NATIONAL MEDICAL MONOGRAPHS

INDUSTRIAL MEDICINE

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ABNORMAL ARTERIAL TENSION
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OBSTETRICS FOR THE GENERAL PRACTITIONER
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INDUSTRIAL MEDICINE
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THE MANAGEMENT OF COLITIS
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DISEASES OF THE CHEST
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INDUSTRIAL MEDICINE

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PREFACE

(Where figures are shown in brackets, either in preface or text, reference is made to Bibliography in back of book.)

TO MOST general practitioners Industrial Medicine is an unknown field. Although in many instances the physician is working in an industrial community and caring for industrial accidents and cases of industrial disease in his private practice, his knowledge of the means by which these accidents and diseases have originated is far from clear. It will be our effort in this book to clarify his knowledge of what Industrial Medicine is, to assist him in understanding the conditions under which his patients work, to discuss his contacts with an industrial medical department when one exists in a neighboring industry, and to inform him of standard practice in the industrial medical field.

Industrial Medicine is difficult to define in

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concise terms. Probably the simplest concept is that of complete medical service to the industrial unit involved. This means intramural service only. When workers are not at work in the factory or mine they do not come within the practice of industrial medicine nor do their families. Factories in mill towns occasionally provide medical care for their employees and for their employees' families whether at work or at home. For instance an excellent service of this type is maintained at the Endicott Johnson shoe factory at Binghamton, N. Y., [22] and another excellent service is provided at Roanoke Rapids, Va. [49]. Such experiments in community medicine are interesting but isolated and do not represent true industrial medicine, which is confined solely to the medical problems of the operating industrial unit.

These medical problems may be divided into two groups, maintenance of the health of workers and diagnosis and treatment of minor sickness and of injury. Prevention of industrial disease is largely an engineering problem as its basis is the separation of the toxic or irritating substance from contact with the worker. The Industrial Medical Department, however, must coöperate with the Engineering Department by discovering the health hazard, determining its

distribution, and pointing out its origin. Then only is the engineer able to direct his attack at the source of the danger. To quote Rector, [141] "when the medically trained mind has unraveled the knot, it requires the application of engineering skill to complete the elimination of the hazard either in construction or process. In no other field of medicine is there such a close relation to the engineering profession. Either profession alone is unable to find the answer, and where there has not been close coöperation between the physician and the engineer the problem has remained unsolved." Industrial medicine is the practice of medical supervision, preventive medicine, and public health within the confines of an industry. Its aim is to safeguard the health of the employee from the time of his joining the working force until his discharge. It further endeavors to minimize time lost from work because of sickness or accident by providing prompt and efficient attention and by arranging for adjusted work when necessary.

To quote an editorial in the *Journal* of the *American Medical Association:* [92] "The foremost objectives of industrial medicine are (1) to fit every person to types and quantities of work according to his ability to perform such work continually without undue impairment,

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without injury to himself or to his fellow workman and with profit to himself and his employer; (2) to procure and maintain fitness for work through efforts applied to the worker as an individual, to groups of workers, and to the work environment; (3) to educate the worker to a comprehension of the value and significance of physical and mental well-being and, in particular, of personal hygiene and accident prevention; and (4) to reduce all loss of time, absenteeism and short work spans in industry the cause of which may be related in any way to health."

At this time, January, 1935, the major problem in industrial medicine in the United States is pneumoconiosis and particularly silicosis. This has not been covered in any recent book published in this country, although the medical literature abounds with clinical, radiological, and experimental papers on the subject. In her recent book on *Industrial Toxicology,* Dr. Alice Hamilton says: "Industrial poisoning is not so important a subject as industrial diseases in general, for not nearly so many working people are employed in the dangerous trades as in the dusty trades or in hot and strenuous labor."

Both of us have been actively engaged in the practice and teaching of various aspects of in-

dustrial medicine for some years. Of constant use to us have been the standard books of Rosenau on *Preventive Medicine,* of Kober and Hayhurst on *Industrial Hygiene,* of Hamilton on *Industrial Poisons* in the United States, of Henderson and Haggard on *Noxious Gases,* of Haldane on *Respiration,* and of Gibbs on *Clouds and Smokes.* We have been at some pains not to duplicate or to abridge Hamilton's recent *Industrial Toxicology* and have avoided any attempt to itemize or tabulate the common industrial poisons. This subject, we feel, already has been adequately covered in brief form by Hamilton. We have, however, given some space to the treatment of industrial maladies considered in their broadest sense.

Finally, this book is written as a handbook or reference book for the practitioner who is going into industrial medicine or who may have industrial medicine thrust upon him. We have tried to keep the bibliography short and deliberately have omitted many references to original sources.

W. Irving Clark
Philip Drinker

Boston,
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Chapter I

THE INDUSTRIAL MEDICAL DEPARTMENT

Before the Industrial Medical Department can be discussed, it is necessary to show its position in the factory organization. Factory organization is a very elaborate and complex structure, but for the purpose of discussion, may be considered as having four great divisions. These are Administration, Production, Sales, and Personnel.

The Administration Division supervises all activities in the factory, tying them together and arranging for their financing; the Production Division has to do only with those machines and with those operations which change the crude to the finished product; the Sales Division has for its object the sale of the goods produced, while

the Personnel Division includes all those activities which are neither administration, production, nor sales.

The Industrial Medical Department falls under the management of the Personnel Division. In this division, it is in close contact with the Employment Department, the Safety Engineering Department, and the Legal Department. These contacts will be discussed later.

Functions. The functions of an Industrial Medical Department consist of:

1. Physical examination of applicants for employment.

2. Physical examination at regular intervals of employed workers who have physical defects.

3. Periodic physical examination of those who are working in departments where the manufacturing process involves a health hazard.

4. In some factories reëxamination of the entire force annually.

5. Placement of those workmen found physically defective in departments where the work will not prove injurious.

6. Diagnosis, and in minor cases, treatment

of workers applying to the factory dispensary for medical care.

7. Diagnosis and treatment of injured workers.

8. Diagnosis of all cases having surgical conditions applying for information or care.

9. Spread of health publicity by lectures, leaflets and similar publications.

10. Coöperation, where possible, with the family physician whose patient is a worker in the factory.

Physical examinations. It will be seen at once that the physical examination of workers is a very important part of the medical program. The examination of applicants for employment is made prior to employment in order to prevent contagious disease such as trachoma, pulmonary tuberculosis, active venereal disease, etc., from being introduced into the factory among the working force. It also gives the physician an opportunity to see that the prospective worker is being placed at work for which he is physically fitted. In some factories the examination is not made until after the employee has been definitely

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hired but this is not the best practice. Thus in a survey of 255 plants made by the National Industrial Conference Board [126] as long ago as 1926, in 212 plants examinations were made prior to or at the time of hiring. "Two hundred and three plants reported that the results of such examinations were used as a guide in placing workers, while only 34 plants reported no such use of information obtained. The assignment of workers to tasks for which they are physically fitted not only lessens the liability to accident but prevents undue strain upon a defective worker, thereby conserving his abilities and increasing his efficiency." It is safe to say that this principle is now clearly recognized and carried out in all first-class industrial medical services. If the procedure of placement is carried out it is obvious that the substandard group of men so placed forms a group requiring more medical supervision than the normal vigorous worker. In order to insure this, the worker in such a group is periodically recalled for examination, in order to check up on his defect, and for advice if such is needed. The same applies to

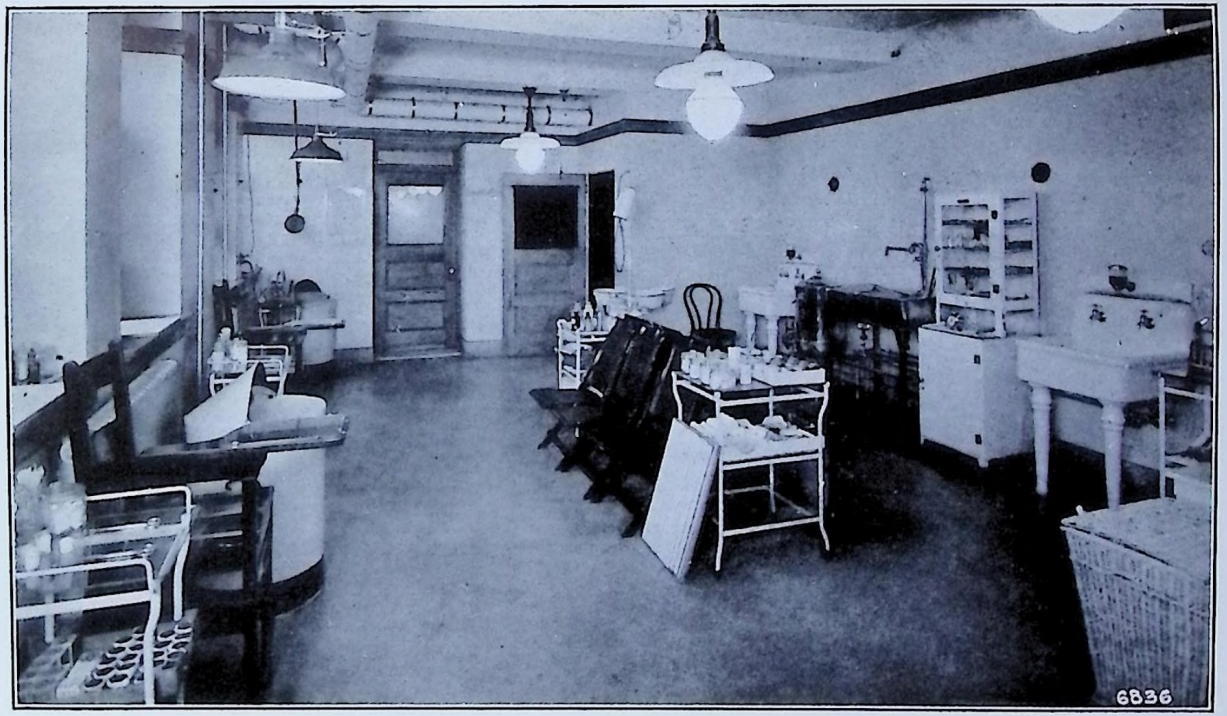


FIG. 1. A factory central dispensary.

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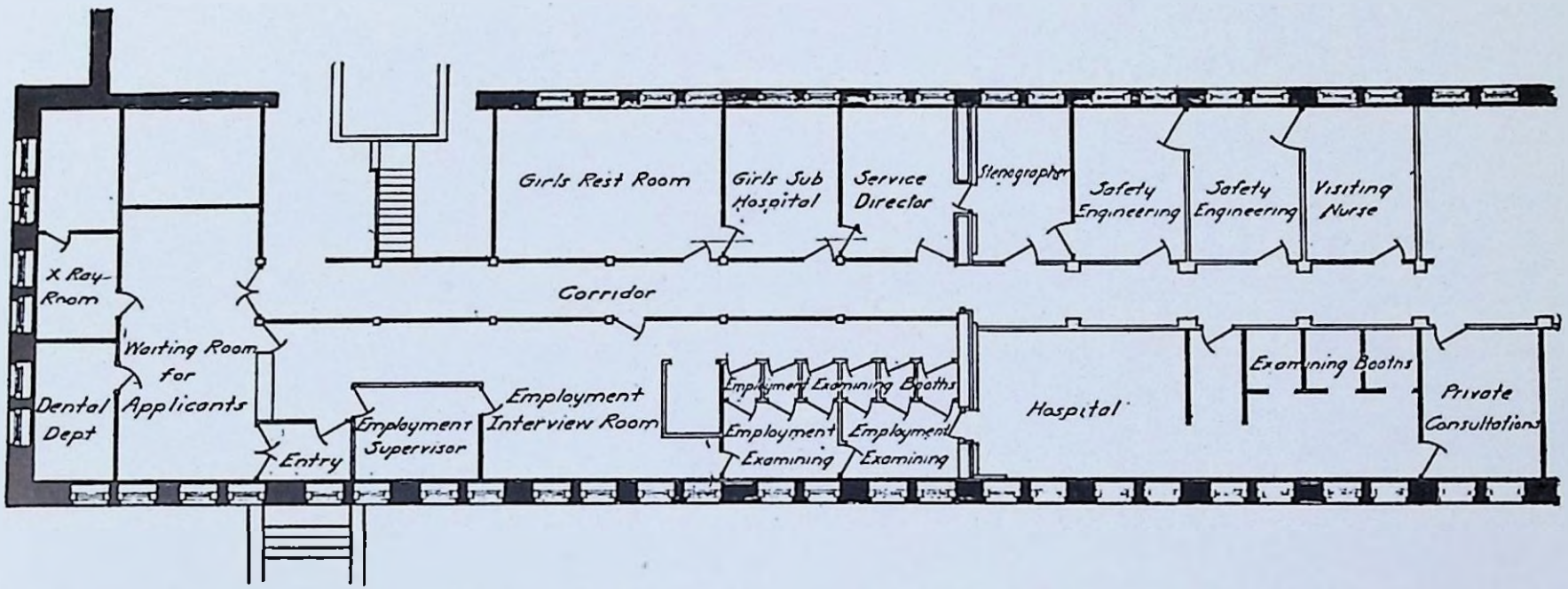


FIG. 2. Layout of central dispensary and contact departments.
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men working in parts of the plant where there is exposure to some toxic product, usually present in the form of a dust or a fume.

