 - Industrial physician, 35, 40, 79, 248
 - attributes, 37
 - duties, 37, 217-218, 253
 - education, 35, 36
 - function, 7, 21, 150-151, 162
 - health education, 41
 - office equipment, 46
 - place in industrial set-up, 40
 - preparation, 35-36
 - relation to family physician, 37
 - relation to foremen, 36
 - relation to management, 36
 - relation to workmen, 37, 216
 - relation to state health department, 37
 - relation to state industrial commission, 37
 - relation to state rehabilitation commission, 37
 - responsibilities, 36, 58
 - Industrial workers, 69-90, 216, 395
 - effects of temperature and humidity on, 69-90
 - high temperature, 70-77
 - heat cramps, 72
 - heat exhaustion, 74
 - heat prostration, 74
 - heat stroke, 75
 - sun stroke, 75
 - low environmental temperatures, 77-79
 - acute transient inflammatory reaction, 78
 - chilblains, 78
 - frost bite, 78-79
 - number of
 - men, 395
 - women, 395
 - relation to plant physician, 216
 - Infections contributing to tuberculosis, 382
 - Infection of injuries, precautions against, 33
 - Injuries by electricity, 288-294
 - burns, 288
 - eye, 294, 432
 - factors in, 290
 - flashes, 288
 - glare, 288
 - morbid anatomy, 289-290
 - resuscitation, 292-294
 - shock, 288, 293
 - symptoms, 291
 - treatment, 292
 - International Labor Office, 4
 - U. S. joined, 4
 - International Ladies Garment Workers Union, 491
 - Intra-ocular foreign bodies, 427
 - non-magnetic steel, 427
 - Iodine, 197, 199
 - Iridium, 199
 - Iron, 189, 199
 - carbonyl, fatal poisoning, 189
 - Irwin, D. A., 188
 - on exposure to aluminum dust, 188
 - Isolation of hazardous processes, 17, 212
 - Italy, 498
 - Ivy, A. C., 545
 - fatigue in industry, 545
- J
- Japan, 326
 - lung cancer, 326
 - Jewelry, 532
 - hazard in industry, 532

Jirouch, E. A., 266
 on toxicity of butyl ethylene glycol, 266
Journal of Aviation Medicine, 171
Journal of Industrial Hygiene, 4
 established, 4
 Johnson, Joanna, 483
 Johnstone, Rutherford T., 458

K

Kansas, 526
 law on work for women, 526
 Kashmir, 326
 kangri cancer, 326
 Kehoe, Robert A., 202
 Kentucky, 486
 nursing consultant, 486
 Keratitis, occupational, 432
 Keratoconjunctivitis, 431
 Koch, Sumner, L., 450
 treatment of burns, 450
 Kronenberg, Milton H., 523
 Kuechle, B. E., 498
 Kuhn, H. S., 423
 appraisal of visual defects in industry, 423

L

Labor, 4, 387, 392, 491, 504, 524
 American Association of Labor Legislation, 4
 attitude, 39
 Bureau of, created, 4
 Child Labor National Committee, 4
 Department of, 504
 First Federal Statute passed, 4
 nursing programs, 491
 organized, 524
 responsibility, 392
 unions provide health service, 387
 Labor legislation, 3, 4, 498, 500, 527, 529
 Austria, 498
 England, 3, 498
 Federal Conference, 4
 Federal legislation, 527
 Fair Labor Standards Act, 527
 Walsh-Healy Act, 527, 529
 France, 498
 Germany, 3, 498
 Italy, 498
 Russia, 498
 United States, 498
 California, New Jersey, New York, 3, 4
 Wisconsin, 498-500

Laboratory, 44
 in medical lay-out, 44
 Laboratory technicians, 40, 46
 Laird, Donald A., 135
 noise adaptations, 135
 Lasague's sign, 476
 Latka, A. J., 23
 death rates, accidents, 23
 Lead exposure, industrial, 202-240
 control, 211-225
 engineering, 211-214
 construction of building, 212
 equipment location, 212
 housekeeping facilities, 212
 localize toxic processes, 212
 medical facilities, 213
 sanitary facilities, 213
 locker rooms, 213
 lunch rooms, 213
 toilet rooms, 213
 wash rooms, 213
 ventilation, 212
 medical, 214
 medical work and facilities, 215
 medical records, 217
 periodic examinations, 216
 plant physician, 217-218
 pre-employment examinations, 216
 significance of observations
 lead analyses, 218
 in blood and urine, 220-225
 lead line, 219-220
 normal blood metabolism, 220
 stippling of erythrocytes, 218
 detection, 206
 history of, 226
 measurement of, 208
 air analysis, 208
 blood analysis, 209
 urine analysis, 209
 feces analysis, 209
 safe, 210
 Lead poisoning, industrial, 15-16, 190, 202-240
 clinical picture, 226
 diagnosis, 226
 differential diagnosis, 232
 industrial, 225
 laboratory findings, 218, 230-232
 management after recovery, 236
 occurrence in industry, 202
 prophylaxis, 233-34
 symptoms, 226
 tetraethyl lead, 261
 treatment, 234

