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February 23, 1944

Mr. Ivan Sabourin
Aldred Building
Montreal, P. Q.
Canada

Dear Mr. Sabourin:

While the matter is still fresh in my mind I am writing you at length on my impression of the situation on Asbestosis in Quebec in so far as it involves relationships between the producers, the medical profession and the Compensation Commission. One conference with Mr. Sharp, Dr. Vidal and Mr. Dick gave me a much clearer picture than I had gotten previously. Before doing so I offer a few comments on autopsy material.

I am enclosing my report on the lung tissues of Philemon Tardif. Of necessity the conclusions are not too satisfying as they are based only upon examination of two blocks of tissue, approximately 2 x 4 centimeters in diameter without even an indication of the parts of the lung from which they were taken. However, they like the material from the two previous cases, do demonstrate that the Thetford operations produce limited amounts of indisputable Asbestosis. In the presence of advanced tuberculosis this is obscured and not likely to be identified except on microscopic examination of pulmonary tissue. X-ray during life will not show it.

Today I have also examined in gross the sagital section through the entire right lung of Majorique Perron which we collected yesterday from the toxicological laboratory in Montreal. This is a much more satisfactory specimen with which to work. It likewise shows a combination of widespread chronic tuberculosis upon a background which will probably prove on microscopic examination to be minimal Asbestosis. Sections will be prepared as soon as possible for microscopic examination. In order to make my report complete in this case, I would appreciate it if you would send me a brief summary of the occupational and clinical history including: How long employed, character and date of first symptoms, date of quitting work, whether Koch's bacillus was found in sputum and date of death. I neglected to copy this information from your record.

This autopsy material on fatal cases and the X-rays that are now reaching the Commission are bound to create in their minds the impression that tuberculosis is an all too common complication of Asbestosis. If I were in their position with my experience limited to evidence of this kind I would find it difficult to resist the conclusion that there is causal relationship between even slight amounts of

asbestosis and tuberculosis of the lungs. I have told you repeatedly as I told Mr. Sharp yesterday that I did not agree with this conclusion but I doubt whether he or Dr. Vidal will be persuaded when they see a succession of cases like those which are now passing before them. They must be made to realize that these cases are the exceptions rather than the rule and that there is a great majority of employees in the asbestos industry who are free from tuberculosis.

I would therefore urge that immediate steps be taken t survey every employee in the asbestos industry in Quebec so that the necessary statistics can be assembled. Dr. Stevenson's X-ray films alre taken on the men at Asbestos will provide you with material for a preliminary report. Coupled with this there might be an analysis of mortal data contrasting the total death rate from tuberculosis in Thetford and Asbestos with that in other industrial communities of the Province. It would be still more convincing if you could show the separate rates of males and females unless the number of the latter employed in the asbestos plants constitutes an appreciable proportion of the female population. Finally the age distribution of deaths from tuberculosis in those employed in the asbestos plants as contrasted with that in other occupations would help in determining whether the occupational factor wa contributory.

My own observations elsewhere and your intimation that the rate for tuberculosis was higher in St. Hyacinthe than in Thetford and / or Asbestos make me confident that the companies would acquire information of greatest service to themselves by such a survey and analysis. If asbestosis specifically is predisposing to tuberculosis in the same sense as silicosis does the effect should be just as apparent in North Carolina and Pennsylvania as it seems to be in Quebec or England But the films of employed asbestos workers in the States show a very low incidence of associated tuberculosis. It is my firm belief that in Quebec and England, factors outside the occupation are responsible.

As I sensed the situation in Montreal yesterday the asbestos industry will have to produce evidence to prove this point and start on it immediately or the industrial commission will become increasingly convinced that our position is biased and not founded upon truth.

Furthermore the industry would undoubtedly profit by undertaking a general program of community control of tuberculosis. By instituting case finding machinery not only among employees but among the members of their families, the open cases of tuberculosis would be discovered and the sources of spread of the infection could be removed.

One of the iron mining companies whom we serve has such a program with a public health nurse, a program for tuberculin testing of school children and follow up of the contacts of every known case of open tuberculosis. The program has paid dividends by practically eliminating tuberculosis with resultant lowering of compensation costs and the good will of the employees is unmeasurable. The asbestos industry of Quebec has a serious problem because its labor force is drawn from a population in which the tuberculosis rate is high but their situation is no worse than that which our other clients attacked successfully in an iron mining community.

The second point which is becoming increasingly apparent to me is that the asbestosis of the Quebec mines and mills is quite generally less severe and extensive than the asbestosis of the fabricating plants of the States. Comparison of the X-ray findings in the two groups reveals very few cases with typical patterns among Thetford or Asbestos mill men. Some asbestosis is unquestionably produced for we have microscopic evidence of it in the four autopsy cases now available but it is not advanced enough to replace much of the functional parts of the lungs and thus cause the X-ray shadows that are generally recognized as characteristic of the disease. (Most of us disagree with Dr. Pendergrass when he says in Lanza's book that there is no characteristic pattern of asbestosis). If my observations are correct this relatively slight amount of disease should not cause disability for only the framework of the lungs is involved leaving most of the parts that carry on the functions of respiration intact.

But here again the Canadian industry is on the defense against its medical profession and their representatives on the advisory board to the industrial commission. The doctors have seen only their own local manifestations of asbestosis without opportunity to compare it with the really severe manifestations that are produced elsewhere. They find the abnormal shadows in the X-ray films of exposed workmen, which they are convinced must cause symptoms and disability. They find asbestosis bodies in the lungs of autopsied cases associated with minimal tissue reactions or overwhelming manifestations of concurrent pulmonary infections and now they are sure of their ground.

In order to defend itself the industry in Quebec must have more facts which will require time to collect, analyze and publicise. These findings will have to be contrasted with those in the fabricating plants either in Asbestos or elsewhere. In the city of Asbestos the spinning and weaving mill has been in operation for only four years which is the minimum time to produce much disease in those exposed, but in another year or two one may expect to find evidence of it here unless I am much mistaken.

As I have told you in conversation, our experimental observations lead me to believe that the true hazard in asbestos plants is proportional to the amount of small, individual, uncrushed fibres in the industrial atmosphere. I suspect that the reason why one sees little or no typical asbestosis in the mines and mills is because there is probably too little such fibre in the air of these plants. Conversely the reason for the "typical" disease in the fabricating plants is the fact that the processes there open the bundles of asbestos fibre and tear them into lengths short enough to be inhaled in dangerous quantities.

The mills and mines are dusty but the dust is largely serpentine with relatively few fibres large enough to do harm to the lungs. This dust enters the lungs where it produces reaction around blood vessels and other framework structures that are just like any other inert dust. We see the shadows of such inert reaction manifested in X-ray as an exaggeration of the normal shadows of blood vessels. This condition is not asbestosis nor necessarily preasbestotic any more than the identical changes in men working in hard rock mines are necessarily presilicotic. In free silica industries regardless of exposure we do not diagnose silicosis from X-rays until we find the characteristic pattern of modulation; in the asbestos industry we should do likewise and refrain from a diagnosis of asbestosis until the X-ray reveals the characteristic pattern of haziness or fine stippled appearance spreading throughout the lower thirds or more of the lung fields. The situation with asbestosis is even more difficult than with silicosis for today the requisite condition of the dust exposure is not appreciated by the medical profession.

Therefore I strongly urge that surveys be made to compare the relative quantity and nature of the dusts in the mills and the fabricating plants in order to present proof that in the first instance the percentage of thin fibre of inhalable dimensions is insignificant in comparison with the quantity created in spinning and weaving. It is going to be hard to persuade physicians of the importance of these factors but similar factors govern the reaction to quartz and the development of silicosis. These physical limitations of atmospheric dust often determine the outcome in contested cases involving a diagnosis of silicosis and I forsee that they will have equal weight in asbestosis. A competent industrial hygienist should collect and analyse enough of the atmospheric samples in weaving and spinning mills and in the mines and processing plants to establish this basic difference so that it will stand beyond controversy. We have the experimental background; we now must demonstrate its industrial application. When correlated with the results upon exposed workmen, I feel certain that our position will be secure.

To summarize: I would make the following recommendations to your clients, the Canadian Asbestos Producers in order that they may defend themselves against a growing conviction among the local physicians that employment in this Quebec industry is highly dangerous.

I. Correct an erroneous impression of the Industrial Commission that asbestosis is frequently associated with pulmonary tuberculosis.

a) Statistical data based on X-ray of all employed workmen to indicate the incidence of pulmonary tuberculosis

b) Comparative mortality statistics on tuberculosis in Asbestos and Thetford and in other communities of Quebec indicating the frequency by sex and age.

2. Consider the possibility of inaugurating a program of tuberculosis case finding and control in the general populations of Asbestos and Thetford in order to eliminate sources of new infection among employees.

3. Make a detailed comparative study of the nature of the dust in the fabricating plants and the asbestos mines and mills.

I trust that you will agree with these recommendations and be willing to pass them along to your clients.

May I take this opportunity to thank you for your delightful hospitality while I was in Montreal and thank you for making my visit profitable and pleasant.

Sincerely yours,

Leroy U. Gardner, M. D.
Director

LUG:RH

PART III

THE MANPOWER PROBLEM

PART II

PREVENTION AND CONTROL OF DISEASE IN INDUSTRY

SECTION I

CHAPTER 9

THE PROBLEM OF OCCUPATIONAL DISEASE

W. C. Dreessen, M.D.

INTRODUCTION

CLAIMS paid for occupational disease under workmen's compensation amount to from 1 to 3 per cent of the amount paid for industrial accidents. This cost comparison, however, does not give the complete picture of their relative importance. In speaking of occupational diseases one ordinarily thinks of such diseases as occupational dermatitis, lead poisoning, and silicosis. In this country these three groups of diseases constitute the most important occupational diseases, with respect to both number of cases receiving compensation and total cost of compensation per case.