The physical examination as made in industry is similar to the examination made for life insurance, for the Army or Navy, or for admission to a hospital. Time, however, is such an important factor that certain routines have been developed which save time for both worker and doctor. Thus the applicant, if a man, is usually examined completely stripped. The physician examines and records from the top of the head to the soles of the feet, his interest being in detecting possible defects which are important from a working point of view. The life insurance examiner is particularly interested in the applicant's basic condition. His problem is to estimate the probable longevity of his patient. The industrial physician on the other hand is particularly interested in the probable working capacity of the individual for the next 10 years, and he, therefore, examines with great care certain abilities and functions which are comparatively unimportant to the life insurance

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examiner. It is particularly important for the general practitioner who is called in by a neighboring factory to make examination of its workers to bear this distinction in mind. The defects of particular importance to the examining physician are as follows:

1. Markedly defective vision uncompensated by glasses, or blindness of one eye.
2. Marked deafness of both ears.
3. Hyperthyroidism.
4. Myocarditis.
5. Blood pressure over 180 systolic or 100 diastolic.
6. Pulmonary tuberculosis; pleurisy with slight effusion.
7. Emphysema and chronic bronchitis—asthma—fibrosis of lung.
8. Gastric ulcer or carcinoma.
9. Cholecystitis with or without gall-stones.
10. Chronic appendicitis.
11. Chronic nephritis with acute exacerbation.
12. Gonorrhea or syphilis, especially in active stage.

13. Chronic arthritis, especially of the spine.
14. Ankylosis of important joints.
15. Severe varicose veins especially with edema of leg.
16. Chronic foot strain—deformities of foot or toes.
17. Hernia of all types.
18. Hypertrophy or infection of prostate.
19. Epilepsy.
20. Diseases of nervous system.
21. Dental conditions suggesting foci of infection.

Having these basic conditions in mind the examination is made following the procedure adopted by the Conference Board of Physicians in Industry.

Physical Examination Procedure
Conference Board of Physicians in Industry
[129]

An outline of the principal points to be kept in mind when making a physical examination of applicants or employees. It is understood, however, that in many cases and for many purposes a partial examination only will be necessary, and that, barring special cases, the examination would consume an average of from 6 to 10 minutes per person. It is

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also understood that the procedure may be added to or subtracted from as the peculiar requirements of each industry may make advisable in the judgment of the examining physician.

Special Senses

(*a*) Eyes:

Visual Acuity: Right Eye:.....

Left Eye:.....

Visual acuity of 20/40 or less in both eyes indicates specified employment. Class III, and further investigation.

Visual acuity of less than 20/30 in one eye and less than 20/200 in the other with suitable correction, specified employment. Class III.

A greater disparity in vision of the two eyes indicates need for further investigation.

Reading Test:

Right Eye:..... Left Eye:.....

.....Size of Type.....

.....Distance .....

Field of Vision:

Right Eye:..... Left Eye:.....

.....Normal or Limited.....

Loss of more than one third of field of vision of both eyes, or loss of two thirds of field of either eye, indicates specified employment. Class III.

Color Sense: Ability to match colors and call them correctly.

Pupils: Reaction.
Puffiness of Lids.

(*b*) Ears:
Drums:
Right Ear:..... Left Ear:.....
.....Appearance .....
.....Hearing Power .....
.....By Watch .....
Use Ingersoll $1 watch at 36″ as normal. Any variation from normal to be indicated by the fraction 36, denominator being distance at which watch can be heard. Deafness to be indicated by the word "contact." Hearing power of less than one half normal indicates specified employment. Class III.

(*c*) Nose:
Septum.
Turbinates.
Obstruction.

(*d*) Mouth and Throat:
Condition of teeth[1].
Condition of gums.
Condition of tonsils[2].
Thyroid enlargement.

[1]Suggested classification for conditions of teeth:
1. Good—no repair needed.
2. Fair—minor defects: treatment advisable.
3. Bad—serious defects needing immediate corrective treatment.

[2]Diseased tonsils in adult should be removed. Such cases should be followed and removal advised. In absence of other disability diseased tonsils should put the person examined in Class II.

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Chest Examination:

(*a*) Heart:

1. Myocardial test.
   Effort Syndrome—Pulse reading before and after exercise.
2. Blood pressure taken on all cases over 40 years of age; others as indicated.
3. Valvular disease—compensated or uncompensated.
4. Arhythmia.
5. Tachycardia.

(*b*) Lungs:

1. Inspection—lagging, apical retractions.
2. Auscultation—Eight points to examine closely as follows:
   Front:
   Apex of lung—right and left.
   Infraclavicular space—right and left.
   Back:
   Suprascapular space—right and left.
   Base of lung—right and left.

Abdomen: Inspection—abdominal tumors.

Hernia:

Inguinal—complete, incomplete, oblique, direct.
Size of external ring.
Impulse, present or absent.
Femoral hernia.
Ventral hernia.
Umbilical hernia.
Post-operative.
Abdominal wall.

Spine:

Curvature.
Mobility.

Anus:

Examined for hemorrhoids, fissures, fistulae, protusions.

Genitals:

Examined for varicocele and hydrocele, condition of prostate.

Extremities:

(*a*) Lower:

Varicose veins.
Joint motility.
Loss of member.
Deep reflexes.
Foot strain.

(*b*) Upper:

Joint motility.
Loss of member.
Loss of digits.
Tremors.

General:

Gait.
Romberg.
Skin.

Height, Weight and Age to be recorded.

All deformities and contagious conditions should be noted in the proper place. Applicants should be rejected on physical grounds only when their employment would make them a menace to themselves, to others, or to property.

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Following questions will give a good background for obtaining important family history information:

1. When did you last consult a physician?
2. Have you ever been in a hospital or sanitarium?
3. Have you ever had heart trouble, lung trouble or tuberculosis?
4. Have you ever had tonsillitis?
5. Have you had fits or fainting spells?
6. Are you well and strong?
7. Have any relatives had tuberculosis?
8. Have you ever had accidents, operations, or injuries?
9. Have you weak lungs or heart trouble?

We have given this rather lengthy description of the physical examination not only for its importance in industrial medicine but because it gives an excellent method of procedure for practitioners who wish some definite outline to follow in making periodic examinations of their patients. While the annual examination of an individual will not invariably detect beginning degenerative changes or early signs of chronic disease, it will in many instances suggest that such is the case and lead to a more thorough examination and physical analysis at a hospital.

Certainly in the course of routine examinations in industry cases of diabetes, albuminuria, early myocardial change and early carcinoma are frequently discovered, of which the patient had no knowledge prior to the examination. In workers exposed to an industrial disease hazard, early detection is of the utmost importance, as in the majority of cases early recognition and removal of the patient from exposure will effect a cure regardless of what palliative measures are applied in the form of treatment.

Diagnosis. The second important function of the Medical Department is its function in diagnosing disease. Patients, particularly of the working group, do not consult a physician unless they are more or less incapacitated from sickness. This is owing partly to neglect, partly to an effort to avoid expense, and partly because of the time lost from work. They will, however, seek assistance and advice at the factory dispensary for very mild disorders many of which are insignificant, but some of which are the prodromata of severe or dangerous conditions. It is in the latter group that the industrial

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physician has an unusual opportunity to make an early diagnosis.

When such early diagnosis is made it is the industrial physician's custom not only to refer the patient to his family physician but to see that he consults him. With the patient should go a letter giving the Medical Department's findings. In this way it is possible for the factory Medical Department to coöperate with the workers' physicians and every effort should be made to develop cordial relations. The industrial physician should not treat any medical case which needs home treatment by a physician. The type of conditions which he should treat are those which, though too minor to need a physician's thought and care, are of enough inconvenience to the worker to require some attention. Such conditions as coryza, indigestion, mild conjunctivitis, myalgia and constipation form the bulk of the medical conditions treated at the factory dispensary.

The practitioner can readily see that he has a valuable ally in the industrial physician of a factory in which some of his patients work.

If he will take the trouble to develop coöperative relations, the factory physician and nurse will not only see that his patients consult him when necessary but will also be glad to do minor laboratory work upon his patients at his request, checking up blood, urine, or blood pressure at regular intervals. Even the routine weighing of patients, changing their work at their physician's request, or other small matters may be of great assistance to the busy doctor in the management of his chronic cases.

Unfortunately in the case of many workers their financial situation is such as to prevent the employment of a private physician. Such cases must be treated at a hospital or public dispensary. On the other hand there are frequently workers who have ample means but for some reason or other have never had a family doctor. It is the custom of the industrial physician to provide such men with a list of doctors practicing in the neighborhood where they live, from which the worker may make his choice.

Working environment. In the case of suspected industrial disease it is very important for

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the practitioner to be familiar with his patient's working conditions. To most non-industrial physicians the factory workrooms are a closed book and the history of working conditions given by the patient may be very misleading. Thus it is advisable before coming to a conclusion for the physician personally to inspect the working place and conditions described and draw his conclusions from what he sees rather than from what he hears. The industrial physician of the plant will gladly show him about, explain the work, and if necessary obtain any technical information desired from a foreman or other official of the company. It is well to remember, however, that in some plants there are secret processes to which no one outside the plant is admitted, and in cases of this type special permission to view must be obtained from the management.

Accident treatment. The third function of the Industrial Medical Department is the treatment of accidents. Here largely because of the Workman's Compensation Acts the full responsibility for the care of an injured worker rests with the

industrial physician. He must be prepared to give primary treatment in any type of accident and in cases of severe accident to see that the patient is properly transferred to the nearest hospital with a minimum of shock. In minor injuries he carries on treatment until the worker is able to return to work cured.

The treatment of accidents in a factory is given at the place the accident occurred or at the factory dispensary, depending upon the severity of the injury. Most injuries can be cared for at the dispensary unless unusually severe in which case it is necessary for the doctor to go to the man rather than the reverse. In factories which spread over a large area or in mines there is often a series of First Aid rooms, each well equipped and having a nurse or trained layman in charge. In other cases there are First Aid stations each consisting of a cabinet containing those things necessary for the immediate treatment of accidents. While laymen trained in first-aid work are necessary in mines and on railroads, they should not be relied upon when any other solution is possible. Every injury, no matter how

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trivial, should be treated by a physician or by a graduate registered nurse and no retreatment of any consequence should be done except under the supervision of the attending physician.

Rehabilitation. In every case of industrial accident the object of both physician and workman is an early return to the occupation at which the worker was employed at the time the accident occurred. This is of the utmost importance to the worker from an economic point of view. Although he will receive financial compensation under the Workman's Compensation Act for the time he is absent from work, the compensation is usually only two-thirds of the amount he is able to earn. Moreover, the worker usually prefers work to leisure, when his activities are restricted and he must remain at home. Frequently, though it is impossible for an injured worker to resume his original work, there comes a time when he can work somewhere in the factory. If this can be done to his financial advantage he should be returned to work, remaining of course under the supervision of his physician.

An early return to work is frequently a therapeutic measure. The worker ceases to think about his injury, actively uses the injured part, and is often able to return to his original occupation in a surprisingly short time. It is a well-known fact among industrial physicians that the longer a man is out of work the harder it is for him to resume and the slower he is in regaining his earning capacity. Moreover workers who are injured at work receive a mental shock as well as a physical injury and restoration to regular work restores confidence.

In order to restore earning capacity at the earliest moment the industrial physician must use the Employment Department. Inasmuch as this department is used in placement of applicants found defective by physical examination, of workers who have been somewhat incapacitated because of sickness or recent operations, and of workers recovering from industrial accident, its theory and functions will be briefly outlined.

When an additional workman is needed in any department of an industry the foreman of that

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department fills out a form called a requisition on which is stated the type of work for which the new worker is needed. This is sent to the Employment Department. The Employment Manager selects from a list of applicants, or from men then in the employment office one whose general appearance and whose past experience seems to fit him for the vacant position. The applicant is then given a physical examination and if approved is taken to the foreman who filed the requisition and is put to work. If the work he is to do is hazardous either the Safety Engineer or the foreman points out any danger present and the safeguards provided.

In making the initial hiring examination the examining physician has to decide whether the physical condition of the applicant permits his working at the job to which he has been assigned by the Employment Department, whether his physical condition is such that some other job is preferable, or whether or not he should work at any job.

The great majority of workers are physically

fit to do the job to which they are assigned. A small minority must be placed at selected jobs, only a very few must be rejected. The basis for rejection is that employment of the individual would constitute a danger to himself, to others, or to the company.

It is obvious that a man having a severe disability should not be permitted to start at work which can only be temporary and which will lead to complete incapacity. Men with contagious disease are not proper employees. Certain defects which of themselves are not incapacitating may be a danger to the company, because they render the worker particularly susceptible to prolonged disability or complete incapacity following a minor injury or because the condition, as in epilepsy, may lead to severe injury.