- Length of work day and work week
 accident production, 136
 fatigue, 136-138
 production, 137-138
 Leukopenia, 259
 in benzene poisoning, 259
 Lewey, F. H., 246, 247
 symptomatology of chronic CS₂ poison-
 ing, 246, 247
 Light, lighting and seeing, 101-132
 Lighting, 102, 104, 123-124, 425
 American practice, 123-124
 benefits of, 102
 direct, 104
 general plus, 104
 indirect, 104
 poor, factor in accidents, 425
 quality of, 123, 129
 science of, 101
 semi-direct, 104
 semi-indirect, 104
 supplementary, 104
 systems of, 103
 Lighting, systems of, 103, 104
 Lighting terms and units, 103-130
 brightness, 103
 candle power, 103
 diffuse reflection-factor, 103
 footcandle, 103
 footlambert, 103
 glare, 103, 125-129
 illumination, 103
 reflection factor, 103
 specular reflection-factor, 103
 Linder's sign, 476
 Lithium, 181
 Local exhaust ventilation, 17, 212, 284
 Locker, 213, 551
 double or separate for street and work
 clothes, 213, 551
 Locker rooms, 213, 551
 Longshoremen's and Harbor Workers'
 Compensation law, 500
 compensation in maritime employ-
 ment, 500
 Lost time due to illness, 80-81
 acute respiratory diseases, 80-81
 Luckeish, Matthew, 101
 Luckeish-Mass Visibility Meter, 115
 Luckeish-Taylor Brightness Meter, 107
 Lunch rooms (cafeterias), 213, 413-414

 M
 Machle, Willard, 180, 198
 fluorine storage in bones, 198
 Magnesium, 182-183
 Malnutrition, 382
 contributing to tuberculosis, 382
 Manganese, 196-197
 Mantoux test, 389-389
 student health services, 384
 Manufacturers Associations
 Connecticut, 496
 National, 399
 Maritime and interstate commerce com-
 pensation coverage, 500
 Married women, 546-547
 home responsibilities, 546
 night and shift work, 546-547
 Maryland, law on work for women, 526
 Masks, 213, 259
 not desirable method of control, 214
 Massachusetts
 code—safe limit on hydrogen fluoride,
 197
 inspector's right to enter factories, 4
 law on work for women, 526
 Massachusetts General Hospital, 446
 Masurium, 196
 Matthewson, S. B., 140
 wage incentive, 140
 Mayer, L. L., 422
 on ocular service for industry, 422
 Mayo, Elton, 137
 rest pauses and length of day, 137
 McConnell, W. J., 50, 520
 medical care for small plants, 520
 non-official agencies in field of indus-
 trial health, 50
 McKee, Ralph W., 241, 244
 Medical care in the control of absen-
 teeism, 540
 Medical department, 43-47
 equipment, 46-47
 floor plans, 43-46, 213
 layout of, 43-47
 Medical occupation and tuberculosis,
 383-385
 Medical personnel in industry, 35-42
 industrial physician, 35-40
 insurance physician, 39
 nurses, 40
 specialists, 39
 technicians, 40
 Medical records, 46-47, 146-147, 215, 217,
 332, 403, 490
 in compensation court, 47
 in tuberculosis control, 389-390
 in research, 41
 necessity for, 47, 391
 nurse and, 490
 space for, 46-47

- Medical services for the smaller plant,
 489, 518-522
 industrial health clinic, 519
 medical training and, 518-519
 medical service plans
 Alameda Medical Society, 521
 Berkeley, 521-522
 New Haven, 520-521
 New Haven physicians, 520
 Philadelphia, 520
 University of California, 521
 Yale School of Medicine, 520
 nursing, 489
 Medical service in industry, 35-40, 41, 42,
 473, 483-497, 518-522
 dental service, 42
 health education, 41
 industrial health clinic, 473, 519
 nurse in, 483-497
 paying for, 40
 physicians in, 35-40, 519
 research, 41
 smaller plants, 518-522
 women physicians, 42
 Medical students
 tuberculosis in, 384-385
 undergraduates in industrial medical
 education, 518
 Medication, improper, 171
 toxic exposure and, 171
 Melanosis, 315
 Menopause, 544
 Mental hygiene in industry, 495
 nurses part in, 495
 Menstruation, 544
 Merchant Seamen Act, 500
 Compensation in maritime employ-
 ment, 500
 Merewether, E. R. A., 372
 roentgenological appearances of asbes-
 tosis, 372
 Mercury, 185, 186-188
 safe concentrations of vapor, 188
 uses, 187, 188
 Mercury poisoning
 from inhalation of vapor, 186-187
 in fur and felting industry, 187
 kidney damage in, 187
 nephrosis in, 187
 symptoms of, 187
 Methanol (wood alcohol), 264-265
 absorption
 gastro-intestinal tract, 264
 inhalation, 264
 skin, 264
 action
 accumulative, 264
 eye, 264
 irritant to skin, 264
 prophylaxis, 264
 protective clothing, 264
 ventilation, 264
 wash from skin, 264
 symptoms, 264
 treatment, 264-265
 Methyl chloride, 265
 Metropolitan Life Insurance Company,
 28, 376, 380, 387, 487
 nursing service, 487
 study of accident proneness, 28
 Michigan, 486
 nursing consultant in industrial hy-
 giene, 486
 Minton, J., 421
 eye accidents in London, 421
 Missouri, 486
 nursing consultant in industrial hy-
 giene, 486
 Molybdenum, 194, 195
Morbis Metallious, 296
 Morgan, J. J. B., 135
 distractions on performance, 135
 Morris, H. P., 221
 on lead accumulation data, 221
 Morris, Sarah I., 375
 Morse, Kenneth, 523
 Moss, Frank K., 101
 Mule spinners cancer, 329
 Mumford, Eleanor W., 486
 nursing services for eyes in industry,
 486
 Myers, C. S., 140
 monotony *versus* fatigue, 140