Based on a year's experience of one State,[1] the compensation costs of individual cases are generally lowest for dermatosis cases (averaging about $50 per case) and highest for silicosis cases (averaging about $5,000 per case); lead poisoning occupies a middle position with an average cost per case of about $150. These differences in costs are related to the degree of permanency and duration of the disability. These three diseases bid well to continue to lead the list in the changeover from peace to wartime production.

The liability of the employer without proof of fault is the essential principle upon which workmen's compensation is based. Within the limitations of wording or mode of administration

of the compensation laws, the provisions common to these acts are the giving of prompt medical care, and payment of monetary benefits, at the cost of the employer without regard to the question of negligence, to an injured or occupationally ill employee or to his dependents in case of death in line of duty.

Under the workmen's compensation laws,[2] the three common law defenses, namely contributory negligence, the fellow-servant doctrine, and the assumption of risk, are no longer available to the employer. Consequently, the burden of economic loss and waste due to personal injury has been shifted from the employee to industry and thus has been made an item in the cost of production ultimately to be borne by the consumer.

Occupational diseases were not specifically covered in the original State workmen's compensation laws. Even as recently as 1920, compensation for such diseases was provided in only seven jurisdictions[3] in the United States—California, Connecticut, Hawaii, Massachusetts, North Dakota, Wisconsin, and the Federal government. Occupational diseases introduced more complex factors in the administration of law than were encountered with industrial accidents.

Occupational Disease Coverage in Various Jurisdictions

There is a lack of nationwide coverage for occupational disease.* Although all of the 48 States, excepting Mississippi, have legislation providing for compensation of industrial injuries, only 25 States have laws providing compensation for occupational disease. The Federal government also provides compensation for such diseases under compensation laws for its civil employees, longshoremen and harbor workers, and for such diseases arising from private employment in the District of Columbia. Compensation is provided for all occupational diseases or for certain specified ones in the jurisdiction shown in the accompanying list. This list also contains the name and address of the agency administering workmen's compensation in the respective State or jurisdiction. Copies of compensation legislation and the rules and regulations of the administrative agency pertaining to the jurisdiction in which he is practicing should be readily available to the industrial physician.

* The discussion in this chapter has been limited to occupational diseases. The reader should not overlook compensation aspects of industrial accidents.

LIST OF JURISDICTIONS IN WHICH OCCUPATIONAL DISEASE IS COMPENSATED AND THE NAME AND ADDRESS OF THE ADMINISTRATIVE AGENCY

Arkansas	Workmen's Compensation Commission Rector Building, Little Rock
California	Division of Industrial Accidents and Safety State Building, San Francisco
Connecticut	Board of Compensation Commissioners 54 Church Street, Hartford
Delaware	Industrial Accident Board Ninth and Market Streets, Wilmington
District of Columbia	D. C. Workmen's Compensation Act Seventh and E Streets, N. W., Washington
Hawaii	Department of Labor and Industrial Relations Bureau of Workmen's Compensation Honolulu
Idaho	Industrial Accident Board Boise
Illinois	Industrial Commission 205 West Wacker Drive, Chicago
Indiana	Industrial Board 404 State Capitol, Indianapolis
Kentucky	Workmen's Compensation Board Frankfort
Maryland	State Industrial Accident Commission Equitable Building, Baltimore
Massachusetts	Department of Industrial Accidents Statehouse, Boston
Michigan	Department of Labor and Industry 630 State Office Building, Lansing
Minnesota	Industrial Commission 137 State Office Building, St. Paul
Missouri	Workmen's Compensation Commission State Office Building, Jefferson City
Nebraska	Workmen's Compensation Court State Capitol, Lincoln
New Jersey	Bureau of Workmen's Compensation Wallach Building, Trenton
New York	Department of Labor Division of Workmen's Compensation 80 Centre Street, New York
North Carolina	Industrial Commission Raleigh
North Dakota	Workmen's Compensation Bureau Bismarck
Ohio	Industrial Commission State Office Building, Columbus
Pennsylvania	Bureau of Workmen's Compensation Harrisburg
Puerto Rico	Industrial Commission San Juan
Rhode Island	Department of Labor Division of Workmen's Compensation Providence

LIST OF JURISDICTIONS IN WHICH OCCUPATIONAL DISEASE IS COMPENSATED AND THE NAME AND ADDRESS OF THE ADMINISTRATIVE AGENCY (*Cont.*)

Utah	Industrial Commission State Capitol, Salt Lake City
Washington	Department of Labor and Industries Olympia
West Virginia	Workmen's Compensation Department Charleston
Wisconsin	Industrial Commission 1 West Wilson Street, Madison
United States	U. S. Employees' Compensation Commission 285 Madison Avenue, New York, New York

Definition of Industrial Disease

Briefly stated, an occupational disease is an affliction due to a specific industrial health hazard. Legal connotations, however, tend to either limit or extend the number of diseases embraced by this definition. There are two schools of thought on the subject of occupational disease legislation and administration. Thus, one school holds that a disease to be occupational must "arise out of and in the course of employment." In other words, it is something characteristic of the employment and not a hazard to which the public is generally exposed. Such diseases are fairly well exemplified by those listed in certain schedule laws (e.g., poisoning by lead, arsenic, and mercury, and silicosis).

The other school holds that any disease contracted by a worker, which arises out of employment or out of an incident of employment and yet not necessarily characteristic of employment, is an occupational disease. This viewpoint provides for the inclusion of such diseases as pulmonary tuberculosis and malaria. Thus, nurses and internes have received compensation for pulmonary tuberculosis contracted in the course of their work in tuberculosis sanatoria, and a railroad laborer is compensated for malaria contracted in railway section work.

The definition of occupational disease has always been found to be very difficult.[4] Much has been written on the subject. One of the commonly quoted definitions is that of the Rhode Island law which states that, "The term 'occupational disease' means a disease which is due to causes and conditions which are characteristic of and peculiar to a particular trade, occupation, process, or employment."

In many instances it is seen that the "compensable diseases" are the "occupational diseases." Brahdy's comments[5] on this point, however, are pertinent: "When physicians differentiate

between the terms, 'occupational' and 'compensable,' they recognize a boundary separating their medical field from the legal administrative field. Physicians—and only physicians—can decide whether a disease is occupational. Lawyers and administrators, but never physicians, must decide if an occupational injury is compensable according to the law of that State."

Comparison of Schedule and General Coverage Laws.—In some of the State compensation laws, no clear-cut distinction is made between "occupational disease" and "industrial injury." This is probably related to the fact that before administering agencies had experience in compensating occupational disease, an attempt was made to provide coverage for such diseases by amending definitions of the term "injury" to include occupational diseases in some form, either by listing a few of them or by the use of broad language.[4] Two general terms are thus ordinarily applied to laws providing compensation for occupational disease. These acts are referred to as (1) *schedule* or limited coverage laws, and (2) general coverage or *blanket* laws.

Under the schedule laws, the specific compensable diseases are listed and briefly described. This type of coverage holds in Arkansas, Delaware, Idaho, Kentucky, Maryland, Michigan, Minnesota, Nebraska, New Jersey, North Carolina, Pennsylvania, Rhode Island, Utah, West Virginia, and Puerto Rico.

In other jurisdictions the law provides compensation for any disability arising from an occupational disease without attempting to name it and is the law in California, Connecticut, District of Columbia, Illinois, Indiana, Massachusetts, Missouri, New York, North Dakota, Ohio, Washington, Wisconsin, and Hawaii.

The language of some general coverage acts adds qualifications to the effect that ordinary diseases of life to which the general public is exposed outside of the employment shall not be compensable, except where the said diseases follow as an incident of an occupational disease. These general coverage acts having a statutory definition of the term "occupational disease" are referred to as *definitive general coverage* laws.

Provisions of Compensation Laws

The industrial physician should know which occupational diseases are compensable in his State. He should also familiarize himself with the administration of the law so that his reports and opinions will convey the proper meaning. Besides the type

of coverage referred to above, the laws include such provisions as scale of compensation, insurance features, limitations as to type of employment, number of employees, medical benefits, extraterritorial provisions, reporting of occupational diseases, medical boards, accrued liability, and waiver or second-injury provisions.

Contrary to industrial accidents which can usually be related to time and place, certain occupational diseases take years to develop and hence as a point of reference the day when disability or incapacity begins is usually recorded as the date of injury.

Workmen's compensation laws are designed primarily to furnish the occupationally ill worker with medical care and monetary benefits during the period of his disability or money payments to his beneficiaries in case of death. Except in some States having a State insurance fund and those States where employees may contribute, the cost of compensation is borne almost entirely by the employer. *Compensation* for loss of wages usually runs from 50 to 70 per cent of the employee's average wage with minimum and maximum amounts usually specified. Most States provide a certain specified period of time immediately following disability, during which compensation shall not be paid. This period varies from one to 14 days, but is 7 days in most States. Claims must be filed within certain time limitations. Failure to provide full compensation for wage loss is meant to be an incentive for the temporarily disabled worker to return to work as soon as possible and to preclude malingering. *Medical costs* are not borne by the worker in most of the States, but maximal limits as regards period of time and cost, or both, of this service are usually specified. According to Newquist,[3] "the acts of 23 States and of the Federal government do not limit the period for medical benefits other than by qualifying terms such as 'reasonable,' 'reasonable time,' during 'temporary disability,' etc. The stated time limits for medical service range from 2 weeks to 1 year and the limited amounts for medical benefits range from $100 to $1,600. Because of the uncertainties and great potential burdens associated with the present occupational disease situation, a few States have seen fit to limit the medical responsibilities of employers for treating workers with such diseases, particularly silicosis or asbestosis."

Financing the payment of compensation benefits is usually accomplished through insuring the employer's liability by *insurance* with a private company, by State fund, or by self-insurance.

The employer is allowed to insure in a private company in most States, but a few States have exclusive State funds. Whether or not an employer may elect or be compelled to carry insurance varies according to jurisdiction.

Depending on the degree of incentive offered employers to accept the benefits and burdens of the compensation law, these laws may be classed as *compulsory* or *elective*. According to Dawson,[3] "a compulsory law is binding upon every employer and employee within its scope; there is no choice. Under an elective act, employers and employees have the option of either accepting or rejecting the act. But in case the employer rejects, the customary common-law defenses in personal injury litigation are usually removed, while if the employee rejects, the workmen's compensation principle of liability of the employer for work injuries without regard to fault is not applicable to an action for damages."