The Workman's Compensation Acts with the free interpretation by Industrial Accident Boards of the word "injury" have enormously increased the liability and insurance rates of industries in a large number of states. The ten-

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dency to consider many common diseases as "injuries" or to consider minor accidents as accelerating factors to existing disease has made it necessary for manufacturers and other industrialists to take every possible precaution that their workers are not harboring conditions which will later entail large payments of compensation for what, in a normal man, would be a trivial injury.

Thus a worker who has vision in only one eye is a hazard because, although he may do his work to satisfaction, an injury to the remaining eye will incapacitate him for a long period and may lead to total blindness.

A worker having a hernia may, following a slight strain, complain of pain and inability to work which means an operation and 6 weeks' disability. A worker having inactive pulmonary tuberculosis, doing heavy, hot or dusty work may, if he develops an activity of the tubercular focus, receive compensation for months or years.

A worker having arthritis of the spine may, following a slight back strain, develop permanent incapacity with consequent payments.

A chronic cardiac may die suddenly at work and become a case of death from industrial accident.

With the high rates for compulsory insurance, the keenness of competition, and the variance of Workmen's Compensation Acts and their administration in neighboring states, it has been necessary for industries in certain states to protect themselves in every way possible against compensation costs. If there had been no changes in the interpretation or of the acts as originally drawn, many workers could be placed in industry who are now considered too great a hazard. Fortunately, the number of qualified workers who are not acceptable for physical reasons is small for the individual factory or mine, though large for the United States as a whole.

In some states the Industrial Accident Board permits the employer to obtain a waiver from the worker freeing the employer from responsibility for injury directly resulting from the defect. Unfortunately, the intricacy of the necessary proceedings has prevented their use in many cases.

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When an employee is laid off by his foreman because of lack of work necessitating a reduction of the department working force, the worker is sent to the Employment Department for a transfer if work is available in another department. If such a transfer is considered, a physical examination is made to be sure that there is no defect which would make work of the new type inadvisable. When men have been absent from work for some time because of sickness or injury the physician and the Employment Manager endeavor to arrange transfers in such a way that the workman may return to a kind of work for which he is physically fitted.

Safety Engineering and the Industrial Physician

Another department in close contact with the Medical Department is Safety Engineering, whose object is to prevent accidents. The Safety Engineer's interest lies in seeing that all machines requiring it are properly guarded, that workers are instructed as to the hazards of the machines or processes at which they are working, and that every accident of any severity is investigated to determine its cause and to study its possible prevention. His department also provides goggles, respirators and other forms of safety appliances to the workers and keeps records of the number and types of accident occurring in the plant. It will be seen that the Medical Department must maintain the closest contact with each of these departments and that the industrial physician must coöperate closely with their managers.

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CHAPTER II

INDUSTRIAL SURGERY

IN THE PREVIOUS chapter the work of an Industrial Medical Department has been outlined. The doctor who devotes part or all of his time will now be discussed. Such a discussion should be of interest to the practitioner who works in an industrial community and particularly to one who may give part of his time to industrial work.

As suggested by the foregoing outline a physician practicing industrial medicine must be considered from a point of view somewhat different from that of the general practitioner. He must have in addition to diagnostic ability and a good grounding in traumatic surgery, the ability to consider the welfare of groups exposed to

an industrial hazard, an interest in and a knowledge of good sanitary procedure, a public health interest in the groups of employees under his supervision, and a kindly, thoughtful personality. In many matters his interest must involve groups of people but in addition to this he will always have a large number of individual cases to examine, advise, and treat and these cases are considered and advised with the same care and thoroughness as are the patients of a physician in private practice.

"The physician in industry is one who applies the principles of modern medicine and surgery to the industrial worker, sick or well, supplementing the remedial agencies of medicine by the sound application of hygiene, sanitation, and accident prevention; and who, in addition, has an adequate and coöperative appreciation of the social, economic and administrative problems and responsibilities of industry and its relations to society." [127.]

The time element is important and the industrial physician's work may be greatly facilitated by the assistance of one or more good

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nurses and by the proper physical layout of his examining room and dispensary. To the practitioner who is trying to save his own time the idea of saving the patient's time is a novel one; however, he must adjust himself to this new viewpoint and realize that one of the important objects of the Industrial Medical Department is to conserve the worker's time. Thus he must not consume too much time in making a physical examination, the dispensary must be centrally located in order that the workman who visits it may lose the least time possible, waiting time for attention at the dispensary must be cut to a minimum, and even the time taken in writing records is of importance if it prevents prompt service. Industrial physicians have developed methods by which a great deal of time can be saved and these methods may be of interest to the general practitioner whether or not he is dealing with industrial cases.

The layout of the dispensary is of importance. It has been found that it is a great saving of time if each type of accident has its special place for treatment. One or more tables equipped with

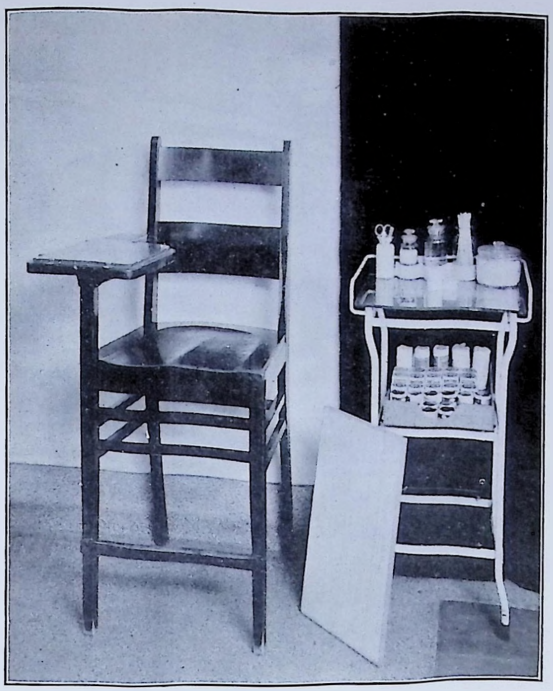
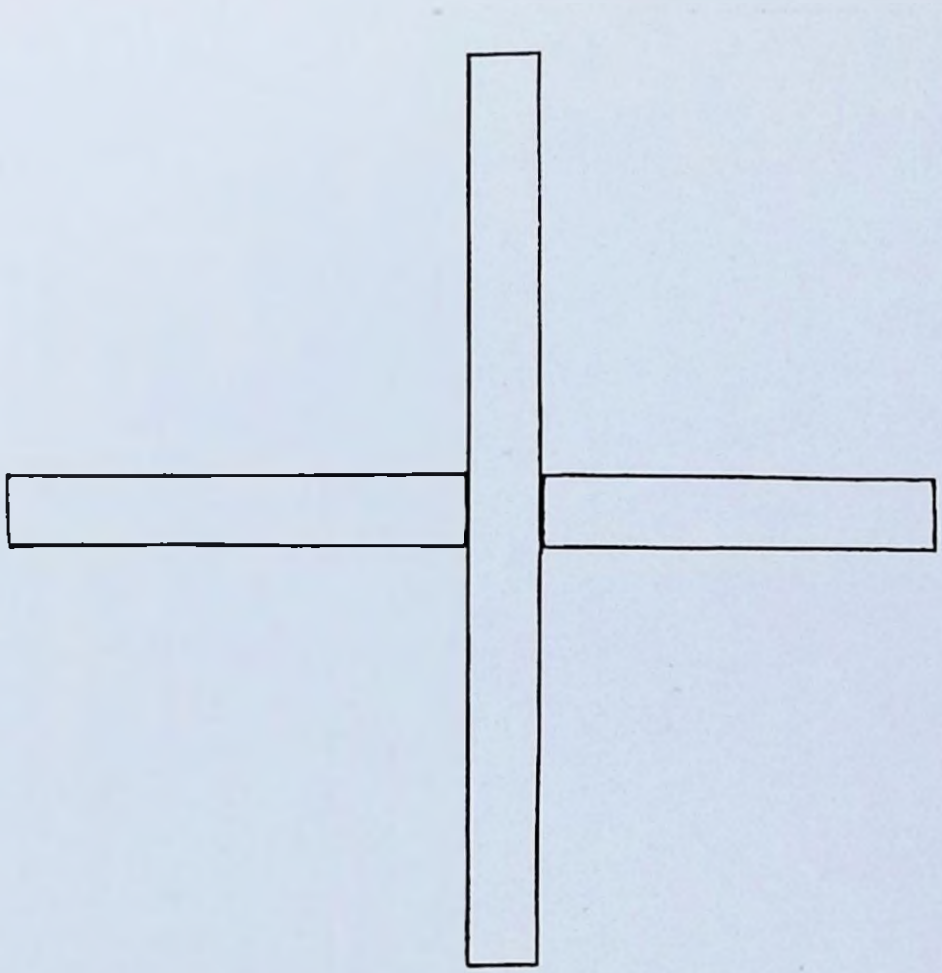


FIG. 4. Unit for hand treatment.
Layout consists of

1. High chair with hand rest.
2. Dressing table equipped (left to right) with; instruments in aseptic solution, Tr. Iodine, gasoline, sterile cotton on applicators, sterile gauze pads. Lower shelf contains bandages.
3. Board (leaning against table) covered with narrow strips of adhesive plaster for reinforcement of dressings, etc.

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FIGS. 5 and 5a. Tin cross-piece finger splint.

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everything necessary for treatment of a hand or finger injury is particularly convenient and the same applies to foot and leg accidents. (Fig. 4.) If possible there should be a dark room where eye injuries can be treated and where examinations of the ear, nose, and throat can be made. An x-ray (bed-side unit) is invaluable for fracture diagnosis since an x-ray of all crushing or twisting injuries is standard practice in modern industrial dispensaries.

Industrial Surgical Service

Wounds. The treatment of wounds at present consists of a thorough cleaning of the skin around the wound with lead free gasoline. This may also be used to clean out the wound itself though for the first skin cleaning a piece of gauze covers the wound. After gasoline either tincture of iodine or one of the mercury preparations is used. Next all foreign matter is gently wiped from the wound and if there is dead tissue it is removed by sharp dissection.

In industrial injuries the wound is usually lacerated and contaminated with grease, dirt,

or other foreign matter. It is, therefore, considered best not to suture but after checking hemorrhage carefully, to dress dry, with loose gauze, to apply a bandage rather loose than snug, and to splint and sling the injured part. The complete rest given by the splint and sling aids healing and prevents the patient using the injured part. One of the rules of industrial surgery is complete rest until healing is well under way, followed by early active motion. During subsequent dressings exposure to the light and heat of an electric bulb with a reflector seems to promote healing and is now commonly used for from 5 to 10 minutes before the fresh gauze is applied. In dressing wounds the no-touch technic should be used. All dressings are removed with one set of forceps, these are discarded and a sterile pair or two of forceps or artery clamps employed to hold the gauze used for cleaning the wound and in applying a fresh dressing. For cleaning small wounds wooden applicators, cotton-tipped and sterilized, are admirable. In wounds where there has been loss of tissue it is advisable to do a loose secondary suture or to pull the edges together with sterile adhesive plaster after one is sure that no infection is present. Adhesive may be sterilized by passing the strip once or twice through a Bunsen flame.

Finger splints. A very convenient method of protecting and splinting injured fingers, especially finger tips, is the use of a cross-piece of light tin. (Figs. 5, 5a.) This consists of two strips of tin about ½ inch wide soldered together at right angles to each other. After the bandage is applied the four ends are bent over the finger tip to form a splint and cage not quite touching the injured portion of the finger. The cross-piece is held in place by a strip of adhesive and another bandage applied over it. With this protection workmen with badly crushed finger tips are able to return to work at once, to their great satisfaction.

Fracture of the middle or proximal phalanx of a finger with displacement of the fragments is a difficult fracture in which to obtain a good functional result. Unless alignment is excellent some, and frequently a major part of function, is lost. The finger is one of a workman's most im-

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portant tools and restriction of its function reduces the speed and accuracy of the work of the whole hand. A stiff finger or one which has lost half its function is often better amputated than left, as it interferes with work.