 N
 Narcotic effect of, 190, 259, 263
 benzene, toluene and xylene, 259
 nitrogen, 190
 trichlorethylene, 263
 National Association of Manufacturers,
 399
 on cast of medical program, 399
 National Committee on Conservation of
 Man Power in Defense Industries, 5
 National Conference of Governmental
 Industrial Hygienists, 492
 National Health Survey, 537-538
 National Industrial Conference Board,
 525
 training for industrial work, 525
 National Institute of Health, 5, 9, 51, 485

- National Organisation for Public Health
Nursing, 485, 488, 489
section on industrial nursing, 485, 488
- National Safety Code, 4
protection of heads and eyes of industrial workers, 4
- National Safety Council, 4, 5, 425, 494, 496, 511, 521
committee on benzol established, 4
- National Silicosis Conference Report on Medical Control, 346
- National Society for the Prevention of Blindness, 486
eye problems, 486
- National Tuberculosis Association, 379
Navy, 379
35 mm. x-ray film, 379
- Naphtha (see gasoline), 261-262
naphtha jag, 261
- Neal, P. A., 188
studies on mercury vapor concentrations, 188
- Nebraska, 526
law on work for women, 526
- Necrosis of jaw, 191, 327
in phosphorus workers, 191
in radium workers, 327
- Neon, 181
- Neri's sign, 477
- Neuritis of the arm, hand and wrist, 542
- Neuro-psychiatrist, 248
- New Jersey, 4, 486, 526
certain occupational diseases compensable, 4
law on work for women, 526
nursing consultant, 486
- New Hampshire, 526
law on work for women, 526
- New Haven Industrial Medical Service, 520
- New Haven plan, 520
medical care for small plants, 520
- New York, 526
law on work for women, 526
- New York City, 186, 379
mass survey, 379
sanitary code, 186
- Nickel, 199
carbonyl, fatal poisoning, 189
- Nightingale, Florence, 483, 494
- Night work, 546-547
factor in high labor turnover, 547
laws on, 526-527
men and, 547
women and, 526-527, 546-547
- Nitric acid, 283
- Nitrogen, 97, 190, 283-285
narcotic effects at high pressure, 97
nitrous fumes, 283-285
nitrous oxides, 283-285
prevention of poisoning, 284
symptoms of poisoning, 284
treatment of poisoning, 284-285
- Noise, 139, 536, 547-548
in absenteeism, 545
in fatigue production, 139
nervousness in women, 536
- Non-occupational illness factors, 543-549
in absenteeism, 543
- North Carolina, 57, 486
industrial occupational disease law, 57
nursing consultant, 486
- North Dakota, 526
law on work for women, 526
- Nuck, 246-247
symptoms from CS₂ exposure, 246-247
- Nucleus pulposus (herniated), 469-473
description of case, 471
diagnosis, 471-473
differential, 472-473
location, 470
pain in, 471-472
- Nurse in industry, 40, 483-497
Dunn, Mary J., 496
eye problems and, 486
first nurses employed in industry, 4
functions of
in governmental bureaus, 54
home accidents, 494
insurance companies, 487
opportunity for advancement, 496
part in safety program, 485, 494
psychic readjustment, 390
post-graduate preparation, 496
Proctor Marble Co., 487
qualifications, 488
relation to public health nursing, 484
U. S. Public Health Service, 485
relationship to physician, 493
Wanamaker Department Stores, 487
- Nursing consultants, 486
Bureaus of Industrial Hygiene, 486
functions of, 486
National Society for Prevention of Blindness, 486
states having, 486
- Nursing programs for small industries, 489-493
Employers Mutual, 487
Henry Street, 490
in the Berkeley plan, 521
in the New Haven plan, 520