Agricultural and domestic workers are excluded from benefits of compensation laws in most States. Among the exceptions are Arizona, California, Connecticut, Illinois, Kentucky, Minnesota, New Jersey, New York, Ohio, South Dakota, Vermont, and some of the territories of the United States. Casual employees are also usually excluded. Employers are also exempt if they employ fewer than a specified number of workers. Special conditions are provided in certain jurisdictions for covering hazardous and public employments as well as disability incurred outside of the State. The limiting provisions relating to silicosis deal with (1) period of employment and exposure within the State, (2) filing of claims, (3) time within which death must occur in compensable fatal cases, and (4) deductions from death benefits.

Some States have included penalty provisions for false statements as a responsibility of the employee. If as an applicant for employment the employee falsely represents that he has not suffered from an occupational disease which subsequently causes disability or death, his compensation is forfeited.

The term *accrued liability* has been used to describe potential compensation claims for occupational disease which existed prior to the enactment of occupational disease legislation. It is particularly characteristic of silicosis, a disease which requires years for the pathologic process to maturate and cause disability. It is an accrual of injuries sustained during previous years of employment. In some acts the last employer of the victim of disease (e.g., silicosis) is held fully liable for compensation. At

the National Silicosis Conference (1938), the Committee on the Economic, Legal, and Insurance Phases of the Silicosis Problem felt that such accrued liability should be at least in part recognized as a public liability. This conclusion is related to lack of nationwide occupational disease coverage. Because of interstate movement of people, States having or planning silicosis coverage fear that they will become the dumping ground for the accrued liability of other jurisdictions.

With the passage of compensation laws making employers responsible, preemployment examinations were adopted by employers to screen out physically defective workers. As Dawson points out, this adverse effect upon the employment of handicapped workers was an unforeseen consequence of these laws. Employers considered the increased risk of loss a good cause for refusing employment. To remedy this injustice and to minimize the difficulty which partially disabled workers have in securing employment, some of the States created special "second-injury" funds and amended the compensation act to provide that in case of a second major disability the employer should be held liable only for the second injury considered separately. The disabled employee, however, is compensated for disability resulting from the combined injuries. Statutory provisions for second-injury funds are included in the laws of Arkansas, District of Columbia, Hawaii, Idaho, Illinois, Massachusetts, Minnesota, New Jersey, New York, North Carolina, North Dakota, Ohio, South Carolina, Utah, West Virginia, and Wisconsin, and also in the Federal Longshoremen's Act.

In the absence of second-injury funds, *waivers* and *limited disability* have been used to meet the issue. These procedures find application particularly in dealing with accrued liability of silicosis. Speaking of waivers, Kessler[19] states: "These waiver clauses take cognizance of the fact that the workmen's compensation law may be an obstacle to employment. They aim to permit the workman to exchange a right for a benefit he may prize more highly. Though it would be possible to safeguard the employer from the suit of a workman who had waived compensation, to allow the workman so to waive this right is open to various objections and abuses. It might become possible, for instance, for employers to require *all* persons with *any* physical disability to sign waivers as a condition of getting employment. . . . In practice, waivers are restricted or prohibited in most jurisdictions." Where restricted, they are issued in accordance with regulations

of the agency administering the compensation law. Under limited disability plans the disabled worker may be compensated only for later injury, or the decreased earning power of the handicapped worker is used as a basis of apportioning compensation. In general, it seems that with reference to handicapped workers an exception should be made to the theory of workmen's compensation which makes industry bear the full burden of responsibility for industrial disabilities and have the government assume part of the burden.

CLINICAL DESCRIPTION OF DISEASES OF PRESENT IMPORTANCE

Classification of Diseases or Conditions

Dublin and Vane[11] classify occupational hazards as follows: (1) abnormalities of air pressure, (2) abnormalities of temperature and humidity, (3) dampness, (4) defective illumination, (5) dust, (6) infections, (7) radiant energy, (8) repeated motion, pressure, shock, etc., and (9) poisons.

Major Groups of Occupational Diseases.—A review of various compensation laws subscribing to schedule coverage shows that the specified occupational diseases fall into six or so major groups when classified according to causative agent. Thus, under *toxic metals or metalloids* may be listed poisoning caused by arsenic, zinc (brass), cadmium, lead, manganese, phosphorus, radium, and mercury; under *dusts* are listed pneumoconiosis and/or silicosis with or without tuberculosis and asbestosis with or without tuberculosis; under *gases, vapors, and fumes*, poisoning caused by hydrogen fluoride, nitrous fumes, sulfur dioxide, carbon disulfide, hydrogen sulfide, hydrogen cyanide, carbon monoxide, nickel carbonyl, halogenated hydrocarbons, methyl alcohol, benzene, and nitro and amino derivatives of gasoline, benzene, and phenol; under *occupational skin hazards*, chrome ulceration or dermatitis, infection or inflammation of the skin or eyes due to oils, cutting compounds, lubricants, dusts, liquids, fumes, gases, and vapors, epitheliomatous cancer or ulceration of the skin or surface of the eye due to pitch, tar, and bitumen, and dermatitis venenata; under *infectious agents* may be listed such diseases as anthrax and glanders; and under *physical agents* may be listed compressed air illness, radioactive substances, cataract, and impaired hearing caused by noise.

War Industries and Occupational Disease.—With few exceptions, the serious and prevalent occupational diseases of prewar days may be anticipated as being the sources of difficulty in war-

time industrial production. Cunningham[12] of Canada has given us some idea of what we may expect from the changeover to war production. He states that the number of cases of occupational disease have increased in the Dominion, but up to the present they have come from increased exposure to common substances rather than from new processes.

Predominance of Industrial Poisoning.—Exclusive of dermatoses, industrial poisonings make up the majority of the occupational diseases. The progressive industrial physician moreover recognizes the need of studying the toxic properties of new substances or chemicals prior to the establishment of a new industrial process. He is aware of the fact that absorption of metals and their compounds does not as a rule induce the same reaction or degree of action in the body as the metal or its compounds when used for therapeutic purposes. In other words, industrial intoxications are characteristically chronic in contradistinction to acute poisonings of usual medico-legal importance. When acute, disability ordinarily occurs on the day of exposure and the disease entity is then usually considered an accident. The portals of entry for industrial poisons are (1) by inhalation, (2) by mouth, (3) through the skin, and (4) through the subcutaneous tissues. Broadly speaking, the respiratory route is characteristic of most industrial poisons.

Medical Questions Arising.—Because an occupational disease is related to the personal activity of the worker and is of the nature of the inevitable consequences of a given type of work, medical questions are bound to arise in the settlement of occupational disease claims.[13] Besides the nature, extent, and duration of disability, questions of etiology and differential diagnosis need to be established. To solve these questions the industrial physician should have a clear conception of the time factor in the evolution of these diseases. Related to this time factor is a latent period without disability somewhat analogous to the incubation period of infectious diseases. In silicosis, for instance, it may require the passage of from 2 to 25 years of industrial dust exposure before the disease manifests itself clinically in a given worker. Needless to say, it is important to have a record of the nature of exposure in different types of work in the form of an *occupational history.* This may be a time-consuming inquiry, particularly in the case of a miner who has worked in many different mines over a period of 30 years or so, or in the instance of a worker potentially a victim of metal poisoning from whom

the physician endeavors to learn the circumstances, such as the lack of ventilation, chemicals involved, and nature of the work process, which have precipitated the toxic episode. Other steps in diagnosis make the same demands on the physicians's acumen as other disease entities.

In the following pages certain selected occupational diseases, which are manifested by systemic reaction, will be briefly discussed. A chapter on occupational dermatoses appears subsequently. An endeavor has been made to present an epitomized account of the most significant occupational diseases.

Lead Poisoning

Industrial Uses of Lead.—About 150 industrial occupations entail a possible lead exposure. The principal hazards[14] occur in:

- Storage battery manufacture
- Paint industry
- Application of paints
- Enameling of such articles as bath tubs
- Pottery glazing
- Reclamation of lead from junk metals
- Lead arsenate manufacture
- Printing industry
- Welding and riveting, in enclosed spaces, steel painted with red lead
- Rubber manufacture
- Lead ore mining
- Tetraethyl lead manufacture, or cleaning tanks in which ethyl gasoline has been stored

Among the most commonly used lead compounds are lead carbonate, lead chromate, red lead, lead sulfate, litharge, lead acetate, lead arsenate, and tetraethyl lead.

Symptoms.—Lead absorption into the tissues is cumulative in the sense that a part of the relatively small daily doses, individually insignificant, which is absorbed each day, is not eliminated promptly. When physiologic tolerance is exceeded, symptoms and disability occur. Lead enters the body in industrial work principally through the respiratory tract. Organic lead compounds, for example, tetraethyl lead, may enter through the unbroken skin. The maximal permissible concentration for lead is 1.5 mg. per 10 cubic meters of air, and this quantity may be considered as a daily dose which should not be exceeded if disability is to be prevented.

Industrial lead poisoning ordinarily occurs following prolonged exposure to lead or its compounds. Classified on the basis of systems, the three more or less distinct clinical types of lead

poisoning* seen currently among American industrial workers are alimentary, neuromotor, and encephalic. Some cases show a combination of two or more of these principal clinical manifestations.

The *alimentary type* is the most frequent in occurrence. It is characterized by abdominal discomfort or pain which culminates in frank colic in the most severely affected cases. Obstinate constipation is occasionally preceded by a brief period of diarrhea. Among the other complaints in this type of case are loss of appetite, nausea and vomiting, metallic taste, lassitude, insomnia, general weakness or asthenia, arthralgia, myalgia, irritability, dizziness, and headache. Accompanying these symptoms may be the following signs: ashen pallor, lead line on gums, pyorrhea, malnutrition, abdominal tenderness, basophilic stippling, reduced hemoglobin and red blood cell count (but may be within normal limits), slight albuminuria, and elevation of lead content of blood and urine. When a typical episode of colic is in progress, the patient is obviously in agonizing pain, bathed in cold sweat, has gray-green pallor, and is likely to be doubled up with hands pressing upon his abdomen. During a spasm the abdomen has a board-like rigidity. Between spasms, the abdominal pain is usually relieved by firm pressure. The pain of lead colic is usually promptly relieved by intravenous administration of calcium chloride or calcium gluconate.