The fractured finger should be x-rayed, set, and splinted with the utmost care. At the American Rolling Mill Co. such fractures are reduced under the fluoroscope, the best possible alignment obtained and a plaster cast applied. The finger is held in position while the plaster hardens. The results have been excellent. The corresponding metacarpal bone and the wrist should be immobilized as well as the finger. In some cases, especially where more than one finger is involved, the best method is by traction and a banjo splint. (Fig. 6.) A cuff of saddler's felt is sewed around the wrist. Over this a plaster bandage is applied in which is imbedded the ends of a piece of telephone wire bent in a ¾ circle. The injured fingers are extended by adhesive plaster traction bands attached to rubber bands which in turn are taped to the wire. The amount of traction is regulated by the pull of

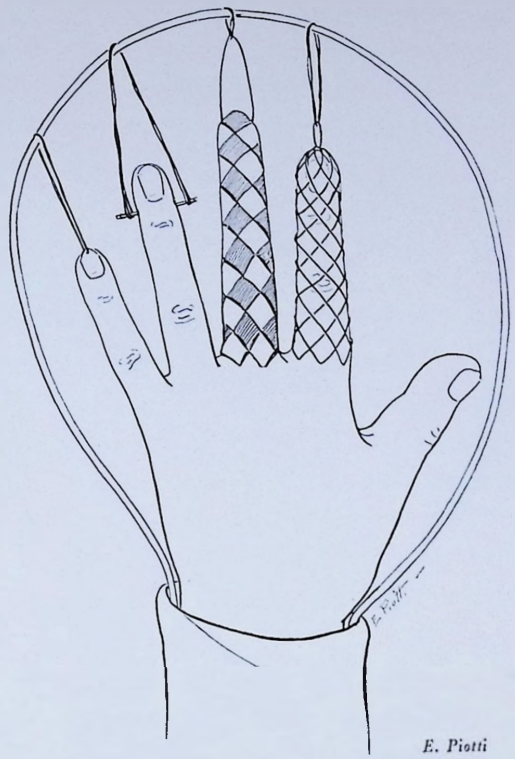


FIG. 6. Devices used for finger splint.

1. *Little finger*—Silk suture through nail. 2. *Ring finger*—Skeletal traction (large needle through terminal phalanx). 3. *Middle finger*—Wire mesh fingercot (well padded). 4. *Index finger*—"Japanese hand-cuff."

Note: Any of these is secured with heavy rubber bands to a banjo splint. The tension of the elastic band traction is increased by twisting them. A matchstick passed through the band does this satisfactorily.

The banjo splint (or heavy telegraph wire) may be solidly secured by imbedding it in a plaster of Paris cuff about forearm.

Any of the above devices is far superior to adhesive strap traction on the finger. (*Courtesy of Reconstruction Hospital* [140].)

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the bands and controlled by the x-ray or the fluoroscope. This type of splint is particularly valuable when there has been accompanying laceration of the fingers or if the fractures are compound.

In splinting fractured fingers it is well to remember that after setting, the finger should be immobilized in a slightly flexed rather than a perfectly straight position. The tongue depressor so frequently used does not make a good finger splint; for a small splint a heavy hairpin slightly bent to the natural curve of the finger, and lightly wound with adhesive plaster or a light piece of block tin well padded, is far better.

Fractures. Fracture of the scaphoid bone of the wrist is not uncommon in industry. It is a fracture easily missed in the x-ray and sometimes does not show on the first picture. Any injured wrist which continues to have localized pain, to show disability and pain on pushing, which has limitation of dorsal flexion and marked weakness must be viewed with suspicion and several x-rays should be taken.

In cases of this injury the treatment is a plas-

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ter cast applied with the wrist in the cock-up position, from the metacarpo-phalangeal joint to the elbow. Long immobilization is important and should continue for at least 6 weeks. After the cast is removed a straight splint is applied for a few days. Where there is wide separation of the fragments operation will be necessary [19].

The treatment of fractures involving other bones of the upper extremity is commonly known and is thoroughly discussed in text books.

Nerve injury. Injury to nerves of the upper extremity occasionally occur as the result of accident. The following comments issued by The Reconstruction Hospital [140], Columbia University, New York, may prove helpful in diagnosis and in prognosis:

> In studying nerve injuries, a consideration of the history is most important. Stab wounds generally cause severance of the nerve, while blunt objects produce contusions with physiological interruption.
>
> In fracture, nerve injuries are caused: early—by sharp fragments; late—by callous and scar formations, and also improper splintage.
>
> Careful preliminary and frequent neurological examinations are required to determine whether nerve paralysis is early or late in onset.

An accurate and quick method of determining nerve injuries in the upper extremities is as follows:

An injury to the *radial nerve in any part* of its course produces loss of extension of the thumb. Confirmatory signs:

a. Inability to extend the wrist.

b. Inability to extend the fingers at the metacarpo-phalangeal joints.

c. Anaesthesia over the dorsal base of thumb.

An injury to the *median nerve in any part* of its course will result in loss of function of the opponens pollicis (inability to rotate the thumb over the palm). Confirmatory signs:

a. Inability to flex the index finger at the metacarpo-phalangeal joint when the other fingers are held in extension.

b. Anaesthesia over the palmar surface of the thumb, index, middle and radial half of the ring finger.

Injury to the ulnar nerve:

a. Loss of individual movements of the fingers.

b. Anaesthesia over the palmar surface of the little finger and ulnar half of the ring finger.

In other words, if an individual can make a cone with his fingers and his thumb—there is no injury to the median nor ulnar nerves, and if he can extend the thumb—there is no injury to the radial nerve.

The only reliable signs of nerve regeneration are:

1. Sensory return.
2. Return of muscular function.
3. Disappearance of reaction of degeneration.

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In all nerve injuries the paralyzed muscles should be supported in the relaxed position.

Uncomplicated nerve lacerations should be sutured at once. Asepsis is paramount as infection is the most common cause of the failure of nerve repair.

Nerve suturing should be end to end without rotation or tension, using interrupted sutures of black silk or plain catgut No. 000 confined to the sheath. Absolute hemastasis is essential.

When nerve is included in callus, it should be freed and transplanted to a muscle bed. Example: In a fracture of the internal condyle of the humerus with inclusion of nerve in callus, the nerve is freed and transplanted to the anterior surface of the elbow.

Time Necessary for the Regeneration of Nerves:

Musculocutaneous	5 to 10 mos.
Musculospiral	5 to 14 mos.
Ulnar at wrist	5 to 10 mos.
Ulnar at elbow	10 to 16 mos.
Ulnar at axilla	16 to 22 mos.
Median at wrist	4 to 10 mos.
Median at elbow	8 to 15 mos.
Median at axilla	8 to 15 mos.
Sciatic, upper thigh	12 to 22 mos.
Sciatic, middle thigh	12 to 18 mos.
Peroneal	10 to 16 mos.
Tibial	11 to 17 mos.

A workman sustaining a fracture of the lower extremity requires immediate attention. The patient is not moved until the physician has applied a Thomas splint and treated shock. The patient is then transported to the nearest hospital or to his home.[1]

Back strain. There are a number of other injuries which occur in industry fairly frequently which are orthopedic in character. Of these the strained back is the most frequent and the most important. Incapacitating strain of the low back may be divided into slight strains which produce serious reactions because of underlying disease and severe strains which injure normal structures. Although the treatment of both types is similar, in the former the period of disability is greatly prolonged. Strain of the low back in industry usually occurs in the region of the lumbo-sacral joint, less frequently in one of the sacro-iliac joints. The patient limps or is carried to the dispensary where examination elicits

[1]An excellent résumé of the modern methods of fracture treatment appears in *Bulletin of the Am. College of Surgeons,* Vol. 15, No. 1, 1931.

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rigidity of the spinal muscles, poker back, inability to flex the trunk forward except from the hips, pain in the lumbo-sacral region or over one sacro-iliac joint. If the patient lies on a table and the physician raises a leg without bending the knee there is resistance and intense pain before the heel is raised more than a foot or two from the table. In cases of sacro-iliac strain, if the leg is flexed on the thigh and then the thigh on the trunk and pressure is made downwards pain is elicited in the sacro-iliac joint of the side affected. An x-ray taken laterally as well as one taken anteroposteriorly is imperative in order to determine whether or not arthritis is present, and if so, how severe. In mild cases tight strapping with adhesive gives marked relief (Fig. 7). The strapping is applied with straps 3½ inches wide and 2 feet long. The patient stands with his back to the doctor who applies the straps from below up, each strap overlapping the one below. The lowest strap is over the middle of the buttocks and includes the trochanters on each side. The strapping is carried up to the line of the ribs. Occasionally a double layer is applied. The

after-treatment consists of rest and heat. Clark has found that in cases having home treatment, repeated ironing with an electric or ordinary flat iron is very helpful. A blanket should be placed over the back and strapping before the ironing is done and the iron should be tested to see that it is not too hot before being used. Electric irons should be disconnected before using.

Severe back strains always require hospital or home care. Traction on both legs of 12 to 15 pounds by some extension apparatus is very helpful, followed by a plaster cast from hips to axilla or by a back brace. All cases of strained back are instructed to sleep on a hard, firm bed made level by boards placed between the frame and the mattress as in cases of fracture of the leg or thigh.

Sprains. An uncommon but very perplexing industrial strain or trauma is periostitis of the external epicondyle. The epicondylitis is marked by tenderness in the region of the epicondyle, by a piercing, burning pain in the same region where the arm is extended beyond 160° at the elbow joint, and by radiation of pain up and down the

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arm. Opening and closing the hand also causes pain along the extensor muscles of the forearm. There is no external evidence of injury. There is complete incapacity for regular work. The treatment consists in applying a plaster cast from the axilla to the finger tips, the elbow being flexed at a right angle. The wrist and hand should be extended. The cast is left on for 3 weeks after which baking and massage are given.

Sprained ankle is such a common injury in all walks of life that its treatment is common knowledge. However, it has recently been shown that in many cases of sprained ankle it is the anterior inferior tibiofibular ligament which is torn or stretched, that the point of greatest tenderness is usually anterior, median to the external malleolus, over this ligament rather than lateral, and that the adhesive strapping must encircle the malleoli of the tibia and fibula. The standard Gibney method of strapping fulfills this requirement as well as supporting the lateral ligaments.

Sprain of the knee joint always suggests possible injury to one of the semilunar cartilages.

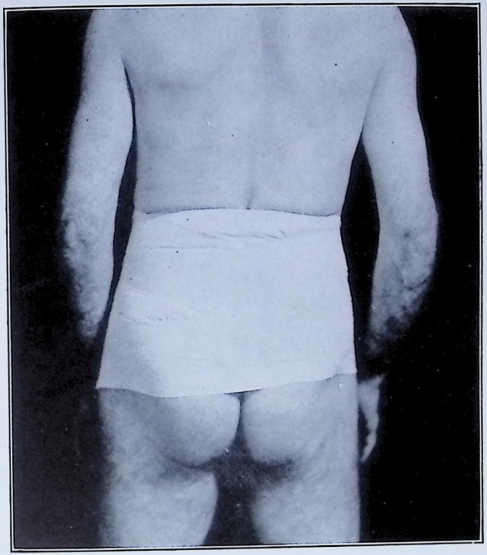


FIG. 7. Strapping for low back strain.

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If no injury to the cartilage is found on x-ray and on careful examination, the best treatment is a circular plaster cast extending well up the thigh, applied at once. This is bivalved later and heat treatment given. Massage is not advisable. Motion is not started until the joint is fluid free. When the cartilage is injured operation is indicated.

A common industrial injury is fracture of one or more toes. Frequently these patients are able to use the foot for part of the day if it is covered by a heavy woolen sock and if an army type shoe with a thick sole is worn.

Burns. Burns occur with frequency in industry. There are two methods of treatment in use; the tannic acid treatment and the gentian violet treatment.

The tannic acid treatment [60] consists in:

1. Immediate removal of burned worker to the dispensary.
2. Removal of clothing from all burned areas.
3. If burned area is greasy remove oil or grease by light sponging with ether.

4. Spray 5 per cent aqueous solution of tannic acid on all burned areas.
5. Cover burned parts with sterile sheeting and transfer patient at once to nearest hospital.
6. For burns of the face 5 per cent tannic acid jelly, (tragacanth base); no spray because of eyes.
7. In milder burns where ambulatory treatment is possible either the 5 per cent tannic acid jelly or a moist dressing of 5 per cent tannic acid may be applied and changed frequently. At each dressing the burn is exposed to dry heat by an electric baker or lamp.

The gentian violet treatment of burns consists in following the same steps as in the tannic acid treatment except that 1 per cent aqueous solution of gentian violet is used. Where jelly is indicated, 1 per cent gentian violet in a tragacanth base is satisfactory. The jelly is left in place for about 36 hours after which it is removed and a sealing of the skin surface will be noted. This

treatment is in favor at the present time [1]. [30].

Rehabilitation. In severe injuries requiring hospitalization or home care the Medical Department maintains a contact with the hospital or private physician through the offices of the visiting nurse. The nurse in such cases sees both the patient and his attending surgeon and brings back to the Medical Department reports of his condition and progress. These are entered on the patient's dispensary record. As soon as the patient is able to leave the hospital or his home, the Industrial Physician and the Employment Manager endeavor to find a place for him to work as soon as he is able to resume any work at all.

Industrial accident cases are checked frequently after their return to work and only discharged from careful supervision when they have resumed their original work or work which is final in character. At this time a complete examination is made to determine the end result obtained and this end result is recorded [96].

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In industrial accident records the history must include not only the general type of work the patient was doing at the time of accident but also the exact operation at which he was engaged. In industrial medical departments the questions asked are "What were you doing at the time of the accident?" "What happened?" The examination must be complete and conclusive. This is followed by diagnosis and treatment. Each subsequent visit to the dispensary is recorded, the patient's condition noted carefully and the treatment outlined. When the patient is able to return to selected work the fact and type of work is entered, and at the time of discharge to regular work a complete examination with measurements and end result is recorded. A simple method of estimating the end result of fractures utilizes the percentage method and emphasizes the value of function over surgical result or appearance. This method is used by Dr. John J. Moorhead on the traumatic service of the Post Graduate and the Reconstruction Hospitals in New York; it is approved by the Conference Board of Physicians in Industry.