- in the Philadelphia plan, 320
- Metropolitan Life Insurance Co., 487
- nutritionist and, 490
- Pittsburgh Public Health Nursing Association, 491
- under supervision of physician, 492
- Visiting Nursing Association of Detroit, 490
- Nursing students, 384
- tuberculosis in, 384
- Nutrition:
 - adequate in CS, exposure, 254
 - toxic exposure and, 170-171
 - visiting nurse program, 490
- Nutrition, what industry can do to improve, 409-420
 - between meal snacks, 414, 418
 - cafeteria, 418-417
 - attractiveness, 413
 - dietitian to supervise, 414
 - efficient serving, 415
 - non-profit basis, 414-415
 - study problems, 415
 - educational program, 419-420
 - lunch box, 416
 - contents, 417
 - result of survey, 409
 - sufficient time to eat, 415
 - vitamin supplements, 171, 233, 418-419
- O
- Ober's sign, 476
- Ocular service for industry, 422
- Occupational Diseases, 4, 58
 - first clinic, 4
 - first laws for compulsory reporting, California and New York, 4
 - first laws for compensation of, New Jersey, 4
 - first clear cut law compensation, Wisconsin, 4
 - reporting of, 58
- Occupational keratitis, 432
- Occupation and tuberculosis, 375-393
 - compensation for, 390-392
 - contributing factors:
 - age and sex, 380
 - chemical irritants, 383
 - dusts, pneumoconiosis and silicosis, 383
 - fatigue, 382
 - heat and humidity, 383
 - heredity and race, 380
 - infections, 382
 - malnutrition, 382
 - trauma, 382
 - general discussion, 376-379
 - general picture, 375-376
 - in medical occupation, 383-385
 - in medical students, 384-385
 - nurses, 384
 - other service groups, 385
 - responsibility for, 390
 - tuberculosis in industry, 385-390
 - control of, 387-390
 - adequate records, 389
 - case finding, 388
 - contact finding, 389
 - diagnosis, 388
 - health education, 389
 - health supervision, 390
 - know problem in community and plant, 388
 - rehabilitation, 390
 - safeguard workers, 388
- Occupational diseases of skin, 296-341
 - cancer, 296
 - actinic causes, 326
 - aniline, 328
 - arsenic, 329
 - chimney sweeps, 296
 - mule spinners, 329
 - petroleum, 328
 - prevention, 329-330
 - tar, 327
 - trauma, 326
 - causes, 300-313
 - predisposing, 300
 - age, 303
 - allergy, 304-306
 - diet, 302
 - lack of cleanliness, 303
 - other skin diseases, 303
 - perspiration, 302
 - race, 301
 - season, 303
 - sex, 303
 - actual, 306
 - biological agents, 312
 - bacterial infections, 312
 - fungi, 312-313
 - chemical, 307
 - inorganic irritants, 308
 - organic irritants, 308
 - sensitizers, 309-313
 - diagnosis, 314
 - appearance of lesion, 315
 - history, 314
 - melanosis, 315
 - site of eruption, 315

- Occupational diseases of skin—continued
 differential diagnosis, 315
 chronic eczema, 317
 fungus infections, 317
 patch tests, 316
 historical, 296-299
 incidence, 299
 prevention
 cleanliness, 322
 clean plant, 322
 clean machinery, 322
 clean workers, 323
 select safe industrial cleanser, 323
 pre-employment examinations, 319
 protective clothing, 320
 cotton, 321
 impervious material, 321
 leather gloves, 322
 synthetic material, 322
 protective ointment, 323
 classes, 324
 properties, 324
 ventilation, 320
 symptoms, 314
 table of, 336-340
 treatment, 318-319
 Occupational poisoning, survey of substances, 180-201
 Official Agencies, responsibilities of, 58-60
 Office of Defense, Health and Welfare, 60
 Ohio, 486, 526
 law on work for women, 526
 Oklahoma, law on work for women, 526
 Ophthalmologists, 421, 422, 423, 427, 429, 430
 duties of, 423
 education of, 422
 Oregon, 526
 law on work for women, 526
 Organic solvents in industry, 256-268
 effects on skin, 257-258
 effects on system, 258-259
 general considerations, 256-259, 266-268
 signs and symptoms, 259
 toxicity of 256-268
 Organic solvents, toxicity, 256-268
 action, 256-259
 effects on skin:
 dermatitis, 257
 prevention:
 aprons, 257
 cleanliness, 257
 gloves, 257
 protective clothing, 257
 protective ointments, 258
 containing chemicals of value against allergens, 258
 containing a light screen, 258
 containing neutralising chemicals, 258
 to facilitate removal of irritant, 258
 to prevent irritant from touching the skin, 258
 pre-placement examinations, 258
 don't use people with acne, 258
 don't use people with allergic eczema, 258
 don't use people with athlete's foot, 258
 don't use people with history of dermatitis, 258
 systemic effects:
 clinical signs and symptoms, 259
 central nervous system, 259
 effects on liver, kidney, heart and blood systems, 259
 gastro-intestinal irritation, 259
 portal of entry:
 absorption through skin, 258
 inhalation, 258
 prophylaxis, 258-259
 adequate ventilation, 258
 exhaust systems, 258
 masks, 259
 respirators, 259
 selection of employees, 259
 Organized labor, 491-492
 health programs, 491-492
 Osmium, 199
 Osteosarcoma, in dial painters, 184
 Oxygen, 95-98, 195
 in prevention of:
 caisson's disease, 95-96
 compressed air illness, 95-96
 toxic effects at high pressure, 97-98
 symptoms of, 98
 Oxygen in treatment, 96-97, 262, 275, 279, 281, 287, 294
 burns, 448
 CO poisoning, 275
 Cl poisoning, 279
 COCl₂ poisoning, 281
 caisson's disease, 96-97
 compressed air illness, 96-97
 electrical shock, 294
 H₂S poisoning, 287