The *gingival lead line*, if present, will show up as finely punctate bluish-black deposits in the gum tissue. It should not be confused with the congestion of chronic gingivitis, discolored calculus on the tooth surface, or the normal pigment deposits observed in the gums of Negroes and other dark-skinned races. The effects of congestion can be overcome by pressure with a transparent applicator such as a glass slide.

In the *neuromuscular type*, the chief complaint arises from weakness, perhaps the paralysis (wrist drop) of the extensor muscle groups of forearm and hand. The paralysis may be unilateral or bilateral. When unilateral, it is likely to affect the arm most used. If a lower extremity is involved, foot drop may be present. Gastro-enteric symptoms, though not absent, are less disturbing. Arthralgia, myalgia, aching, and stiffness of muscle groups are likely to be more severe than in the alimentary type.

* The presentation of the discussion of the clinical manifestations of lead poisoning follows a section (to be published) of a report prepared by the Committee on Lead Poisoning, American Public Health Association.

Headache, vertigo, insomnia, and disturbed sleep are likely to be prominent symptoms. True palsy is uncommon today; it is usually the result of prolonged and severe lead exposure, and clinical history may give evidence of repeated episodes of intoxication of milder type.

Lead encephalopathy is the most severe but fortunately the rarest manifestation of lead poisoning. In the industrial worker it follows rapid, heavy lead absorption. Certain organic lead compounds, such as tetraethyl lead, are absorbed rapidly (through the skin as well as other portals of entry) into the body and especially into the central nervous system. With these compounds, encephalopathy is the rule. Comparable concentrations of lead are absorbed into the brain from inorganic lead compounds only when the workplace is heavily contaminated with lead vapor, fume, or dust.

Lead encephalopathy begins abruptly and is characterized by signs of cerebral and meningeal involvement. The patient may be in a heavy stupor at the onset and go into coma, with or without convulsion, and die. Excitation, confusion, and mania occur less frequently. Headache, dizziness, insomnia, and somnolence are symptoms in cases recovering and of shorter duration.

The cerebrospinal fluid may be increased in pressure and show slight increase in cellular elements and globulin.

Laboratory Findings.—Laboratory findings supplement the clinical findings and are of considerable assistance in differential diagnosis. They should not be expected to yield the diagnosis.

Evidence of the effect of lead on the hematopoietic system is ascertained by a study of the blood picture. Basophilic granulation or stippling of erythrocytes should be ascertained in quantitative terms and related to an established normal standard. According to Mayers,[18] abnormal cell morphology and abnormal cells (including nucleated erythrocytes) are more characteristic than stippled cells and polychromatophilia of lead anemia, even in such cases in which the hemoglobin is as high as 80 per cent and there is a red blood cell count of 4 million.

The blood of normal North Americans has an average lead content of 0.03 mg. (range 0.01 to 0.06) per 100 gm. of whole blood.[18] Normal urinary lead values average 0.03 mg. per liter with values ranging from 0.01 to 0.08 mg. per liter or ranging from 0.005 to 0.12 mg. per liter, depending on the size of the sample submitted for analysis. The finding of abnormal quantities of lead in blood and excreta means only abnormal lead ab-

sorption and hence points to the existence and severity of lead exposure.

Lead intoxication[15] occurs rarely if the *mean urinary* lead concentration of representative groups of workers is kept below 0.10 mg. per liter, and if individual results are generally below 0.15 mg. per liter and very rarely in excess of 0.20 mg. per liter. The upper limit of safety for the concentration of lead in the blood lies somewhere between 0.05 and 0.07 mg. per 100 gm. The blood levels in frank cases of lead intoxication are usually considerably higher (0.09 to 0.30 mg. per 100 gm. of whole blood). Samples for chemical analysis to determine lead content of blood or excreta should be obtained near the height of an acute episode. Extreme care is necessary to avoid contamination from the time of taking the sample until it is completely analyzed.[17]

Differential Diagnosis.—The *alimentary type* must be differentiated from such conditions which may require surgical intervention, as acute appendicitis, acute cholecystitis and cholelithiasis, perforated peptic ulcer, intestinal obstruction, and acute pancreatitis. Neglect of a surgical condition is far more serious for the patient than giving undue weight to apparently significant lead exposure. In such cases "it is better to err on the side of surgical exploration." Jaundice in lead poisoning is rare today. Leucocytosis, and abnormal differential count, would favor inflammatory lesions. Hematuria is very rare in lead colic cases. Intestinal obstruction is difficult to differentiate, but if stippling is absent lead colic may be eliminated. The medical history will afford material differential points and it is particularly helpful in cases of peptic ulcer or coronary thrombosis.

With respect to *lead neuropathy or encephalopathy*, neurologic changes induced by viruses, infections, arsenic, malnutrition, and alcoholism must be ruled out.

Treatment.—Treatment may be briefly summarized as follows: (1) discontinue the worker's exposure to lead, (2) treat the acute episode with large doses of calcium and calcium-rich diet, (3) later induce catharsis, (4) during convalescence active deleading procedures[18] may be instituted, although some investigators assert that the body will gradually rid itself of excess lead if lead exposure has ceased, and (5) treat cerebral symptoms and sequelae palliatively.

Generally speaking, on recovery from acute lead poisoning, the worker may return to his former occupation provided the lead exposure responsible for his disability has been brought

under control or eliminated. Otherwise, it will be necessary to place him in a job entailing no exposure.

Periodic occupational examinations or check-ups to detect early evidence of dangerous lead absorption should be performed on all workers exposed to a lead hazard, but their frequency must be related to the problem at hand. Workers who are exposed to less than 1.5 mg. of lead per 10 cubic meters of air need not be examined regularly more than once or twice a year, but in extremely hazardous exposures and in young employees it may be necessary to raise this frequency to every fortnight.

Metal Fume Fever

Metal fume fever is an acute transient illness often referred to as brass founders' ague, metal shakes, oxide chills, brass chills, galvo, and zinc oxide fever. Although at one time the disease was thought to be caused exclusively by zinc,[19, 20] it is now known to be produced by other metals, for example, cadmium, lead, manganese, mercury, and magnesium. It follows the inhalation of rather heavy concentrations of finely dispersed metal fumes, usually in the form of oxides. Then, under certain circumstances, toxic proteins or albuminates of the metals are said to be formed which produce a severe transient febrile reaction resembling protein shock in nature and symptomatology.

Symptoms.—A few hours after exposure,[21, 22, 23] the nose, throat, and substernal region feel dry and sore, burn, and give rise to a dry cough. A feeling of constriction in the chest, headache, and lassitude may be complained of and sometimes nausea and vomiting occur. Symptoms at this stage are similar to the prodromes of an acute respiratory infection. Within one to several hours, the symptoms become aggravated, the headache becomes worse, vision may become blurred as chilly sensations begin to appear, and the victim usually takes to his bed. Shivering or trembling rapidly increases into a more or less severe rigor which may last from ½ to 2 or 3 hours. Fever and leucocytosis[23] not uncommonly accompany and follow the chill. Myalgia and arthralgia are also usually present at this stage. The symptoms associated with the chill end almost by crisis and are followed by profuse perspiration. Considerable prostration usually follows an attack but by the next morning recovery is usually complete. An entire attack seldom lasts longer than from a few to 20 hours, and for this reason compensation is rarely claimed.

New workers and employees upon their return to work fol-

lowing a holiday or lay-off are particularly susceptible to an attack. Workers become "immunized", but this artificial immunity lasts only about 5 days.[14] The illness is also more likely to develop in winter and is aggravated by chilling the body. If symptoms persist for more than a day, it is necessary to look to lead, manganese, cadmium, and arsenic as specific causes. Malaria, influenza, tuberculosis, leukemia, Hodgkin's disease, acute bronchitis, onset of tonsillitis, and septic processes also must be considered in differential diagnosis.

Control.—Sayers[24] is of the opinion that metal fume fever may be eliminated through the adoption of an adequate medical and engineering program. A study of the industrial exposure responsible for the illness should be requested by the industrial physician. In conducting preplacement or transfer examinations, it should be remembered that the clinical course of chronic respiratory conditions, such as bronchiectasis, asthma, and arrested tuberculosis, and chronic heart disease may be unfavorably influenced by the fever and chills.

Cadmium Poisoning

The great increase in the use of cadmium, not only for coating marine hardware but also for many fittings that were formerly zinc coated, has created a new problem in industrial hygiene. Most cases of industrial cadmium poisoning have resulted from accidents or short exposure to excessive concentrations of cadmium dust or fume. Little is known of chronic effects upon humans. Acute industrial poisoning is characteristically produced by inhalation of the fumes, particularly where cadmium has been heated to give off the oxide in yellowish-brown fumes.

Symptoms.—The clinical picture[25] is characterized by irritation of the respiratory mucous membrane which may eventually lead to pulmonary edema, pneumonitis, or bronchopneumonia. The first symptoms are those of metal fume fever, usually dryness of the throat, cough, chills, headache, vomiting, and a sense of constriction of the chest. Later symptoms are predominantly referable to the respiratory system and are characterized by cough, pain in the chest, severe dyspnea, and prostration. A few cases have gastro-intestinal complaints.

Differential Diagnosis.—Poisoning with nitrous fumes and methyl bromide may be ruled out by history. Metal fume fever caused by zinc oxide fumes usually clears up within 24 hours. Therefore, the continuance and aggravation of symptoms such

as occur with irritant gases which produce acute pulmonary congestion make it imperative to consider cadmium. These serious delayed symptoms[27] should not be confused with nonoccupational diseases.