The estimation is divided into three elements as follows:

Function, 60 per cent Union, 20 per cent Contour, 20 per cent	Equals 100 per cent for perfect result.

Function denotes capacity to perform.

Union denotes the state of repair.

Contour denotes the external appearance.

Following recovery these elements are estimated on the above basis and the sum represents the percentage of usefulness remaining. The percentage of disability can be determined easily from this.

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CHAPTER III

INDUSTRIAL MEDICAL SERVICE

JUST AS PART of the dispensary is arranged for the systematic treatment of injuries so a section is set apart and equipped for the examination and advice of medical cases. The requirements are those of the office of a general practitioner and include a laboratory for urinalysis, blood work, and microscopic examinations.

Every type of disease encountered by the general practitioner may appear at the industrial dispensary but in addition certain industrial diseases peculiar to the specific industry must be diagnosed. As indicated in a previous chapter the majority of workers applying at an industrial dispensary have mild disorders but it must be remembered that in the mass many of these dis-

orders are of great importance in industry. Thus an epidemic of common cold sweeping through a department or a group of workers may completely demoralize production, while epidemic influenza has a serious effect upon the whole organization. Because of its contagious character infectious respiratory disease of any type is studied with interest and efforts are made to check its spread. While these efforts have been frequently disappointing, prompt attention, advice, treatment, and, in certain types, segregation have had considerable effect. The attack upon this type of disease is similar to the public health worker's attack upon an epidemic of contagious disease except that in the case of the industrial physician the control is better. Thus it is easy to isolate an infected individual from the industrial community by simply sending him home or in severe cases to a hospital, and preventing his return to work until he is no longer in danger of infecting others.

The more important functions of the Industrial Medical Service are to make an early diagnosis in an effort to avert beginning disease,

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to initiate early operative treatment in surgical conditions, and to discover signs of early degenerative changes in the organs of the older group of workers.

While it is impossible to show the effect of early diagnosis and prompt treatment by statistics, industrial physicians feel that many early cases of sickness can be and are minimized. This is owing not only to early diagnosis but to the complete control possible. Thus local treatment when indicated can be given as many times a day as is necessary, and given by a graduate nurse or by the doctor himself, with excellent equipment available. It has been noted that treatment carried out at the dispensary appears to be more effective than when carried out at home without medical supervision.

If a patient has a temperature exceeding 99.6° he is sent home and advised to see his family physician. The chronic diseases give an excellent opportunity for medical control. In industry the patient is always available for the physician's supervision. He can come to the dispensary for examination or advice any day the physician

wishes to see him, without serious loss of time. Examinations of patients suffering from cardiovascular disease, diabetes, anemia, or nephritis may be made as frequently as desired and changes in the patient's condition noted shortly after their first appearance. The more intelligent patients will come to the dispensary at regular intervals as requested, but with the less intelligent group the physician uses a tickler system which notifies him when the patient should be next examined. As was pointed out the industrial physician should coöperate with the patient's doctor, if the patient has one, in the management of these chronic cases.

The study and control of any health hazard in the plant is one of the most important duties of the industrial physician. He must be familiar with all processes in which any toxic or harmful substance is used, must know every possible contact between the worker and this substance, and must know the early symptoms and signs of its injurious effects. His coöperation with the Industrial Engineering force has already been described.

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To sum up, the Medical Service in industry provides diagnosis, advice, and in mild disorders treatment. It endeavors to prevent the progress of disease in individual workers and in groups of workers. It studies and endeavors to prevent industrial disease. It follows the course of disease of those who are at home or in hospitals and determines their ability to work on their return. With the assistance of other departments, it places workers at occupations for which they are physically fitted, and finally it is responsible for the health of the workers in the plant or mine.

Sickness records. In order to carry on its functions successfully the Medical Department must keep accurate records of the accidents and sicknesses of the workers. An approved form of physical examination has been shown. A form of this type may be printed on the outside of a heavy envelope as a record of the initial examination or the form may be inclosed in folder bearing the worker's name, number and department. The method by which records are kept is not uniform but individual records are always kept and

built up in the container, each case of sickness or injury and every physical examination being carefully recorded. In addition the envelope or folder contains all letters from physicians and copies of hospital records or other data relative to the worker's health. Inasmuch as there is no standard record system used in industrial medical departments we will describe the system which has been in use by one of us in his industrial medical department for over 20 years, and which has been found to be efficient and economical [24].

The unit of the system consists of a single manila envelope 5¼ by 8¼ inches on the face of which are printed the physical examination headings. The back contains space for recording transfers and date of discharge. Within the envelope, which opens end up, are placed the cards bearing the sickness and accident record of the worker, any letters from outside physicians, specialists' reports or other medical information. Thus each envelope has on its face the physical examination of the worker on entrance to the factory with his complete subsequent record within (Table 1).

The method by which the sickness and accident record is built up is as follows. In each sub-dispensary as well as in the central dispensary, there are

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two forms, one for sickness and one for accident (Tables 2, 3). Each form is so arranged that after the history is taken, the doctor or nurse can, by a series of checks, designate the injury, its site, diagnosis and treatment, or in case of sickness the history, physical examination, diagnosis, and treatment. These forms are in a block and torn off as needed. Every dressing is thus recorded and initialed by the doctor or nurse treating the case. When more detail is needed in special cases the back of the slip is used for full longhand notes.

Each nurse brings to the central dispensary at noon and at night, the forms she has filled out. They are immediately arranged in alphabetical order by the clerk. She then goes to the central file and takes from the physical examination envelope the last record card on each case. Upon these she transcribes the record on the form slips. These cards are then filed in a drawer near her desk as "live" cases. In case of redressing or retreatment the card is simply taken from this live file. When a case is discharged the card is transferred from the live file to the main file being replaced in the physical examination envelope.

The main file is cleared weekly from a list of discharges sent to the Health Department by the Employment Department. Monthly reports can be readily made from the cards in the live file by not refiling until the monthly report statistics have been taken off.

Forms for special examinations as heart, lungs,

HEALTH DEPARTMENT — Defect

Name — Age

Address — Date

Dept. / Number

General Appearance — Skin

Height — Weight

Eyes: Vision Dist 10 Ft. {R. L.

Ears: Hears Watch {R. L. — Inches / Inches

Nose

Throat

Tongue

Teeth

Neck

Chest Contour

Heart

Pulse — Arteriosclerosis

Blood Pressure

Lungs

Abdomen

Extrem. {Upper / Lower

Ing. Reg.

G. U.

Spine

Joints

Reflexes

TABLE 1. Face of envelope used for sickness and accident records.

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ACCIDENT SLIP

_____ Hosp.

HEALTH DEPARTMENT

No.

Name Age. M. S. W. Dept.

Address

	Date	Time
Accident		
Treatment		

Exact place of accident .

What patient was doing

What happened

Injury	Site	Treatment	Condition	Return
Lac. wnd.	Rt. Lt.	C. & I. M.	Clean	Today
Abrasion	Eye. O'Clock	D. D.	Improved	Tomorrow
Contusion	Hand	W. D.	No Change	D. after T.M.
Puncture	Wrist	F.B.R. Coc.	Lost Time	See Doctor
Incised	Forearm	Hot Comp.		R. to work
Sprain	Elbow	Silvol		Discharged
Strain	Shoulder	Sp. Eye Drops		Main Hosp.
Burn	Back	Splint		Plant 6 Hosp.
Foreign Body	Foot	Strapped		
Fracture	Ankle	Xray		
F. B. Conj.	Knee	T. Lamp.		
	1, 2, 3, 4, 5,	Massage		
	Finger			
	Toe			

TABLE 2. Accident record.

SICKNESS SLIP

HEALTH DEPARTMENT

TIME TREATED

NAME NO. DEPT.

ADDRESS AGE M. S. W. DATE

DURATION D. W. M.	PHY. EXAM.	DIAGNOSIS	TREAT
RESP.	RESP.	RESP.	AC. COMP.
RUNNING NOSE	CORYZA	BRONCH. AC.	ASPIRIN GRS.
COUGH	SPTS. ON TONS	" CHR.	BISMUTH SUBNITR
CHILLS	GRANULAR PHAR.	" SUBAC.	BRONCHITIS
EXPECTORATION	CREPITANT R.	CORYZA	BROWN MIXTURE
SORE THROAT	SUB. CREP. R.	DRY PLEURISY	C. C. & C. R.
RIGHT CHEST	NEGATIVE	INFLUENZA	CAPSOLIN OR LINIMENT
PAIN		LARYNGITIS	CASTOR OIL
LEFT CHEST		PHARYNGITIS	CHLOROFORM & CLOVES
HOARSENESS		TONSILITIS	DOVERS POWDER GRS.
NIGHT SWEATS			LOCAL ANODYNE
ABD.	ABD.	ABD.	MASSAGE
NAUSEA	DISTENDED	GASTRITIS	PAINT I. & G.
DIZZINESS	TENDER AP	ENTERITIS	R. & I.
VOMITING	MASS IN CAECUM	GASTRO ENT.	SALINE GARG.
DIARRHOEA	RASH	CONSTIPATION	SEIDLITZ PULV.
CONSTIP.		APPENDX	SILVOL
ABD. PAIN		INDIGESTION	SODESIA
GEN.	GEN.	GEN.	SPRAY
TOOTHACHE	CAVITY	CARIES TOOTH	SPUTUM
EARACH.	IMPACTED C.	OTITIS MEDIA	STRAPPED
CONJ.		CHR. RHEUM.	THERAP LAMP
NOSE BLEED	ULCER OF SEP.	NOSE BLEED	TA. GINGER
MALAISE	T.	MALAISE	WASH EARS
GEN. PAINS	P.	MYALGIA	
HEADACHE		HEADACHE	RET. TO WORK
		DERMATITIS.	SENT HOME

REMARKS:

TABLE 3. Sickness record.

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special industrial diseases or poisoning, clinical reports, x-ray reports and similar data are filed in the individual envelope as received. It has been found that the space on any general physical examination form is much too small for detail. In order that adequate records may be made, there should be special forms for recording heart, lung, and special examinations.

The American College of Surgeons has established the following minimum standard for medical service in Industry.

I. The industrial establishment shall have an organized medical department or service with competent medical staff including consultants and also shall have adequate emergency, dispensary, and hospital facilities and personnel to assure efficient care of the ill and injured.

II. Membership on the medical staff shall be restricted to physicians and surgeons who are (*a*) graduates from an acceptable medical school, with the degree of Doctor of Medicine, in good standing and licensed to practice in their respective states or provinces, (*b*) competent in the field of industrial medicine and traumatic surgery, (*c*) worthy in character and in matters of professional ethics; in the latter connection the practice of the division of fees, under any guise whatsoever, shall be prohibited.

III. There shall be a system of accurate and complete records filed in an accessible manner, such records to include particularly a report of injury or illness, description of physical findings, treatment, estimated period of disability, end results, as well as other information pertinent to the case or required by statute for Workmen's Compensation claims or other purposes.

IV. All patients requiring hospitalization shall be sent to institutions approved by the American College of Surgeons.

V. The Medical Department or service shall have general supervision over the sanitation of the plant and the health of all employees.

Industrial Medicine in a Small Factory

We have just outlined the practice of industrial medicine in a large factory employing over 1000 workers. The great majority of factories, however, are small.

"In 1929 nearly three-fourths of the manufacturing units in the United States were relatively small in size employing not more than 20 wage earners. Units equipped to employ more than 100 wage earners represented only 7.9 per

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cent of all manufacturing establishments. As employers of labor the relative importance of the two groups named is approximately reversed. While the smaller establishments provided for 9.9 per cent of all the wage earners engaged in manufacturing plants the larger establishments employed 70.8 per cent of the wage earners" [128].

While most large factories and mines employ a well-trained staff of doctors and nurses, these smaller factories either make no medical provision, sending their cases of industrial accident or industrial disease to the practitioners in the community, or employ a physician for part of a day, two or three times a week to make physical examinations and call him in when an accident occurs. The small factory is frequently equipped with a first aid room in charge of a nurse who cares for trivial injuries and calls in a physician for those of any consequence.

Many of these small factories have a severe health hazard of which the management is frequently unaware, and it is among such that advanced cases of industrial disease may be

expected. Thus the Special Industrial Disease Commission of Massachusetts [112] in 1933–34 found a large number of cases of silicosis among the workers in small granite cutting establishments. Similarly cases of lead poisoning may appear among the workers in a storage battery factory not under medical supervision.

The practitioner in the neighborhood of several small factories may be called in to treat a variety of industrial injuries and possible severe industrial diseases.

When a practitioner gives part of his time to one or more small factories, he adapts the basic principles in use in the industrial medical departments of larger factories to meet his particular requirements. Thus while physical examination may be done, it is impossible for a part-time doctor to make them prior to hiring. He must spend a part of his time in the factory, studying the types of work done and estimating the health hazard of each process. In order to do this a knowledge of all materials entering into the process and all by-products formed is essential, though difficult to ascertain.