- gasoline poisoning, 262
- Oxygen want, 174, 448
 - in burns, 448
 - in toxic exposure, 174
- P
- Pain, 227, 264, 471-472
 - abdominal:
 - lead poison, 227
 - wood alcohol poison, 264
 - ruptured intervertebral disc, 471-472
- Palladium, 199
- Paralysis in caisson's disease, 94
- Parkinsonism, 196, 246, 247
- Parran, Thomas, 547.
 - on lengthening shift periods, 547
- Patch tests, 316-318, 332-334
- Paying for medical service in industry, 40
- Penicillin, 444
- Pennsylvania, 526, 529
 - accidents to women in, 529
 - law on work for women, 526
- Pennsylvania Committee on Child Labor, 4
- Peracelsus, 2, 296
- Peterson, C. M., 65
- Petroleum as cause of cancer, 328
- Philadelphia Health Council and Tuberculosis Committee, 520
 - medical care in small industries, 520
- Philadelphia plan, 520
 - medical care in small industries, 520
- Physicians, 35-39, 145, 421, 422, 518, 519
 - industrial, 35-39, 421, 422, 519
 - attributes, 37
 - duties, 37-38, 519
 - full time, 38-39
 - insurance, 39
 - part time, 39
 - preparation, 35-36
 - responsibilities, 36-37
 - specialists, 39, 421, 422
 - general practitioner, 37, 145, 518
- Phosgene, 280-281
 - action, 280
 - pathological findings, 280-281
 - prognosis, 281
 - symptoms, 280
 - treatment, 281
- Phosphine, 192
- Phosphoric acid, 192
- Phosphorus, 190, 191
 - red, 191
 - white, 191
 - yellow, 191
- Physical examinations, 144-149, 150-164, 215-216, 258, 319-320, 405, 421, 505
 - control of accidents, 505
 - control of disease, 505
 - labor and, 505
 - medical control of exposure to toxic chemicals, 150-179
 - Wisconsin program, 57, 505
 - periodic, 100, 115, 151, 215-217, 422
 - analysis of results, 164-175
 - frequency of, 216, 422
 - in chemical industry, 151
 - in lead exposure, 216
 - determined by medical department, 145
 - for failing vision, 422
 - use of results, 175
 - pre-employment, 100, 144, 150, 216, 405, 421
 - benefits, 144
 - blanks for, 146, 147
 - classification, 148, 149
 - eye in, 421
 - method, 145
 - psychological aspects, 148
 - purpose, 144, 151
 - rejections, 145, 148, 404, 405
 - time necessary for, 145
- Pickrell, K. L., sulfa drugs in translucent pliable film, 452
- Pitchblende, mining of, 183
- Plant construction, 212-213
 - locker rooms, 213, 551
 - lunch rooms, 213, 413-414
 - sanitary equipment, 213, 549-552
 - toilets, 213, 550
 - wash rooms, 213, 551
- Plasma in traumatic shock and burns, 435
- Plumbism. See lead poisoning, 202
- Pneumonia, 11-14, 80, 272, 273
 - in steel industry, 11-14
 - steel, mining and outside occupations, 80
- Pneumonoconiosis, 345-374
 - the disease, 355-363
 - acute, 356-357
 - anthracosilicosis, 357-363
 - advanced stage, 361-363
 - early stage, 357-358
 - intermediate stage, 358-361
 - diagnosis, 356, 359, 362
 - pathology, 357-358, 359, 362
 - prognosis, 357, 362
 - signs and symptoms, 358, 361-362
 - simple, 357

- Pneumoconiosis—continued
 the problem, 345-347
 classification, 346
 control problem, 347
 definition, 346
 dust in lungs, 345
 early history, 345
 incidence, 346-347
 in Italian mill workers from inhaled
 dust of barium sulfate, 183
 pathogenesis, 347-355
 action of silica, 348
 acute silicosis, 354-355
 causative agent, 347-348
 predisposing factors, 351-352
 progressive development, 352-354
 related anatomy, 349-351
 with silicosis
 bronchopneumonia in, 363
 silico-tuberculosis, 363-367
 diagnosis, 364
 pathogenesis, 363
 pathology, 365-367
 prognosis, 367
 progress, 365
 roentgenopathology, 365
 signs and symptoms, 364
 other
 anthracosis, 373
 asbestosis, 372
 siderosis, 372
 Poisonous gases, some, 278-287
 CO, 269-277
 chlorine, 278-280
 action, 278
 prognosis, 279
 symptoms, 278
 treatment, 279
 hydrocyanic acid and cyanide group,
 281-283
 after effects, 283
 post-mortem findings, 283
 prevention, 283
 symptoms, 282
 treatment, 283
 hydrogen sulphide, 285-287
 prevention, 286
 symptoms of acute poison, 286
 symptoms of subacute poison, 286
 treatment, 287
 nitrous fumes, nitrogen oxides, 283-285
 prevention, 284
 symptoms, 284
 treatment, 284-285
 phosgene, 280-281
 action, 280
 pathological findings, 280
 prognosis, 281
 symptoms, 280
 treatment, 281
 Polonium, 195
 Post-graduate education, 5, 65, 66, 496,
 519
 medical, 5, 519
 Council on Industrial Health, 5, 66
 American Association of Industrial
 Physicians and Surgeons, 65
 nursing:
 Curriculum guide for public health
 nursing, 496
 Simmons College, 496
 Postgraduate medical education, 519
 Potassium, 181
 Pott, Percival, 296
 Pregnancy, 545
 Pressure, abnormal atmospheric, 91-100
 Prevention of eye injuries, 424-425
 Princeton University, Department of
 Economics and Social Institutions
 Industrial Relations section, 525
 training of workers, 525
 Private insurance companies, 504
 in compensation insurance, 504
 Proctor Marble Company, 487
 early use of nurse, 487
 Proden, L., 186
 on toxicity cadmium oxide fumes, 186
 Profit versus cost of visual correction,
 422
 Protective clothing, 320-322
 Protective ointments, 323-325
 Protoactinium, 190
 Psychiatrist, neuro in CS₂ poisoning, 248
 Psychology:
 consider in examinations, 148
 in fatigue, 133, 135
 Psychologist in pre-placement examina-
 tions, 148
 Public Health Administrators, 58, 59
 Public Health Service, 51-52, 387, 394,
 399, 400, 485
 Puerto Rico, 391
 some occupational diseases compens-
 able, 391
 Pulse pressure, 156-160
 in exposure to toxic chemicals, 156-160
 normal limits, 158
- Q
- Quality of lighting, 129-130
 Qualifications of nurse in industry, 488