Treatment.—Treatment is palliative. Oxygen should be used in moderately and severely affected cases without waiting for signs of pneumonia. Rest is acutely essential as is the withdrawal of the worker from the source of contact.

Manganese Poisoning

Manganese is used principally in the manufacture of alloys, for example, ferromanganese, and to a lesser extent in the manufacture of dry cell batteries, paints, matches, and fireworks, and in leather tanning. In the crude black ore most of the manganese is present in the form of the dioxide.

Symptoms.—Industrial manganese poisoning is a chronic disease characterized by neurologic symptoms and is usually the consequence of inhaling manganese dust or fumes. The important symptoms are muscular stiffness, twitching and incoordination, giving rise to difficulty in walking, and propulsion gait. Speech defects, a mask-like facial expression, drowsiness, weakness, and emotional instability may also be present. In *differential diagnosis*, disseminated sclerosis, paralysis agitans, and progressive lenticular degeneration must be ruled out.

Control.—Flinn, Neal, and Fulton[28] advise quarterly medical examination of workers exposed to manganese dust and the immediate transfer to a manganese-free environment of any workers showing signs of early poisoning, until the hazard has been controlled.

Mercury Poisoning (Hatters' Shakes)

The principal ore from which mercury is derived is cinnabar, or mercuric sulfide. Some cinnabar mines also yield native quicksilver. In 1940 there were 159 mercury-producing mines[29] in the United States and Alaska, with the greatest production coming from the States of California, Oregon, and Nevada. Besides the mining of mercury, some other potential sources of mercury hazard arise from its use in thermometers, barometers, extraction of gold from its ores, dental alloys, mercury arc lamps and rectifiers, anti-fouling marine paints, agricultural disinfectants, radio equipment, analytical laboratories, explosives, and certain chemical industries. Dublin and Vane[11] list about 100 occupations in

which mercury may be a hazard. Mercury is being replaced in the fur-hatting industry by less toxic chemicals.[30]

Industrial mercury poisoning occurs almost exclusively from the inhalation of mercury vapor or dust of the metal and its salts, yet poisoning through the ingestion, cutaneous, and subcutaneous routes may infrequently occur.[31] Elemental mercury gives off vapor at ordinary room temperatures. Mercury is "a general protoplasmic poison." After it gains entrance to the circulation, it is rapidly taken up by the tissues. The form in which mercury circulates in the body is not definitely known[32] though some feel it is as an albuminate such as mercury chloro-albuminate, or oxy-chloro-albuminate.

Symptoms.—The cardinal symptoms of industrial mercurialism are stomatitis, psychic disturbance, and tremors. These symptoms are not present simultaneously in all cases nor in the same degree.[33] Industrial mercury poisoning is typically chronic though cases of severe, rapidly developing mercurialism characterized by colicky pain, diarrhea, painful stomatitis, and excessive salivation may occur occasionally "in such jobs as mining metallic mercury, when the silver runs free, as the miners say, and the mine is hot."[34] There is rarely much kidney involvement in the industrial form of the disease. A blue line on the gums resembling that due to lead absorption is seen in a few cases. Tremor and other signs of neurologic origin are more characteristic of an insidious, slow form of poisoning. The tremor is observed mainly in the muscles of the face, hands, and arms; it is intention in type, becoming most apparent while the patient is doing an unusual task. As the tremor grows worse, shaking or convulsive movements are added to the tremor, giving rise to the typical picture of hatters' shakes. Mercurial erethism or psychic irritability is intimately related to the tremor and may include or lead to loss of memory, insomnia, and depression. Hyperactive knee jerks and scanning speech are frequently present in advanced cases.[35]

Mercury fulminate rarely produces symptoms of systemic mercurial poisoning; the cases are usually characterized by a *dermatosis* associated with conjunctivitis and inflammation of mucous membranes of the nose and throat.[36]

Radium Poisoning

Because luminous dials are needed on instruments in night operations of the armed forces, there has been a great increase

in the use of luminous paints. These paints are usually a mixture of phosphorescent zinc sulfate and radium, mesothorium, or other similar radioactive substances.[37] Another source of industrial disease is in the mining and refining of radioactive ores.

The *harmful effects* of radium are caused by *ingestion*, *inhalation*, *injection* of radioactive substances, or *whole body exposure* to gamma radiation. It is necessary to understand the genetic connection of all members of the radium family because these elements taken all together produce the effects ascribed to radium.

The damage produced by various types of radiation is determined by their nature and properties,[38] thus, (1) alpha rays have enormous energy but low power of penetration and to produce injury they must come in intimate contact with the tissues they injure, (2) beta rays penetrate several feet of air, and (3) the gamma rays can penetrate several centimeters of lead.

All radium[39] disintegrates at a slow but definite rate into radon, a radioactive gas. In the body of a victim of radium poisoning, some of the radon is exhaled and that which is not exhaled disintegrates *in situ* into a series of solid radioactive substances, eventually becoming lead. Like lead, radium has been shown to be stored largely in the bones. Radium induces bone injury as well as damage to the hematopoietic system. It is eliminated mainly in the feces. Although only about 10 per cent of the amount of radium taken into the body becomes fixed in the tissues, this fraction exposes the tissues to the destructive action of the radiations from the various members of the radium family. Among dial painters tissue damage is almost entirely due to *alpha* radiation, as studies of bone deposits show that about 92 per cent of the radiation is *alpha* and only 8 per cent *beta* and *gamma*.

The common industrial form of radium poisoning,[40] such as occurred in radium dial painting, should not be confused with the deleterious effects of external application. The industrial form is an insidious chronic disease. The patient for many years after exposure remains in good health. According to Martland[40] patients having 120 to 180 micrograms of radium in their bodies will usually develop extensive radiation osteitis within 1 to 5 years. A complicating bacterial, dental infection will not uncommonly lead to extensive necrosis of the jaw bones. Macrocytic, hyperchromic anemia may develop at first, followed by aplastic anemia. Patients retaining 2 to 20 micrograms of radium often escape jaw necrosis, but tend to develop crippling bone lesions

such as coxa vara, osteoporosis of the flat bones of the skull, deformities of the spine, spinal fractures, and osteogenic sarcomata.

Even such small amounts as 1 to 2 micrograms may produce definite bone change.[41] When the amount of radium a worker has deposited in his body exceeds 0.1 microgram, as revealed by the expired air test, immediate change of his occupation and treatment by decalcification therapy or other mode of therapy which may have been developed is recommended.[37] Curtiss[36] states that if a sample of exhaled air is found to contain more than 10^{-12} curie of radon per liter, it indicates that at least 0.1 microgram of fixed radium exists in the body.

Treatment is largely symptomatic. The decalcifying therapy of a low calcium diet and ammonium chloride, as used by Aub and his coworkers, has been shown to increase the excretion of radium, but it does not greatly reduce the total deposition of radium in the body. This method of therapy is possibly of value early in the disease when the radium is contained in the trabeculae rather than in the cortex of the bone.

As regards *medical control*, thorough medical and dental examinations should be performed before employment. The complete blood count made at the time of preemployment examination serves as a reference index for subsequent blood counts. Periodic occupational examinations including hematologic studies should be made at intervals of about one month and particular attention given to the trend of successive blood counts. Many authorities prefer to have the expired air radon test, which is made at intervals of six months or one year, because a dangerous radium accumulation may be detected in this way before it has had time to induce changes in the blood picture. Leucopenia, relative lymphocytosis, or beginning anemia calls for careful investigation and possibly change of occupation for the worker concerned.

Silicosis (Occupational Pulmonary Fibrosis, Pneumoconiosis)

Definition.—Pneumoconiosis is a broad generic term applied to all dust affections of the lungs. In a more restricted sense, it means pulmonary fibrosis induced by inhaled *mineral* dust. The committee on Pneumoconiosis of the American Public Health Association[42] defines 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."

Classification.—On the basis of the structural changes induced in the lung and the type of dust, the pneumoconioses or pulmonary fibroses may be classified as follows (compare reference 43):

1. *Simple benign pneumoconiosis*, which is virtually a deposition of dust in the pulmonary tissues usually accompanied by pigmentation, and includes, for example, anthracosis induced by coal dust and siderosis induced by iron. It does not incapacitate and roentgenologically shows as a maximum change only an exaggeration of linear pulmonic markings and is of clinical interest only by way of differential diagnosis. Such cases should never be diagnosed as silicosis.
2. *Silicosis*, which is a pathologic pulmonary reaction due to free silica; for example, sandblasters' silicosis. This type of pneumoconiosis results in a classical nodular fibrosis of both lungs, demonstrable both in the X-ray and at post mortem. Massive fibrosis, varying degrees of pigmentation, atelectasis, emphysema, fibrous pleurisy, bronchitis, cavitation, and pneumothorax are among the concomitant pathologic changes.[44]
3. *Mixed forms*, that is, silicosis resulting from the inhalation of mixtures of varying amounts of free silica and more inert pneumoconiogenic dust constituents, for example, anthracosilicosis and siderosilicosis. Most cases of silicosis undoubtedly come under this classification.
4. *Asbestosis*, a characteristic diffuse, interstitial fibrosis of the lungs induced by fibrous minerals which, according to animal experimentation, is due to the mechanical action of the asbestos, producing a ground-glass appearance on the X-ray. It may cause disability and a few fatal cases have been recorded.

Etiology.—Silicosis is incurable and large numbers of workmen are potentially exposed to conditions favoring the development of the disease. The majority of cases of this chronic pulmonary disease occur among workers engaged in mining,[45] quarrying, ceramics industry, tunnel construction, sandblasting, and foundry work.

Inhalation of siliceous dust almost invariably results in sili-

cosis if a certain set of conditions has prevailed during the working experience of the worker. Among these silicosis-producing conditions are:

1. The dust must be of respirable size (usually 0.5 to 3 microns).
2. The dust must be present in the atmosphere at the breathing level of the worker in concentrations exceeding 5 million particles per cubic foot.
3. The dust must be inhaled for a number of years.
4. The dust must contain silica in a free state, such as quartz.