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Insurance companies and the industrial physician. Useful information along these lines may be obtained from the insurance company which insures the factory under the Workman's Compensation Act. In starting physical examinations it has been found that if high officials, foremen, and older employees are willing to be examined, that the younger and less skilled workers will have no objection.

Industrial Medical Department's social significance. The Industrial Medical Department is a distinct public health asset. The constant medical supervision of a large group of workers, most of whom have families, the information as to facts of disease and of hygiene given them, the segregating and reporting of cases of contagious disease, and the constant effort to protect against industrial disease, all have an effect upon the general health of the community. Where the community is largely industrial the effect of several well-run industrial medical departments is considerable.

In any community the health of the citizens

and their families is cared for by the following agencies:

1. The state health department.
2. The local health department and public health nurses.
3. The school physicians.
4. The industrial medical departments.
5. The hospitals of the community.
6. The private physicians and nurses of the community.
7. The social service agencies such as
   a. Visiting nurse service
   b. Anti-tuberculosis society
   c. Clean milk stations.

"To consider a concrete example of the method of handling a public health condition which is familiar, let us consider the management of a case of early pulmonary tuberculosis developing in a worker while in the factory.

"The worker comes to the Medical Department complaining of cough and lassitude. Following the dispensary rule that every case of

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cough lasting more than a week shall be thoroughly investigated, the worker is given a complete physical examination from head to waist, inclusive. His temperature is taken and he is given a sputum box, which he then sends to the local health department. If positive signs of tuberculosis are found at the examination, the patient is referred at once to his family physician, and the department in which he works is notified not to allow him to return to work.

"If the patient presents questionable physical signs and an x-ray appears necessary, he is sent either to a local roentgenologist or to the general hospital for a series of diagnostic plates.

"If the case appears to be one of active tuberculosis and the patient has no family physician and cannot afford one, arrangements are made with the local hospital to care for him until he can be admitted to a state sanatorium. In either case the patient is visited at intervals by the industrial visiting nurse who reports his progress back to the Industrial Medical Department, and obtains advice and possibly relief for the patient's family through the local Tuberculosis Society.

"On the patient's discharge, arrested and able to work, a careful physical examination is made at the factory, its results recorded and the patient assigned to work for which he is fitted. Every effort is made to return him to his original work or to his original earning power if this is possible.

"In this sequence it is interesting to note that practically every health group of the community is called upon at some time between the first diagnosis and the final disposition of the case" [25].

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CHAPTER IV

INDUSTRIAL DISEASE

AN INDUSTRIAL DISEASE is one arising from and peculiar to a given occupation. Industrial Accident Boards, in states in which the coverage of industrial disease does not specify a definite group, consider as an industrial disease any which may result from an "injury" incurred at work. Such an injury might be the result of constant inhalation of a dust or fume occurring in the workroom or it might result from some bacterial infection peculiar to industry, as anthrax, or to some physical condition as in compressed air illness and in heat cramps.

Industrial diseases are, for the most part, toxemias resulting from continual exposure to

some toxic product or to an unfavorable working environment. We shall treat industrial diseases, therefore, by discussing first the environmental factors and then the effects of various substances of present importance in causing industrial diseases. We shall not attempt, in this small volume, to give an exhaustive presentation of the subject.

METABOLISM AND WORKING ENVIRONMENT

Although man is peculiarly sensitive to his environment he can adapt himself, without conscious effort, to great changes. In the course of a year he experiences annual outdoor temperature fluctuations of over 100°F. He heats his house in winter to 70° but enjoys out-door exercise at temperatures below freezing. His eyes are required daily to accommodate accurately at illumination intensities varying from 1 or 2 foot-candles to 10,000.

He changes his dress somewhat with the season but generally does not realize that he undergoes a very definite acclimatization each summer and winter. Sudden low temperatures in summer or high temperatures in winter may be se-

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rious catastrophes but if they occur in their normal course they pass unnoticed.

Effective temperature. It has long been known that there are various combinations of temperature, humidity, and air motion under which man feels comfortable. It is equally well known that in no group will all persons be suited by any given set of conditions—some will want the air warmer and some cooler. From the physiological and medical standpoints there is a broad zone of conditions which satisfies the majority and it is the aim of modern heating and cooling equipment to make the majority as comfortable as possible.

The engineer rightly demands a method or scale for appraising his results. After much study in coöperation with the U. S. Bureau of Mines, the Research Laboratory of the American Society of Heating and Ventilating Engineers at Pittsburgh devised the *Effective Temperature Scale* as an "index of the degree of warmth or cold felt by the human body in response to temperature, humidity, and movement of the air." They say further that "effective temperature is

not a temperature at all; it is a composite index which combines the readings of temperature, humidity, and air motion in a single value" [2].

In Figure 8 is shown in terms of effective temperature the Comfort Chart for *low air velocities*—mild air currents such as one experiences in a living room during the winter months. For practical purposes these low velocities may be considered as still air and are referred to as such in the literature.

As abscissas the chart gives dry bulb temperatures—those recorded by the ordinary thermometer; ordinates are wet bulb temperatures while the lines with the percentages indicated at the upper right hand corner of the chart give the percent relative humidity [171]. A temperature of 60°F. dry and 60° wet bulb gives an effective temperature of 60°. Note that the 60° effective temperature line runs down slightly to the right of vertical. At the bottom where it meets the 10 per cent relative humidity line it also intersects the 65° dry and 43° wet bulb lines. Thus we see from effective temperature chart that 60° air which is saturated or has 100 per cent

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relative humidity feels just as warm or just as cool as 65° air which contains but 10 per cent relative humidity. The chart shows two cross-hatched areas representing the comfort zones for winter and summer [172]. During the winter months most persons prefer the 66° E.T. region and in warm summer weather the region of 71° or over is the most popular. Such seasonal preferences in temperature are utilized rather extensively in general air conditioning, notably in theaters and department stores—in winter colder temperatures are maintained than in summer.

Within these so-called winter and summer comfort zones we look in vain for physiological optima. Although it has been shown that these comfort zones correspond in general to optima as interpreted from basal metabolism, morbidity, and mortality statistics, they are useful figures only for Americans, normally clothed and engaged in sedentary activities, and do not apply to other countries.

Ventilation and air conditioning. In stores and plants (other than the "hot industries") the industrial physician should not overlook ventila-

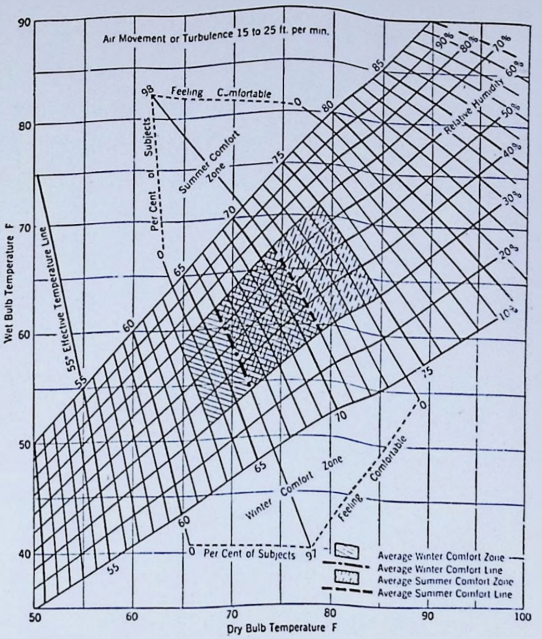


FIG. 8. Effective temperature chart for low air velocities. (*Courtesy of Professor Yaglou and the American Society of Heating and Ventilating Engineers* [2].)

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tion since it may have a marked effect on the workers. Not only their health, but to some degree their efficiencies also, will vary with the air conditions in which they work.

Ventilation is defined as "the process of supplying or removing air by natural or mechanical means to or from any space"[2]—it deals with the quantity but perhaps not with the quality of the air. Air conditioning includes ventilation but adds control of the quality of the air. Ventilation, in one form or another, we have always had but air conditioning is new and today is being exploited avidly.

A large scale example of simple ventilation is that afforded by the Holland vehicular tunnels under the Hudson River where ventilation is necessary to keep the carbon monoxide content in the exhaust of the motor cars down to safe levels. The fresh air brought into the tunnel is not heated, moistened, dried, or cleaned.

The modern moving picture theater would be intolerable either summer or winter without air conditioning. The heat supplied by the audience, even in winter, reaches such large figures that

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the air generally enters at a temperature lower than that in the auditorium. Department stores, restaurants, hotels, office buildings, and railroad trains all, more or less, utilize air conditioning. Recently it has been applied successfully to private residences, to operating rooms in hospitals, and perhaps will soon be within the range of the average householder's pocket book.

Recirculation and Odors

From the standpoint of engineering it is desirable to recirculate as large a percentage of indoor air as possible. Recirculation reduces the heating load in winter and the cost of cooling in summer. The space being ventilated always will be more or less contaminated by body odors, tobacco smoke, chemicals, or dusts. If these impurities are to be kept down to reasonable healthful limits, they must be removed in part by some special equipment or else sufficient uncontaminated air must be brought in, tempered to the desired conditions, and mixed with the air in the room.

There is no physiological basis for the use of

a constant set quantity of out-door air per person per minute. The familiar 30 cu. ft. per person per minute, so widely used in various state laws, should be changed and conditions judged on the basis of results obtained and not on the type and rated capacity of any of the equipment. Yaglou finds that 10 to 15 cu. ft. of clean out-door air per adult per minute is a reasonable figure for the winter months while 20 to 30 feet are needed in warm weather. He explains these differences as owing to odors from insensible perspiration which increases with temperature, or more accurately, with "effective temperature."

These figures apply to occupied spaces which are ventilated but not air conditioned. Air conditioning, particularly dehumidification, will effect a certain degree of odor control.

The physician who is asked today to give his opinion of the air conditions in a theater, a restaurant, school, or office should notice odors in addition to temperatures and drafts and judge the extent of odors by his first sensation on entering from an odorless place. There is no longer any excuse for asking people to do office

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or desk work in smelly or badly ventilated rooms any more than it is necessary for factory or mine workers to be exposed to dangerous amounts of dusts or gases. Perhaps the physician cannot measure with a ruler the benefits resulting from eliminating non-toxic odors, but neither can he measure the benefits resulting from many other rules of conduct.

The ventilation figures we quoted from Yaglou apply only to the control of average human odors and not to tobacco smoke or intense odors as from cooking. Also they do not apply to control of harmful impurities like gases or dusts. Such contaminants are common in industrial medical practice and each must be judged for itself. The index of permissible recirculation or of adequacy of ventilation is, after all, the health and comfort of the persons concerned.

High Temperatures and Salt Depletion

In moderate exercise as represented by a man walking 2 miles an hour and consuming 0.65 liters of oxygen per minute, body heat is lost mainly by radiation, a lesser percentage is given

off by evaporation of sweat from the skin, and a little as moisture in the exhaled breath. If the environmental temperature is raised, a greater proportion of heat is given off by evaporation of sweat, less by radiation, and the panting effect increases slightly.

It has been known for many years that men exposed for long periods to severe heat in which they sweat continuously are liable to heat cramps which may not appear until several hours after stopping work. The actual cramp may be induced by any slight movement and often is excessively painful.

Edsall, followed by Haldane, Moss, and Glover [59], suggested the drinking of salty water to relieve this condition and for the specific purpose of making up the salt lost in the sweat. Ice water, once thought harmful, is not dangerous, according to Sayers, but it does not relieve cramps as salt does. A quicker method of relieving salt deficiency is, of course, by intravenous injection of physiological saline. Sweat contains about 0.25 per cent chlorides, usually recorded as sodium chloride, although

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potassium chloride makes up about one third of the total. This latter chloride has a somewhat diuretic effect, according to Hogler and Ueberrack [59], and is very bitter. If the loss of salt is allowed to continue for long periods, the organism is unable to retain fluids, urine, and sweat, and this disability induces the cramps. During this period of extreme salt lack, salt concentration in the urine falls far below normal.

Glover describes this chain of events, gives an admirable review with bibliography, and shows from his own experience that machines dispensing 16-grain salt tablets are popular and effective among workmen exposed to severe conditions. Haldane and Moss advocated drinking water containing 10 grams of salt per gallon, but in our experience it is unnecessary to judge the concentration to such a nicety. The salt-depleted individual simply takes one or two 16-grain tablets with a little water.

The time honored drinks, barley and oatmeal water, are generally popular in hot industries, but not unless well salted. Likewise salted beer is popular in hot weather.

It is usually found that the inexperienced worker is more affected than the veteran and generally drinks more water. Probably his cooling by sweating is less effective than that of the seasoned worker. The veteran often works in a cotton undershirt while the novice strips to the waist. The sweaty, shirt-clad individual actually presents a greater and more effective evaporative or cooling surface through the porous surfaces of his shirt than does the wet naked skin of the novice. Under very severe conditions, then, stripping to the waist is not generally as effective as working in a cotton undershirt.