R

Radium, 182, 183-185, 327, 542
 carcinoma, 184, 327
 dial painting, 184, 542
 ventilation requirements, 185
 women, 542
 necrosis of jaws, 184, 327
 osteosarcoma, 184
 radon gas exhaled through lungs, 184
 stored in bones, 184
 Radon, 181, 184
 Ramazzini, 2, 296
 Reconnaissance survey, 8
 Red phosphorus, 191
 Reflection-factor, 103
 Rehabilitation, vocational and industrial, 390, 503, 507-517
 compensation and, 503
 Federal and State Rehabilitation Service, 507
 extent of, 511
 U. S. Office of Education, 511
 functioning of
 consultation and guidance, 514
 discovery of proper cases, 513
 physical restoration, 514
 placement, 515
 selection of trainees, 513
 vocational or trade training, 514-515
 origin of service, 510-511
 maimed troops, 510
 workmen's compensation, 510
 purpose, 507
 problem:
 economic, 511
 employer resistance, 512
 inadequate staff, 512
 social, 512
 selection of cases for, 508, 513
 factors affecting, 508-510, 513
 tuberculosis, 390
 Veterans Rehabilitation Service, 510, 511
 Resnick, L., on cost of eye injuries, 424, 432
 Respiration, artificial Schafer prone pressure method, 275
 Respirators, 32, 213, 214, 259
 Respiratory protection, 18
 Resuscitation, 275, 294
 Drinker apparatus, 294
 E. and J. Resuscitator, 275
 pulmotor, 275
 Schafer prone pressure method of artificial respiration, 275
 Rhenium, 196

Rhode Island, 57
 Rhodium, 199
 Robson, W. D., 188
 on exposure to aluminum dust, 188
 Robertson, D. F., 435
 Roentgen rays, 183
 Rossiter, Frank S., 269, 278
 Rothwell, H. E., 193
 on arsenic in hair of normal and exposed persons, 193
 Routes of absorption of toxic chemicals:
 ingestion, 204, 205
 inhalation, 204-206, 258
 skin, 204-206, 258
 Royal Eye Hospital, London, 421
 eye accident statistics, 421
 Rubidium, 181
 Russia, 498
 Ruthenium, 199

S

Safety code:
 for foundries approved, 4
 sanitation of foundries adopted, 4
 Safety committee in control of accidents, 32
 Safety facilities in control of absenteeism, 540
 Safety shoes, 532
 Safety supervisor in control of accidents, 32
 Sanitary equipment, 213, 549-552
 Sawyer, William A., 409
 Sayers, R. R., 270
 table on time required of various concentrations of carbon monoxide for certain per cent of blood saturation, 270
 Schafer prone pressure method of artificial respiration, 275, 292, 293
 Schrenk, H. H., 266
 on exposure to dioxan, 266
 Schwartz, Louis, 20, 257, 258, 296-341
 Scott, C. N., 425
 on protection of eyes, 425
 Second injury funds, 503
 Secretary of labor, 527-528
 rules for working of girls, 527-528
 Section on dermatoses investigation, 298
 Section on industrial hygiene formed, 4
 American Public Health Association, 4
 Seeing, 104-131
 aids to, 130-131
 ease of, 118-123
 precision and production, 112-114
 Selenium, 195