Generally speaking, the inhalation of high concentrations of respirable high quartz dust produces disabling disease in a shorter period of time than the inhalation of low concentrations of dust or dust of low quartz content.

Legislation.—The following States, which include the 11 States subscribing to blanket coverage, either list silicosis in schedule occupational disease laws, or make certain provisions for the disease:[46] Arkansas, California, Connecticut, Delaware, Idaho, Illinois, Indiana, Kentucky, Maryland, Massachusetts, Michigan, Missouri, Montana, New York, North Carolina, North Dakota, Ohio, Pennsylvania, Utah, Washington, West Virginia, and Wisconsin.

Incidence.—It has been estimated on the basis of the 1930 census that between 500,000 and 1,000,000 workers are exposed to silica dust.[45] Clinical investigations, including X-ray studies, in various dusty trades have shown that from 8 to 25 per cent of the employed workers have potentially disabling pneumoconiosis; thus a high proportion of workers in these dusty trades would appear to have escaped the disease. This resistance of some workers, however, is more apparent than real[47] if consideration is given to the relatively long latent period required before the disease can be demonstrated clinically or even by X-ray study.

A great majority of the cases develop after at least 7 years of exposure, although a few cases have developed in as short a period of time as 1½ years. At the other extreme, with exposures to low concentrations of free silica, more than 30 years may have to elapse before the disease develops to a stage when it can be diagnosed.[45]

Symptoms and Signs.—The worker showing X-ray evidence of an early to a moderate amount of simple silicotic involvement has few symptoms. Moreover, symptoms and physical signs are of little help in determining whether the patient has a silicosis, a modified silicosis (for example, anthracosilicosis), or asbestosis.

Symptoms and signs do, however, assist in the determination of disability. Among the symptoms and signs of importance are shortness of breath, particularly upon exercise; cough, usually dry; chest pain, varying from a feeling of tightness in the chest to the sharp, excruciating pain typical of pleurisy; hemoptysis; general complaints such as digestive disturbances, insomnia, and dizziness; decrease in chest expansion; prolonged expiration, especially in association with emphysema; altered breath sounds; rales; and the presence of areas of increased density in the lungs. Infection may complicate the picture at any time and is usually manifested by pleural pain, fever, night sweats, weight loss, aggravation of dyspnea, anorexia, weakness, and a cough producing large amounts of blood-tinged sputum.

Diagnosis.—In establishing a diagnosis of one of the pneumoconioses, all of the following three factors should be considered.*

1. An occupational history which reveals definite prolonged exposure to siliceous dust or asbestos dust.
2. Symptoms and physical signs which furnish valuable information in (*a*) gauging the extent to which pneumoconiosis has progressed, (*b*) showing the degree of disability, and (*c*) excluding other diseases.
3. X-ray findings, which, if classified on the basis of the system recommended by the U. S. Public Health Service, show bilateral ground-glass, nodular, or a more advanced type of lung-field marking.

So long as the chest roentgenograms show predominantly linear pulmonic markings, silicosis or modified silicosis need not be given serious consideration. It is when the shadows in the lung field assume a ground-glass, granular or nodular appearance that they become specific and assume more diagnostic characteristics. Using Irvine and Steuart's analogy,[48] the usual linear pulmonic markings are likened to the branches of a tree and the granular, stippled, or nodular lung-field markings simulate the leaves. Complete foliation then means silicosis. Viewed stereoscopically, the films at this stage will show fine nodulation. Some writers describe a related change in the lung-field appearance as reticulation.[49] Although not definitely determined, this type of lung-field marking may result from the inhalation of mixed dusts. Later stages will show massive areas of fibrosis, and if infection is present the X-ray shadows tend to be asymmetrical.

Asbestosis is exceptional from the standpoint that the pul-

* In the field of forensic medicine it has been frequently demonstrated that autopsy studies will yield essential information.

monary fibrosis is caused by dust containing little if any free silica. The asbestotic patient will usually show clinical symptoms and signs which are out of proportion to the apparently small amount of pulmonary involvement shown by the chest roentgenogram. Large massive shadows are very rarely seen in the chest roentgenogram except in the presence of a complicating infection. On the contrary, the diffuse interstitial fibrosis is manifested by a ground-glass appearance, frequently with fine pinpoint stippling of the middle and lower lung fields. It progresses to terminal diffuse fibrosis usually without nodular predominance in about twenty years.[58] A shaggy appearing cardiac silhouette due to involvement of superimposed lung structures and pleuropericarditis is not infrequent. Asbestosis bodies may be demonstrated in the sputum or the lung tissue.

Medical Control Measures.—All applicants for employment in dusty trades should be examined by X-ray. Periodic medical examinations at intervals of one year to possibly three years, including X-ray study of the chest, should be made of all workers in dusty trades in order to detect evidence of active pulmonary tuberculosis and early silicotic changes. The length of the interval between examinations depends mainly on the degree of hazard and the prevalence of endemic tuberculosis.

Clinical study of patients suspected of having pulmonary tuberculosis should be made to determine the dynamic status of such complication. No worker should be rejected on preemployment examination or removed from work, which he is accustomed to perform, merely because of a diagnosis of simple silicosis, but rather the atmospheric dust in which he works should be brought within safe limits. The worker whose first roentgenogram shows healed primary tuberculosis should not be denied employment in a dusty trade on this account alone. If the worker has minimal, arrested, or healed reinfection tuberculosis, he should be allowed to continue his work but should be observed with the same precautions as a man with simple silicosis. Close medical supervision is recommended for all silicotic workers in order to control or prevent serious complications of the common respiratory infections.

Benzene (Benzol) Poisoning

Benzene (benzol) is an excellent solvent for gums, resins, fats, and oils, and as such has found many industrial applications. It is used in the manufacture of rubber, rubber goods,

linoleum, quick drying paints, lacquers, stains, paint removers, and plastics. Benzene should not be confused with the less toxic petroleum product, *benzine*, which is a mixture mainly of aliphatic hydrocarbons.

The most characteristic *pathologic changes*[51] in cases of benzene poisoning are seen in the bone marrow which may show gradations of change from hyperplasia to hypoplasia and occasionally complete aplasia of the myeloic cells. Other parts of the hematopoietic system may also be involved. Depending on the degree of exposure, secondary degenerative changes are observed in the liver, kidneys, and heart.

Symptoms.—Acute benzene poisoning follows the inhalation of benzene vapors in high concentration and provokes narcotic symptoms such as inebriation, fatigue, sleepiness, vertigo, tinnitus, nausea, vomiting, and headache. If exposure is prolonged, muscular twitching, convulsions, paralysis, and loss of consciousness may result. With very large doses, unconsciousness, convulsions, and death due to respiratory paralysis may occur rapidly.

The more typical industrial benzene poisoning is *chronic*, and is a complex hematologic syndrome characterized by anemia, purpura, and granulocytopenia. The associated subjective complaints are fatigue, somnolence, headache, vertigo, general debility, and gastro-intestinal disturbances. The *blood picture* may be variable. A drop in white cell count (especially involving polynuclears) below 5000 to 5500 is usually considered a sign of incipient poisoning. Anemia and corresponding change in hemoglobin usually occur after toxic effects on white blood cells become manifest. Leucocytosis, eosinophilia, and polycythemia are occasionally observed.

The *urine* may contain albumin, casts, and bile pigments.

In the proper placement of personnel, juvenile and pregnant workers and those suffering from chlorosis, tuberculosis, organic heart disease, hemorrhagic diathesis, and anemia should ordinarily be excluded from positions entailing a hazardous exposure.

Medical Control.—Occupational examinations of exposed employees, including blood studies, should be made at intervals of approximately one month, gauging the frequency to severity of exposure. The ratio of inorganic to total sulfates should be determined, a reduction of which will indicate the existence of exposure to benzene.[52] In case, upon repeated examination, the percentage of organic sulfate is 30 per cent or more, the concen-

tration of benzene in the air of such operations should be determined and reduced by proper engineering methods.[31]

Carbon Monoxide Poisoning

Carbon monoxide poisoning may occur in a great number of industrial operations.[32] The gas, CO, is formed by the incomplete combustion of organic materials. Its action on the body is related to its affinity for hemoglobin which is 300 times that of oxygen. When inhaled it forms carbon monoxide-hemoglobin, and thus causes anoxemia in proportion to the amount of carbon monoxide-hemoglobin in the circulation. Carbon monoxide poisoning is usually acute. Whether or not chronic carbon monoxide poisoning exists as an entity is controversial and seems largely dependent on the interpretation of the word "chronic." It appears that continued exposure to moderately toxic concentrations will result in disturbances of the circulatory and nervous system.

Symptoms.—Blood saturation up to 15 per cent HbCO rarely produces symptoms, but when the blood saturation is from 15 to 20 per cent, tightness across the forehead, possibly slight headache, and dilation of cutaneous blood vessels are observed. When the blood saturation is 30 to 40 per cent, the common symptoms are severe headache, weakness, dizziness, dimness of vision, nausea and vomiting, and collapse. Coma with intermittent convulsions, depressed heart action, and possibly death are symptoms occurring with HbCO concentrations of 60 to 70 per cent. With exercise, latent symptoms often become manifest and existing symptoms are aggravated. On exposure to high concentrations, the victim may notice few, if any, symptoms, yet he may without warning become unconscious and die without regaining consciousness.

Treatment.—The treatment of carbon monoxide poisoning should always be carried out by a qualified physician, although first aid must be given pending his arrival. In summarizing experience with the treatment of carbon monoxide poisoning, the following procedure, outlined by Sayers,[33, 34] is recommended:

1. The victim should be removed to fresh air as soon as possible.
2. If breathing has stopped, is weak and intermittent, or present in but occasional gasps, artificial respiration by the Schäfer method should be given persistently until normal breathing is resumed or until after the heart has stopped.
3. Pure oxygen or a mixture of 5 per cent carbon dioxide and 95 per cent oxygen should be administered using an inhaler, beginning as soon as possible and continuing for at least 20 minutes in mild

cases and as long as 3 hours, if necessary, in severe cases if the patient does not regain consciousness. The administration of oxygen or of the mixture of carbon dioxide and oxygen when given immediately will greatly lessen the number and severity of the symptoms from carbon monoxide poisoning and will decrease the possibility of serious after-effects.