Heat stroke. The loss of consciousness from over-exertion in very hot atmospheres generally comes slowly and with ample warning, particularly to the experienced persons who are, therefore, rarely incapacitated by heat. The pulse rate and blood pressure gradually increase, and with them body temperature. In experimental work we have often raised the body temperature well over 102°; the subject becomes irritable and querulous long before he is in danger of collapse.

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Actually one is more likely to meet cases of true heat stroke or sun stroke in ordinary life than in industry. Such cases are an annual occurrence in Fourth of July parades. The victims may be brought in unconscious and quickly may run a temperature of 110° or even more. They should be given ice baths, ice sponging, and ice water enemata until the temperature is reduced to about 102° or 103°. They should not be cooled to normal as the body temperature may continue to fall for a time after the ice baths have been stopped.

Compressed Air Illness

For somewhat less than 100 years man has been sinking caissons for bridges, piers, and submarine tunnels. Early in the nineteenth century Siebe, in England, supplied suits and equipment for deep diving. Paul Bert [12], the French physiologist, in 1878 published his studies on the effects of high and low barometric pressures and described at length the basic physiology of compressed air illness. The best short account

of the subject is by Haldane [65], while Hill [82], Levy [107], and others have published more extensive articles.

In caisson work, compressed air is used so that water or mud or both will not seep into the working place. The air pressure is kept, therefore, at a figure slightly above that of the water or mud against the shield or shell of the caisson. There is always some outward leakage of air rather than a leakage of water in.

In deep diving the diver wears a suit and helmet which are tight against both water and air. His helmet is connected to the surface by at least two hose lines, one for compressed air and one for his telephone. As he descends he regulates the air supply so that the upper part of his suit remains inflated and air escapes in a steady stream of bubbles through the exhaust valve in his helmet. At a depth of 33 feet his suit must be inflated by a pressure of one atmosphere, at 66 feet to two atmospheres, and so on. So far as compressed air illness goes he has the same risk as the caisson worker but with the added hard-

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ships of cumbersome equipment and cold water.

A healthy person who enters an air lock or tank and who is gradually subjected to pressures above normal notices first the effect upon the middle ear—he must equalize the pressure on both sides of his ear drum by swallowing or by holding the nose and blowing. Failure to do so can result in a ruptured ear drum and the precautions noted are carried out automatically by all persons familiar with compressed air work. At about 1½ atmospheres one's speech becomes somewhat thickened and at about 2 atmospheres it is impossible to whistle. Smoking is difficult if not dangerous, since the tobacco burns too vigorously or may even burst into flame. Since the compression of air generates heat, the men are likely to take off clothing during compression and conversely to put it on again during decompression.

Compressed air illness "is a condition caused by a too rapid decompression after exposure to higher pressures for a period of time. It is characterized anatomically by the presence of free nitrogen in the tissues and body fluids and clinically by one, or a combination, of the following symptoms: Localized pain, vertigo, prostration, or symptoms referable to the central nervous system" [107].

Nitrogen. The rate or the extent to which the nerve tissues and the body fluids absorb nitrogen depends first upon the air pressure and secondly upon the duration of the exposure. One may enter three atmospheres pressure as fast as the valves and air lines will permit, remain a few minutes, and decompress at full speed with impunity, but if one stays at three atmospheres for 2 hours, decompression must be carried out according to a definite schedule, for the origination of which Haldane is responsible. If one comes out of two atmospheres pressure rapidly after long exposure, nitrogen bubbles form and with them comes the inevitable attack of the bends.

Haldane showed that, regardless of length of exposure or pressure, one can halve immediately the *absolute pressure*[1] while from that point on

[1]The absolute pressure is the gauge pressure plus the ordinary sea level pressure, which is 14.7 lbs. per square inch or 760 mm. mercury.

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the drop is made in regular steps or at an even rate. The ultimate result has been the formation of safety schedules governing the permissible exposure and the method of decompression. Levy gives the following from the New Jersey law:

Section 10. . . . . In every case the maximum number of hours shall be divided into two working periods of equal length, and the minimum time interval which shall elapse between such working periods shall be as hereafter stated:

When air pressure—	*Number of hours in 24*	*Interval between working periods*
Exceeds normal, but does not exceed 20 lbs.	8	30 minutes
Exceeds 21, " " " " 30 "	6	1 hour
Exceeds 30, " " " " 35 "	4	2 hours
Exceeds 35, " " " " 40 "	3	3 "
Exceeds 40, " " " " 45 "	2	4 "
Exceeds 45, " " " " 50 "	1½	5 "

Except in cases of emergency no person shall be permitted to work or remain in any tunnel, caisson, compartment, or place where air pressure exceeds 50 lbs.

Decompression in Locks

Sec. 11. . . . . When the employee is passing from a caisson, compartment, or place to atmosphere of normal pressure, the time of decompression shall be as follows:

	Time of decompression in minutes
Exceeds normal, but does not exceed 10 lbs.	1
Exceeds 10, but does not exceed 15 "	2
Exceeds 15, but does not exceed 20 "	5
Exceeds 20, but does not exceed 25 "	10
Exceeds 25, but does not exceed 30 "	12
Exceeds 30, but does not exceed 36 "	15
Exceeds 36, but does not exceed 40 "	20
Exceeds 40, but does not exceed 50 "	25

The U. S. Navy in their submarine rescue work has had to disregard these civilian specifications on many occasions. We have seen their divers ascend from 100 feet (corresponding to 3 atmospheres), be stripped of their diving clothes in 3 or 4 minutes, be recompressed at once and then brought out by stages in the usual way. Advantage is taken of the fact that nitrogen bubbles require several minutes to form and it is perfectly safe, therefore, to decompress in a comfortable lock on the deck of the mother ship instead of in water which may be icy cold and often inconveniently rough.

Symptoms. The symptoms of compressed air illness fall into two groups, serious and mild, depending upon the quantity and the location of free gas in the body. Gas bubbles in the heart

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and blood vessels of the lungs and the central nervous system cause collapse, asphyxia, vertigo, and paralysis, while bubbles localized in the extremities produce the less serious though frequently intense pains known as the bends. The symptoms may simulate those of many diseases and frequently are mistaken for alcoholic intoxication.

Treatment. Workers in compressed air should be required to wear tags stating their occupation and the location of the recompression chamber. Any abnormal manifestation following exposure to compressed air should be treated by immediate recompression.

Since about 85 per cent of the cases of compressed air illness occur within the first hour after decompression, it is advisable to keep the men on the premises and under observation for at least an hour if any chance of illness is in prospect.

The value of oxygen inhalations combined with recompression in the treatment of compressed air illness has been known for some time. The toxicity of oxygen at high pressures, discovered by Paul Bert [12], has restricted its use in compressed air work but Behnke [9] has shown recently that pure oxygen can be breathed safely for half an hour at 4 atmospheres pressure[1], for 2 hours at 3 atmospheres, 3 hours at 2 atmospheres, and for 4 hours at 1 atmosphere.

In view of the unsatisfactory results and the length of time required when recompression is carried out in air, Behnke and Shaw [10] have reëmphasized the value of oxygen therapy and have outlined the following schedule for the treatment of compressed air illness:

1. If an individual complains of pain or shows signs of circulatory or respiratory distress following decompression, oxygen inhalation in the pressure chamber should be given immediately. The oxygen can be administered by means of a mask attached to a rubber bag or to the usual basal metabolism spirometer.
2. If the symptoms are severe or are not relieved by oxygen at 1 atmosphere, recompression *with oxygen inhalation* should be applied.
3. If it is necessary to raise the pressure above

[1]Convulsions and loss of consciousness occur at this pressure after 45 minutes exposure so that, in practice, 3 atmospheres pressure of pure oxygen may be regarded as the safe limit.

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3 atmospheres, the partial pressure of oxygen can be kept at this level by diluting the oxygen with air.

While any illness may occur coincidently with or following decompression, the symptoms of pain or any abnormal manifestation following exposure in compressed air require treatment for compressed air illness. If the symptoms are the result of too rapid decompression, oxygen inhalation and recompression will bring immediate relief. This therapeutic test will determine the diagnosis in any doubtful case.

CHAPTER V

DUSTS

DUSTS, FUMES, MISTS, AND SMOKES

PHYSICAL FACTORS

DUSTS ARE FORMED by processes of abrasion, attrition, grinding, blasting, and the like. Typical examples are the dusts thrown into the air by rock drilling or sandblasting. Such dusts have the same chemical compositions as the rocks from which they are derived; they have not been altered chemically by the process of subdivision. Their particle size will range from ultra-microscopic up to the visible (Fig. 9).

Fumes result from some chemical or physico-chemical change, for instance zinc-oxide is formed by burning zinc vapor arising from the crucible containing molten brass, or lead is found in the exhaust of a motor car burning gasoline

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containing tetra-ethyl lead. Particle size is of the 0.1 to 1.0μ[1] order (Figs. 10, 10a).

Mists are represented by the common atmospheric fogs and are essentially tiny liquid droplets, often microscopic in size, condensed about solid particles like carbon or salt as the nucleus. A rather common example of interest in industrial hygiene is the mist entrained from the acid used in chromium plating baths.

Smokes from burning carbonaceous fuel like coal, oil, and wood contain droplets as well as dry particles. Tobacco smoke typifies the wet smokes and is in reality composed of minute tarry droplets. Particle size here is of the order of 0.25μ.

No fine particles can float for long in quiet air unless their particle size is well under 10μ. A healthy red blood cell is about 7.8μ, a white cell about 16μ, and a pollen of giant ragweed (Fig. 11) about 18 to 25μ. A person with normal eyes can easily see an object 50μ in diameter or pick up a shiny fiber of quartz 20μ in diameter.

[1] 1 μ = .001 mm. = 1/25,000 inch.

In the lungs of men who have died of silicosis, in the sputum of men exposed to dust (Fig. 12), and in the lungs of animals which have been dusted, the most representative particle size is about 1μ. Recently Gardner and Cummings [57] showed that dust below 3μ was very effective in producing silicotic pathology in the liver of experimental animals, whereas 12μ dust was not. Policard [137] believes that the lower limit of hygienically significant dust—that is, of the silicosis producers—has not been fixed but that it certainly is under 1μ. Bloomfield [13] maintains that 1μ average represents the dusts ordinarily found in industry, a statement on which Hatch and Pool [75] have thrown much doubt. Drinker [43] produced metal fume fever in subjects who breathed 0.6μ zinc oxide fume (Fig. 10) and also with oxide particles averaging 0.15μ. It is probable that radium poisoning could be produced with radio-active dusts well below 0.1μ in size.

Hay fever and other allergic types of disease from organic dusts of the larger particle size, can

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be caused by breathing the offending material into the nose and not having it reach the lungs at all [17]. Ragweed pollen (Fig. 11), wood dusts, dust from castor beans, and numerous other substances can be air floated, and if breathed, can produce some disability. It is incorrect to maintain that we are concerned in industrial medicine only with the 0.5 to 5.0μ dusts.

In mists, which are mostly water, particle size is of no particular importance, but the droplets must be in the microscopic range if they are to travel any distance. A year or two ago one could see a silica wash being used to face the moulds in steel foundries and observe this wash being applied with a spray gun. All persons in the immediate vicinity were then showered with a mist of which the essential ingredients were water and fine silica made from ground flint, one of the best silicosis producers known. Spray painting likewise is a known dust hazard, the dust being transported in a droplet of paint solvent such as linseed oil and turpentine, or one of the amyl acetate base lacquers.