- Self-insured, 504
 - Self-medication, toxic exposure and, 173
 - Semi-direct lighting, 104
 - Semi-indirect lighting, 104
 - Service groups and tuberculosis, 385
 - Severity rate (industrial accidents), 24-25
 - Sex and accidents, 24
 - and skin diseases, 303
 - and tuberculosis, 380
 - Shift work, 546-547
 - frequency of shift, 547
 - single people and, 547
 - women and, 546
 - factors against, 546-547
 - Shoes, safety, 532
 - Siderosis, 372
 - Silica, 189
 - Silicosis (see pneumoconiosis), 16-20,
 - 148, 214-218, 368-369, 506
 - compensation of, 367-371, 506
 - disability and, 370-371
 - tests for, 370-371
 - table on, 368-369
 - control of, 371
 - mechanical, 16-20, 371
 - medical, 148, 214-218, 371
 - Silico-tuberculosis, 363-367
 - Silver, 181, 182
 - Simmons College, 496
 - course for nurses in industry, 496
 - Single women, 547
 - night work, 547
 - shift work, 547
 - Skin diseases, 296-341
 - see occupational disease of skin, 296-341
 - Snell, A. G., 433
 - Snellen Chart, 46, 111, 421
 - Sodium, 181
 - Soto-Hall's sign, 477
 - South Carolina, 526
 - law on work for women, 526
 - Spine, 459-463
 - anatomy and physiology, 459-463
 - Sprain, 479
 - Stippling, 218
 - of red blood cells, 218
 - Strain, 473, 474, 479, 544
 - Strontium, 182, 183
 - Substitution, 16
 - of less toxic substance, 16
 - Sulfa drugs, 171-172, 455
 - in toxic exposure, 171-172
 - in treatment of burns, 455
 - Sulfur, 195, 285-287
 - irritation of skin, 195
 - Sulfuric acid, 195
 - Superficial foreign bodies in cornea, 426
 - Supplementary lighting, 104
 - Surgeon General, 400
 - on shift work, 400
 - Sweet, William M., 427
 - on localizing position of foreign body in eye, 427
 - Symblepharon, 431
 - Synovitis of the arm, hand and wrist, 542
 - Systolic blood pressure, 155-179
 - in exposure to toxic chemicals, 155-179
 - difference between two arms, 158
 - normal limits, 158
- T
- Tantalum, 190
 - Tar as cause of cancer, 327
 - Tasks, visibility of, 117
 - Taylor, J. H., 135
 - studies on fatigue, 135
 - Technicians, 40, 46
 - laboratory, 40, 46
 - x-ray, 40
 - Tennessee Valley Authority, 61
 - Tellurium, 195, 196
 - poisoning, 196
 - symptoms, 196
 - Territory of Hawaii, 391
 - all occupational diseases compensable, 391
 - Tetrachlorethane, 262-263
 - action, 262
 - irritation, 262
 - narcotic, 262
 - solvent, on skin, 262
 - prophylaxis, 262-263
 - symptoms, 262
 - treatment, 262-263
 - Tetraethyl lead, 206, 261
 - poisoning, 206, 261
 - Thallium, 189
 - Thompson, Lorin A., 133, 141
 - Thorium, 190
 - Tin, 190
 - Titanium, 190
 - Toilets, 213, 550
 - accessible, 550
 - entrances, 550
 - equipment, 550
 - markings, 550
 - one for each 15 women, 550

Toluene (see benzene), 259, 260
 central nervous system, depression in poisoning, 260
 narcotic effect, 259
 Tompsett, S. L., 221
 on progressive accumulation of lead in bones, 221
 Toxicity of gases and vapors, high altitudes, 174-175
 Training of first-aid personnel, 423
 Training of physically impaired, 514-515
 Trichlorethylene, 263, 264
 action:
 narcotic, 263
 skin irritant, 264
 solvent, on skin, 263
 prophylaxis
 ventilation:
 closed system, 264
 downward draft, 264
 protective ointments, 264
 Transfusion, 439, 440
 technic of, 439
 blood grouping, 439
 reaction following, 440
 Trauma, as cause of cancer, 326
 and tuberculosis, 382
 Traumatic shock, 435
 clinical features, 436
 pathological features, 435
 treatment:
 plasma, 438
 saline solution, 438
 whole blood, 438
 dosage of above, 439
 Trumper, M., 246, 247
 symptomatology of chronic CS, poisoning, 246, 247
 Tuberculosis and occupation, 375-393
 see occupation and tuberculosis
 Tungsten, 194, 195
 Twort, C. C. and J. M., 329
 carcinogenic tars, 329

U

Uniforms for women workers, 531-532
 U. S. Army, 379
 United States Department of Agriculture, 531
 United States Department of Commerce, 185, 425, 542
 Bureau of Standards, 425
 control of radium poisoning, 185, 542
 United States Department of Interior, 4, 33, 50, 56

Bureau of Mines, 4, 33, 50, 56
 training in first aid, 33
 first study of health of workers, 4
 United States Department of Labor, 50, 56, 346, 347, 395, 433
 Bureau of Labor Statistics, 50, 395
 Children's Bureau, 4, 50
 Division of Labor Standards, 50, 56, 347
 National Committee on Conservation of Man Power in Defense Industries, 5
 Women's Bureau, 50, 529, 530, 531
 United States Employees Compensation Act, 500
 Compensation for Federal Government employees, 500
 U. S. employment service, 524, 526
 United States Federal Security Agency, 61, 62
 War Production Board, 61, 62
 U. S. Navy, 327, 379
 cancer of lip and skin, 327
 miniature x-ray films, 379
 United States Office of Education, 511
 rehabilitation, 511
 U. S. Public Health Service, 4, 51-52, 298, 387, 394, 399, 400, 485, 537, 547
 Division of Industrial Hygiene, 9, 62
 First study of health of workers, 4
 National Health Survey, 537
 Office of Industrial Hygiene and Sanitation, 4
 reporting morbidity among industrial workers, 4
 Section on dermatoses investigation, 298
 surgeon general, 400, 547
 Uranium, 194
 Utah, 57