4. Circulation should be aided by rubbing the extremities of the patient and keeping the body warm with blankets, hot-water bottles, hot bricks, or other devices, care being taken that these objects have been wrapped or do not come in contact with the body and cause burns.
5. The patient should be kept at rest, lying down to avoid any strain on the heart. Later he should be treated as a convalescent and should be given plenty of time to rest and recuperate. Exercise was at one time recommended; however, the procedure is hazardous, as the patient quite often loses consciousness, and in some cases death occurs.

CONCLUSION

Occupational disease legislation has been discussed and certain occupational diseases have been briefly described. It is apparent that in the practice of industrial medicine, many questions of compensation arise. It is for this reason that the industrial physician should have a thorough knowledge of the occupational disease laws applying to the workers for whom he is responsible. Many of the questions that arise in connection with such compensation are medical in nature and demand medical solutions.

As more and more handicapped workers must be taken into industry, deficiencies in present compensation laws become apparent. Legislation amending certain of the laws is indicated in many instances, and, in others, enlightened administration of the law is needed, particularly as related to waivers and second-injury provisions.

In his daily practice, the industrial physician should secure accurate and complete occupational and other histories, perform thorough physical examinations, and, in the instance of a claim, submit his impartial report promptly to the administrator of compensation.

The physician has a major role to play, not only in the treatment but also in the control and prevention of disease in industry.

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CHAPTER 12

MEDICAL CONTROL OF RESPIRATORY DISEASES

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THE PROBLEM

TODAY, as never before, the comparative lack of specific control measures and the terrific toll of the respiratory diseases—colds, influenza, pneumonia, and tuberculosis—are making rigid demands upon the acumen of every industrial physician, and at the same time cast out a challenge to preventive medicine which cannot be ignored. Fully one-half of the sick absenteeism and over one-third of the work days lost by industrial workers through disability are attributable to respiratory disease. The strategic position of the plant physician for the control of these diseases is not as favorable as it is in the case of such diseases as malaria and syphilis for which specific chemotherapeutic agents are available or in the case of smallpox against which active immunity can be attained.

Workplaces not infrequently provide many of the predisposing factors regarded as significant in respiratory infections. Many workers for the first time in their lives are coming face to face with such conditions as a relatively crowded environment and exposure to gases, fumes, and dust. They must contend with occupational fatigue in becoming hardened to a new form of work. Many are exposed to inclement weather and extreme changes in weather conditions. Malnutrition, allergy, alcoholism, personal handicaps, and other factors contribute to the decreased ability of the individual to resist infection.

It is the purpose of this chapter, first, to review certain measures as they pertain to the control of acute respiratory diseases, and second, to outline similar measures as they apply to tuberculosis and other chronic pulmonary diseases in industry.

ACUTE RESPIRATORY INFECTIONS

The acute respiratory infections are a heterogeneous group of ailments caused by various agents. It is highly probable that many of the so-called colds are caused by filterable viruses. More

than thirty types of pneumococci are involved in pneumococcal pneumonia; more than two types of viruses are known to cause epidemic influenza. The filterable viruses are particularly likely to initiate the disease process. Moreover, it is now recognized that the common pathogens of the respiratory tract participate in the pathogenesis of acute respiratory syndromes. Only too frequently is the common cold followed by such formidable sequelae as pneumonia and tuberculosis.

Prevalence of Colds

The extensive studies by the U. S. Public Health Service and other organizations have contributed many important facts regarding the common cold. Since it is obviously impossible to present a complete review, only certain etiologic and epidemiologic features will be stressed. The attack rate per person usually averages 2 to 3 per year. Almost all persons are susceptible and very few persons avoid an attack of acute respiratory infection during the course of any one year. Townsend,[1] for instance, stated that in a group followed for 5½ months 90 per cent reported one or more attacks. There is typically, but not always, a period of high prevalance in early spring, a decline in midsummer, another period of high prevalence in autumn, and some decline in December. The peak in the fall of the year is reached with almost explosive suddenness and a high incidence will usually be reported simultaneously in all sections of the country.

Gover, Reed, and Collins,[2] studying data from students in various universities in the United States over a period of 18 months, concluded that there is "no definite association of respiratory attack rate with marked variations in climate as represented by six American cities with wide geographic and climatic differences."

Other investigators[3] have also failed to observe a consistent relation between the incidence of colds and latitude, longitude, and climate.

The above conditions apply to continental United States, but, lest we overlook the infectious nature of colds, the Spitzbergen observations[4] may be recalled. Spitzbergen is comparatively free of colds in winter, but with the opening of the shipping season about three-fourths of the community's population suffer an attack of the common cold within a week following the arrival of the first ship. Similar observations, as well as clinical and experimental investigations, indicate that the incubation period

for the common cold is probably between 12 and 48 hours or possibly as long as 72 hours.

Cold Vaccine

Prophylactic immunization against the common cold has been tried by means of filterable virus vaccine and by vaccines prepared from mixtures of various pathogens of the respiratory tract. The virus vaccine has had only limited trial and has not proved to be efficacious. According to Dochez,[5] the future value of common cold virus vaccine is problematical because an attack of cold gives little or no immunity. Bacterial vaccines containing a variety of respiratory pathogens have been used on a wide scale. Many industrial physicians have had first-hand experience with them.[6] The stock vaccines contain killed pneumococci, *H. influenzae*, *M. catarrhalis*, staphylococci, and streptococci. These organisms tend to be present in the normal nasopharyngeal flora and in case of an attack are assumed to act as secondary invaders. As a result of using such vaccines one would expect a lessening in the harmful effect of these organisms. According to some authorities, these secondary invaders play an important role in a small group of adults who suffer each year from prolonged infections of the upper respiratory tract with such complications as sinusitis and bronchitis. For theoretical reasons, some believe that such vaccines are of considerable value if restricted to that group of individuals in which secondary bacterial infection presumably plays an important part. *The use of these vaccines, however, has not received the endorsement of public health workers, mainly because of lack of scientific proof.*

Influenza Prophylaxis

The etiologic agent of certain epidemics of influenza, as in the instance of the common cold, is a filterable virus. Influenza occurs mainly in a pandemic, an epidemic, or an endemic form.[8] The cause of pandemic influenza, such as occurred in 1918–1919, is not yet known. In the epidemic form, at least three filterable viruses have been isolated. The cause of endemic influenza, or sporadic grippe is also undetermined. In influenza such organisms as *H. influenzae*, streptococcus, pneumococcus, and *M. catarrhalis* are regarded as secondary invaders which aggravate the pathologic picture by frequently inducing severe and fatal bronchopneumonias. Immunization would be an ideal method of prevention, but no efficacious procedure has been available in past

epidemics, probably as the cause had not been fully determined. "Individual immunity to the virus of influenza as measured by the development of neutralizing antibodies in the blood can be readily accomplished by inoculation of human beings with either living or killed cultures of influenza virus."[5] Extensive studies are now being carried out with different antigenic strains of the virus. A flu-distemper viral vaccine has been used recently.[7] Horsfall[8] in his recent review, however, concludes that the average extent of immunity induced by vaccines containing influenza A virus was too insufficient to allow it to be considered as a practicable prophylactic measure.

Pneumonia Prophylaxis

Artificial immunization against lobar pneumonia, whether active or passive, is of questionable value for prevention. It is known that immunity develops to pneumococci, but the duration of the immunity thus induced is limited.

Environmental Control

Besides control along immunologic lines, measures directed at environment have recently been receiving increased attention. The new "air-borne" or indirect hypothesis "postulates that the greatest spread of respiratory infection is produced by small dried droplets floating in air for relatively long times and distances or by the resuspension of dried droplets in air after they have settled to surfaces such as floors, clothing, and bed clothes."[9] Alpha hemolytic streptococci of nasopharyngeal origin are widely distributed. The virulence of the microorganism is never more than slightly impaired by sojourn in rooms. One of the outgrowths of these observations as related to surgery is that it has been possible to reduce wound infections by less frequent dressings.

The object in environmental control is to destroy the respiratory pathogens in air either by radiation, or by chemical sprays or aerosols. It has been observed that the concentration of organisms in the air is reduced by natural sunlight; even diffuse daylight has been found to have a lethal effect. These observations suggest planning for maximum window space in occupied buildings.

Ultraviolet radiation, while more potent than visible light, cannot, at present, be unreservedly endorsed for use in industrial plants for the purpose of controlling acute respiratory infections.

If it is used, special attention must be given for protection of individuals, particularly as regards exposure of skin and eyes.

Aerosols.—Various germicides in an appropriate solvent and sometimes incorporating a wetting agent have been used as aerosols. Among the various agents that have been used in experimental work are resorcinol, resorcinol glycerine, hexyl resorcinol in propylene glycol, formaldehyde-water, and hypochlorite solutions. Twort and his coworkers[10] found that 10 per cent hexyl resorcinol dissolved in propylene glycol added to 0.05 per cent sulfonated lorol was the most effective all-around germicidal mixture of those they tested. Minimal effective mist concentration of resorcinol glycerine aerosol was about 1:400,000,000 by volume. This level was confirmed by Williamson and Gotaas.[11] The latter, in referring to the practical use of aerosols in places occupied by persons, state that the aerosol must be nontoxic to man, nonirritating to the eyes and lungs, rapidly lethal to the bacteria when present in small concentrations, inodorous, invisible, noninflammable, noncorrosive, persistent, and should not leave films and coatings on walls and furniture. Needless to say, considerable investigation is necessary before aerosols can be used in occupied rooms. It may be used in such rooms as laboratories and media rooms where the aerial germicide may be allowed to dissipate before the room is occupied.