V

Vanadium, 190
 Venereal disease control in industry, 394-407
 administration of program, 401
 confidential records, 403
 employment policy, 404
 follow-up, 406
 frequency, 395
 informational program, 402
 industry's responsibilities, 396
 integration with community program, 397
 morbidity reporting, 406

- Venereal disease control in industry
—continued
objectives, 401
preliminary approach, 400
reasons for program, 398
- Ventilation, 8, 9, 17, 197, 212, 258, 320
designed for specific need, 212
in manganese industry, 197
in organic solvent plants, 258, 259, 262
local exhaust, 17, 320
downward draft, 262, 264
skin diseases, 320
- Vermont, 486
nursing consultant, 486
- Visibility, 108, 114, 115, 117
measurements of, 114
Luckiesh-Moss Visibility Meter, 115
physical factors and, 108
tasks, 117
- Visiting nurses, 487-491, 492, 494
Association of Detroit, 490
Henry Street Settlement, 490
home accidents, 494
Metropolitan Life Insurance Company, 487
nutritionist and, 490
Pittsburgh Public Health Nursing Association, 491
- Visual acuity, 110
- Visual defects in industry, appraisal of, 423
- Vitamin supplements in industry, 171, 233, 418-419
in exposure to toxic chemicals, 171
in industry, 418-419
in lead exposure, 233
- Vitamin therapy, 74, 456
requirements in burns, 456
requirements for wounded, 456
- Vitelles, Morris, defines fatigue, 133
- Vocational training, 514-515
- Valtmer, 246-247
symptoms from CS₂ exposure, 246-247
- Voluntary election in compensation law, 500
- von Oettingen, W. F., 266
on toxicity of butyl ethylene glycol, 266
- W
- Walker, S., 427
caring for injuries to eyes, 427
- Walsh-Healy Act, 527, 529
on employment of girls, 527
- Wampler, Fred J., 1, 35, 43, 144, 518
- Wanamaker Department Stores, 487
early nursing service to employees, 487
- War Production Board, 61, 62
section of industrial health, hygiene and safety, 62
- Washington, 526
law on work for women, 526
- Wash rooms, 213, 551
- Watt, S., 141
on factors influencing output, 141
- Weaver, W. L., 74
vitamins in control of heat cramps and heat prostration, 74
- Welfare facilities in the control of absenteeism, 540
- Wet method in dust control, 17, 197
in manganese industry, 197
- White-Calvert apparatus, 243
- White phosphorus, 4, 191
matches abolished, 4
- Wiley, F. H., 244
procedure for determining CS₂ in blood, 244
- Williams, George Zur, 345
- Wisconsin:
compensation, 500
first law, 498
industrial commission, 530
law on work for women, 526
legislative committee, 499
night work for women, 526
nursing, 493
physical examination program, 57
- Womens Bureau, 50, 529, 530, 531, 546
detailed study of accidents, 529
- Women in industry, 523-555
absenteeism and illness, 537
health factors, 536
non-occupational illness, 543
fatigue, 545
menstruation factors, 544
nervous manifestations, 548
noise, 547
pregnancy, 545
occupational illness, 540
dermatitis, 541
other diseases, 542
radium dial painting, 542
prohibited employment, 527
Federal laws, 527
night work, 526
states prohibiting certain work, 527
safety factors, 529-536
emotional stability, 536
falls, 529
fatigue, 535
jewelry, 532
long hair, 532

machine guarding, 531
 physical limitations, 533
 lifting, 533-535
 shorter, 533
 smaller, 533
 strength, 533
 safety shoes, 532
 state regulations, 536
 training, 530
 work clothes, 531-533
 caps, 531
 gloves, 531
 shoes, 531-533
 uniforms, 531-532
 sanitation facilities:
 dressing and rest rooms, 551
 housekeeping, 552
 maintenance, 552
 toilet facilities, 550
 washing facilities, 551
 selection and training, 524
 welfare facilities, 552
 child care, 554
 recreation and social activities, 553
 seats, 553
 Wood alcohol (methanol), 264, 265
 Work clothing, 80, 213, 257, 264, 320-321,
 531-532, 551
 changes of, 80, 213, 257, 264, 320-321,
 531-532, 551
 Work day, length of, 136-138
 accident production, 136
 fatigue, 136-138
 production, 137-138
 Work week, length of, 136-138
 accident production, 136
 fatigue, 136-138
 production, 137-138

Workmens Compensation Act, 482
 objectives, one of, 482

X

Xenon, 181
 X-ray, 387-389
 in diagnosis of:
 backache, 477
 sacroiliac slip, 469
 spina bifida occulta, 466
 in medical lay-out, 44
 periodic examinations, 145
 pre-employment examinations, 145,
 147
 tuberculosis case findings, 379, 384,
 388-389
 mass surveys, 379
 paper films, 379
 sub-standard films, 379
 portable, 387
 precautions against, are necessary, 183
 value of technicians, 40
 Xylene (see benzene), 259, 260

Y

Yale University Medical School and New
 Haven plan, 520-521
 Yant, W. P., 266, 270, 272
 colorimetric test for carbon monoxide
 in blood, 272
 on exposure to dioxan, 266
 table on time required of various con-
 centrations of carbon monoxide
 for certain per cent blood satura-
 tions, 270