General Measures in the Control of Colds, Influenza, and Pneumonia

Since no satisfactory immunity can be established against these acute respiratory infections and even the new chemotherapeutic agents are of little avail in viral pneumonias and most other viral diseases, it is apparent that many procedures are necessary to cope with an epidemic of one or all of these diseases. Specific measures of hygiene and sanitation are to be recommended during epidemic periods. Such measures have been tersely summarized for these respiratory diseases, as well as other communicable diseases, in a report of a committee of the American Public Health Association.[12] This pamphlet should be in the hands of every plant medical director.

Because the minor and acute respiratory ailments, such as common cold, not infrequently lead to disabling pneumonia, otitis media, and mastoiditis, a major effort should be directed toward controlling colds. Remembering that colds are spread by close contact in the acute stages of the disease, certain public

health procedures should be seriously considered. These include, among others, (1) isolation at home during the early and highly contagious stage of a cold which will lessen the spread of the infection, and (2) avoidance of close contact with persons suffering from colds, and of closed spaces where large numbers of infected persons congregate, which may be recommended through a health education program.

The spread of these diseases most frequently takes place through the medium of infected particles sprayed into the air by talking, coughing, and sneezing. Where general public health measures for control have been applied, they have included improvement of general hygiene and living conditions, especially overcrowding. Persons suffering from minor respiratory infections should take unusual care in the avoidance of dangerous contacts, allow themselves periods of rest, and avoid such predisposing factors as chilling, exposure to severe climatic changes, fatigue, and excesses of all kinds.

Many procedures in the plant medical department if adopted as routine may aid materially. The preemployment examination should pick out those prone to acute exacerbation, sinusitis, asthma, and bronchitis. Employees should be required to check through the medical department when they become ill on the job and after acute illness. These checks afford an excellent opportunity for directing those ill to prompt treatment by the new therapeutic techniques now available. Contact between persons in the more contagious phases of minor respiratory diseases can therefore be avoided. By thus encouraging prompt treatment, disabling effects may be reduced in number and severity. That an all-around industrial hygiene program will yield substantial results is attested to by a number of recent reports by industrial physicians.

CHRONIC RESPIRATORY INFECTIONS

In addition to the acute respiratory diseases, those of a chronic nature exact a large toll of lost time. These diseases are exemplified by pulmonary tuberculosis, which claims the lives of sixty thousand Americans each year. It is more and more becoming recognized as an affliction of older occupied men.[13] As an example of American industrial tuberculosis experience, it is gratifying to note that the death rate[14] among industrial policyholders of the Metropolitan Life Insurance Company has been reduced to about one-fifth that of 1917. With the changeover to

war production, however, attention must be directed to a possible increase of this disease, particularly as regards women. The single exception to a satisfactory public health situation in Britain was a rise of almost 10 per cent in the mortality from tuberculosis in 1939 and 1940.[15] The increment in this case was greatest in young women and was thought to be related to malnutrition.

Control Programs

By directing medical measures for the control of the chronic respiratory diseases at pulmonary tuberculosis, tangible results are obtainable with nontuberculous affections as well. The essence of the medical program is the radiographic examination of the chest. This procedure has been the basis of medical control of silicosis, in which disease the infective element, particularly tuberculosis, is the principal cause of disability. As in pneumoconiosis, X-ray examination of the chest, if supplemented by clinical study, will aid in obtaining proper medical management for such other pulmonic conditions as bronchiectasis, asthma, and cancer.

As an illustration of what can be accomplished in tuberculosis control by a plant, reference is made to the experience of Sawyer.[16, 17] An X-ray survey of 3,280 apparently healthy employees in 1921 showed 2.3 per cent positive for clinical tuberculosis. From 1928 on, the medical department has been doing serial or annual X-rays of the chest. In a group of 4,665 employees, averaging 33 years in age, and studied from 1928 to 1935, only 0.5 per cent showed active or clinical tuberculosis. By 1940 this percentage was down to 0.2 per cent, which is probably close to the irreducible minimum. What has been done by Sawyer can to a considerable extent be achieved in any employed group if a definite program is outlined and followed.

The Metropolitan Life Insurance Company,[18] following the adoption of a tuberculosis control program, reduced the number of cases of significant pulmonary tuberculosis from 40 per 10,000 employees in 1930 to 10 per 10,000 in 1939. Use was made of routine preemployment X-rays and the Metropolitan sanatorium. It was observed, however, that during the period, 1930 to 1939, the prevalence of tuberculosis among applicants for employment had not been reduced; the experience indicates 80 cases of significant tuberculosis per 10,000 applicants and, according to Sherman,[18] this probably represents a fair average.

Other industries also have had anti-tuberculosis programs in effect. There should be no relaxation in the program of controlling pulmonary infection and indeed an extension of the program to the smaller industrial plants is indicated.

X-ray Methods.—Recent radiographic surveys of employed groups, who had not been X-rayed previously, have revealed 1 to 2 per cent of the employees with significant tuberculosis. Case finding is an important feature in a tuberculosis control program that can be effected by the industrial physician. A number of X-ray procedures are available, such as single or stereo 14 by 17 inch films, 14 by 17 inch paper, 35 mm., 4 by 5 inch, or 4 by 10 inch photofluorographic methods, and fluoroscopic examination.

Photofluorograms have been adapted for mass X-raying work in the armed forces, as well as in the medical departments of a number of large industrial plants. If a grid is used, very satisfactory photofluorograms can be obtained. For best results this will require at least 200 milliampere equipment. It has been the practice not to rely on the fluorograms, particularly the 35 mm. size, for diagnoses, but, instead, this technique is expected to pick out persons with definitely abnormal or suspicious chest findings. These individuals are then rechecked with standard 14 by 17 inch single or stereoroentgenograms taken for diagnostic interpretation.

In advocating small film X-ray pictures as a routine for all patients admitted to general hospitals, Hilleboe[20] refers to the experience of the University Hospital, Ann Arbor, Michigan, where it was found by this technique that 10 per cent of the patients admitted showed lesions of the organs in the chest. The U. S. Public Health Service now has eleven photofluorographic units in the field, each accompanied by a medical officer, technician, and clerk. Plants may appeal to the Public Health Service through the State health department for the services of one of these 35 mm. units if no other means for mass X-raying is available. Experience has shown that 300 to 500 photofluorograms a day can be made with one of the units.

Administrative Procedures.—Sawyer,[16, 17] who has had years of experience administering a tuberculosis control program, offers helpful suggestions in its conduct. His program is directed at three groups: (1) applicants for employment, (2) employees with negative X-rays, and (3) employees with suspected tuberculosis. He has noted that it has been too time-consuming to furnish each individual a report on the findings yielded by his radiogram. Hence, he recommends issuing a mimeographed statement

to the patient at the time of X-ray, stating that if his X-ray is negative he will not receive a report, but if his lungs are affected he will be so notified. Employees having latent, clinically insignificant lesions are reported as having lungs that are negative or clear.

Employability.—Applicants with healed primary tuberculosis or inactive types of reinfection tuberculosis are ordinarily acceptable for employment. For a number of years the Public Health Service has advocated, even in the dusty trades, that the worker with evidence of healed primary tuberculosis should not be denied employment on this account alone. There are many types of employment where persons are acceptable with clinically negative, stabilized lesions, minimal or moderately advanced in extent. If adequate X-ray equipment is available, it may be possible in some instances to accept individuals with small soft lesions, but these workers will require close medical supervision. The individuals with active and questionably active tuberculosis are not acceptable for employment, but should be referred to their physician, the local public health authorities, or a clinic.

Frequency of Periodic X-Ray.—The means toward the end of obtaining satisfactory periodic X-rays is an adequate record system. This will make it possible to call back the employee for reexamination, according to the need for recheck. The Public Health Service has recommended annual medical examination of workers in the siliceous dusty trades to detect evidence of pulmonary tuberculosis and early silicotic changes. Sawyer[21] recommends annual X-ray for young workers (ages 18 to 25), whose preemployment X-ray was negative for clinical tuberculosis, gradually increasing the time interval between examinations to three years if the chest roentgenogram continues to be negative. Individuals 30 years of age and over with negative X-rays at the beginning of employment are scheduled for re-X-ray at 3-year intervals.

The frequency of X-raying workers with evidence of tuberculosis depends on the extent of pulmonary involvement and dynamic status of the disease. Sherman.[19] in the instance of expatients, has suggested X-ray and sputum study every three months for the first two years following their discharge from a sanatorium, and every six months thereafter. He feels that those with a history of contact should be X-rayed at once and every six months for a period of three years and annually thereafter.

Follow-Up, After-Care, Rehabilitation.—Next to finding the case of tuberculosis, adequate follow-up is of extreme importance,

particularly from the patient's standpoint. The patient should be referred to his private physician or to a clinic and the case reported to the local health department. Public health officials and local voluntary tuberculosis agencies are especially helpful in this follow-up stage. They can assist in arranging for sanatorial care and be of material assistance in checking on possible contacts in the home. If sanatoria are not available, outpatient supervision is far superior to no care at all.

In the development and the successful prosecution of a tuberculosis control program in any community, it is essential for public health officers to have the cooperation of the State and local medical society, management, labor, and voluntary agencies. This procedure will insure appropriate follow-up and after-care, and also pave the way for rehabilitation of the affected individual to industry. In this connection, from 65 to 75 per cent of the workers with minimal and moderately advanced tuberculosis can be expected to return to their former positions in industry after an adequate stay in a modern sanatorium. Many industrial organizations reemploy workers with arrested tuberculosis. Factors to be considered in the employment of a worker with arrested tuberculosis are as follows: (1) extent of the lesion, (2) completeness of the cure, (3) character of the job, and (4) the necessity for adequate medical supervision after return to work.

CONCLUSION

In this period of profound socio-economic stress every facility for searching out active cases and getting them under treatment should be employed to prevent an unfavorable effect on the present downward trend of the American tuberculosis mortality rate. The X-ray is a potent tool to this end and the medical departments of many industrial organizations have demonstrated how much can be done in promoting the control of tuberculosis. The plant physician should formulate a program making use of the X-ray for preemployment and periodic examinations of his employees. To provide for satisfactory disposition of cases found, the plant physician should also establish liaison with local workers in the field of tuberculosis control.

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