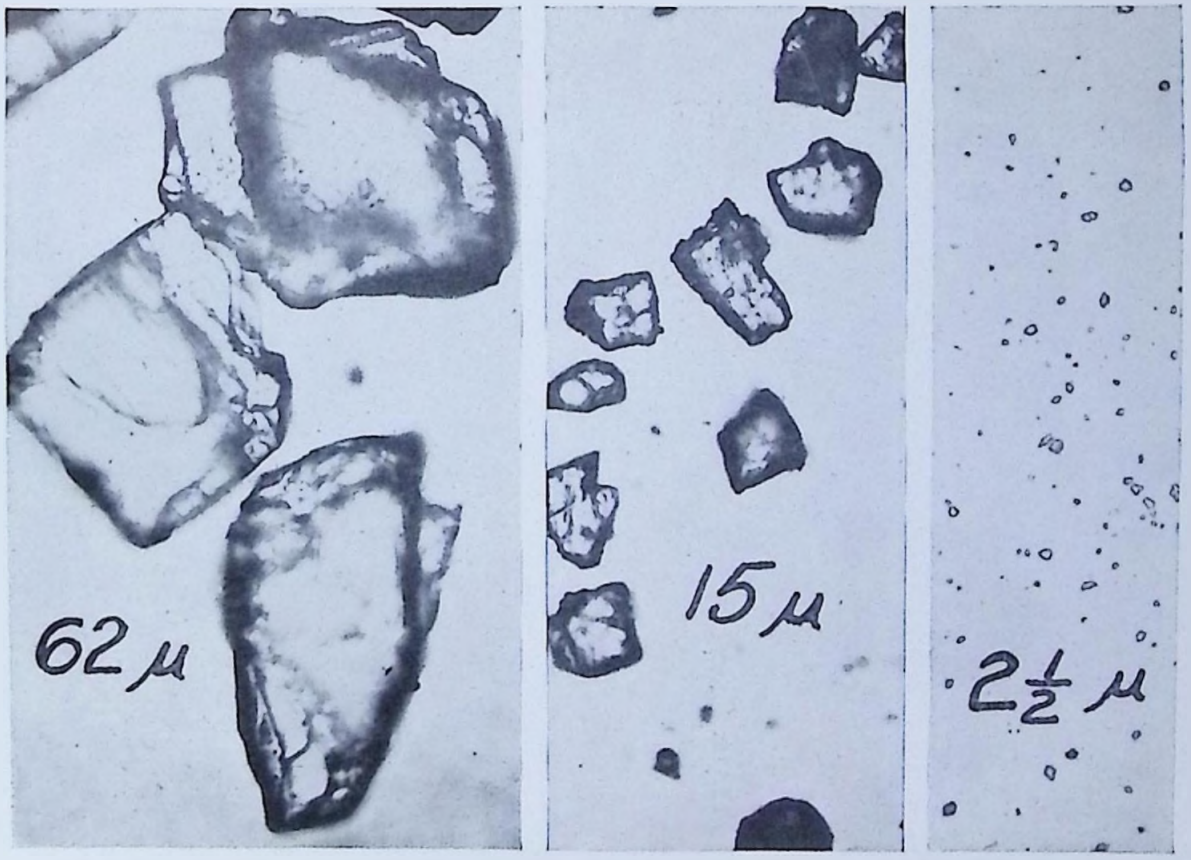


FIG. 9. Particles of marble dust separated into different groups according to size by repeated settlings in water. x500. (*Courtesy of the J. Indust. Hyg.*)

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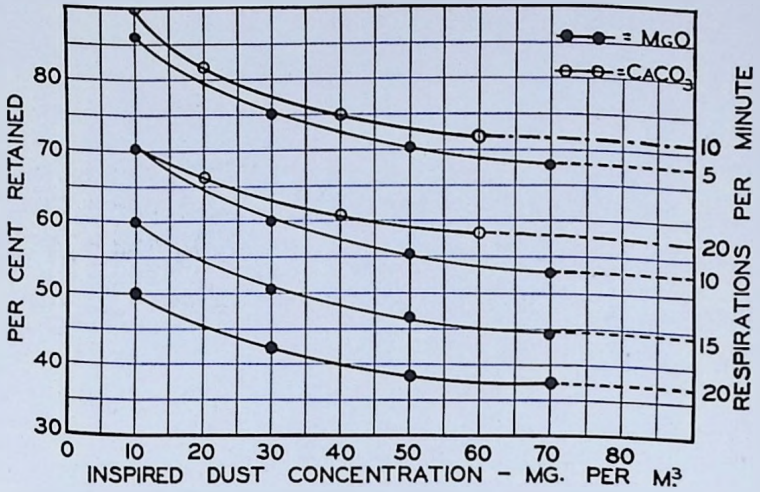


FIG. 13. Brown's dust retention curves. Average percentage of magnesium oxide and calcium carbonate retained by subjects breathing at respiration rates indicated. (*Courtesy of Dr. Brown and J. Indust. Hyg.* [18].)

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lead from entering the organism. When absorbed by the lung, lead at once enters the general circulation—all the absorbed lead is distributed by the systemic blood.

"Lead is absorbed by the gastro-intestinal mucosa, the entire respiratory tract [17], and subcutaneous tissue. When it enters by the gastro-intestinal route, lead is transported in the portal blood to the liver. This organ protects the rest of the organism rather efficiently by removing most of the lead carried in the portal vein and by excreting a large part of it in the bile.

"Lead compounds insufflated as solid particles are absorbed directly into the general circulation. Thus lead is distributed throughout the organism much more rapidly when it enters through the respiratory tract than when the liver can exercise its protective function of removal and excretion during the process of absorption."

K. B. Lehmann, Sayers, Drinker, Brown [18], and Ishikawa determined on men and animals the percentage of various dusts retained when known concentrations in air were breathed. Of

these studies, Brown's gives the most useful information. He showed that retention increases with particulate size[1] and inversely with respiratory rate up to the rates of about 20 breaths per minute. Mouth breathers had retentions considerably lower than nose breathers, the difference being a direct index of the dust retained by the nasal filtering mechanism. In Figure 13 are shown Brown's most suggestive data.

The obvious conclusions from Brown's data are that in dusty atmospheres rapid shallow breathing is better than slow deep breathing and nose breathing is better than mouth breathing. As he worked only with particles in the 0.4 to 3.0μ range his data cannot be applied to the asthmatic or allergic types of dusts such as pollen and numerous other organic materials having particles many times larger than those in dusts causing metal fume fever, lead poisoning or silicosis.

Individual variation. More recently G. Lehmann [105] has studied the dust filtering capac-

[1]Particulate size refers to the size of the masses or aggregates of particles, while particle size refers to the individual discrete particle.

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ity of the human nose and has obtained very suggestive results. His subjects were selected at random from ordinary healthy persons and from groups with known dust exposure. Of the latter some were definitely silicotic and many were perfectly healthy. His subjects remained seated and held their breath for a moment during which dusty air was blown into the nose and escaped through the mouth. Dustiness of the affluent and effluent streams were estimated by a modification of the konimeter. Since no dust was breathed by the subject Lehmann was able to use silica while Drinker and Brown were forced to use harmless materials like limestone and magnesium oxide.

Lehmann found human nose filtering efficiencies varying from about 10 to 70 per cent, the highest figures usually being for the younger subjects. Of his workmen exposed to dust those with poor nasal filters showed the greater susceptibility to silicosis. The conclusion is inescapable that Lehmann's technic may well have a practical place in physical examinations of men applying for dusty jobs. It is well recognized,

as Lehmann points out, that silicosis susceptibility or personal idiosyncrasy varies immensely and quite unpredictably—we cannot select by any recognized examinations the men best suited to dust exposure. Here, suggests Lehmann, is a possible explanation to this heretofore elusive factor of susceptibility.

Further confirmation is required before Lehmann's suggestion can be accepted fully. His procedure in controlling and measuring his dust clouds is open to criticism, but very strongly in his favor is the constancy of his results and more particularly the large number of workmen, 426, tested.

The fate of inhaled dusts. Relatively insoluble dust particles which have passed through the nose or mouth and have thus entered the trachea are eliminated from the body in a number of ways, or else they may remain in the lung tissue [54], [98], [133].

They may be exhaled according to the general laws of retention as noted previously. They may be caught on the walls of the bronchi or the bronchioles and be removed by ciliary action

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without either phagocytosis or solution having taken place.

They may pass to the alveoli before being trapped, where they are taken up by phagocytes derived from the alveolar epithelium [21]. Since there is no mucus in the alveoli and no cilia, the all-important step in elimination from the alveoli is phagocytosis.

Once phagocytosed these cells can migrate by amoeboid movement to the finer bronchioles where ciliary action commences, or the cells pass into the lymph circulation and are stopped or filtered out at the lymph nodes, particularly the tracheo-bronchial nodes.

As the dust cells themselves are relatively short-lived, they may die natural deaths, disintegrate, and thus deposit their dust burden, which may or may not be taken up again by other dust cells. Such deposition is found particularly in the small pulmonary lymphatics and may cause no pathology whatever.

Or the dust may be definitely toxic to the cell and kill it rather quickly. The dead cell then becomes mummified, the surrounding tissue be-

gins to lose its spongy elasticity, fibroblasts form, and true fibrosis commences.

The ultimate results are areas or lung volumes with fibrosed tissue in which the capillary circulation is definitely and significantly reduced. The fibrosed portions of the lung are then worse than useless so far as respiration is concerned.

Finally, the fibrosed areas, particularly if quartz was the causative factor, are rendered much more susceptible to the growth of tubercle bacillus than is normal tissue. The terminal result of excessive inhalation of dust with a high quartz content is generally tuberculosis [28].

The normal defensive mechanism can therefore cope with a moderate or slight degree of quartz dustiness. Elsewhere, we discuss permissible dust concentrations both for quartz and for other substances.

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Chapter VI

PNEUMOCONIOSIS

The prevalence of pulmonary disease, notably of tuberculosis, among men exposed to mineral dusts has been known and described for almost four centuries. Miners' phthisis, stone cutters' asthma, grinders' rot, and similar terms have been recognized for years in the trades concerned. Within the last 30 years there has been an increase rather than a decrease in pulmonary disease in the dusty trades and this increase has followed inevitably the uncontrolled pulverization of rock containing a high percentage of free silica or quartz.

Among physicians and pathologists a certain amount of confusion in nomenclature resulted

because the silicotic or dusted lung generally appeared black, the black color obscuring the more important substance, quartz, which is white. The word anthracosis (denoting a lung blackened with carbon) dates back, according to Willis [170], to Stratton in 1838. Zenker coined the word *pneumonoconiosis* in 1867 [174], and with it siderosis (from iron dust). A few years later came silicosis, followed slowly by other dust "-oses," such as chalicosis, calcicosis, asbestosis and silicatosis. Doubtless there will be others ere long.

American writers, notably Pancoast and Pendergrass [132] about 1925, shortened the more inclusive term to *pneumoconiosis,* which is now current usage. Then followed a brief period in which silica, in the form of quartz, was given the blame for all, or nearly all, dust fibroses with the result that anthracosis, siderosis, chalicosis and the rest were more or less shelved as misnomers. In 1933 Jones [90] offered convincing evidence against the mineral, sericite, while Policard [137] supported Jones and pleaded for reintroduction of the word pneumoconiosis and

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more circumspection in the promiscuous use of the word silicosis.

Today the word pneumoconiosis includes the manifestations of all dust inhalation whether the dust is injurious or harmless; silicosis, on the other hand, presupposes a perfectly definite etiological factor, free silica or quartz, and embraces by far the greatest part of the morbid pathology for which dust inhalation is directly or indirectly responsible.

Reviews of the pneumoconioses, particularly silicosis, have been published by Collis [27], Rice [81], Pancoast and Pendergrass [133], Greenburg [61], Sayers [149], Teleky, and Willis [170]. The Johannesburg Conference [88] of 1930 brought the subject up to date while the Saranac Silicosis Symposium of 1934 [147] presented informally the present trends of current opinion on silicosis. The classified critical bibliography of the International Labour Office [86] lists 633 references on pneumoconiosis, while the recent compilation by Davis [33] gives some 2750 unculled references—a veritable wilderness of confusing repetition. Brief but carefully

prepared booklets for the layman have been published recently by the Metropolitan Life Insurance Company [121] and by McCord [118] for the National Founders Association.

Silica and Silicosis

Lanza [102] uses the definition of silicosis adopted by the Committee on Pneumoconiosis of the American Public Health Association, 1932: "Silicosis is a disease due to breathing air containing silica (SiO_2) characterized anatomically by generalized fibrotic changes and the development of miliary nodulation in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present) and by characteristic x-ray findings."

Since silica is the etiological factor in silicosis, the most important of the pneumoconioses, the silica content of the dust breathed should be known. Silica occurs in nature in most rocks while the mineral quartz (pure SiO_2) is also

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widely found. Among the common forms of free silica are quartzite, flint, sandstone, chert, chalcedony, tripoli, diatomaceous earth, and opal.

Silica combines with many of the basic oxides, like magnesium oxide to form silicates with formulas such as $MgO.SiO_2$. Some common silicate minerals in wide industrial use in powdered form are feldspar, mica, talc, or soapstone, various clays, chrysotile (asbestos) and garnet.

One must distinguish between minerals which contain pure silica like quartz, and the silicates, like talc. Quartz has been proven beyond reasonable doubt to be phagocytosed slowly, to cause definite cell necrosis, cellular mummification, and to render lung tissue inordinately susceptible to tuberculosis. From the standpoint both of geology and physical chemistry, all forms of pure silica are by no means alike; neither is their physiological action alike, but we have accurate physiological data on only a few.

Of the silicates so far studied only asbestos (and possibly sericite) have the specific power to produce disabling fibrosis of the lung tissue,

but as yet no mineral dust other than silica has been found actually to render tissue unduly susceptible to tuberculosis. One must never lose sight of the fact that the usual cause of death in the fatal pneumoconiosis is infective tuberculosis and not tissue fibrosis. It is reasonable, therefore, always to ask what the "free silica" content of a dust may be before deciding whether it is likely to produce fibrosis.

A significant table has been made by Thompson et al. [163] showing the relation of the quartz content of rock dust to causation of silicosis (Table 4).

Symptoms—Silicosis (Simple)

Silicosis has usually been divided into three stages. These stages in England, South Africa, and Canada are called ante-primary, primary, and secondary. In the United States they have been classified as first, second, and third stages. Recently Pancoast and Pendergrass [134] [136] have reclassified the disease according to x-ray interpretation into the perivascular-peribronchial-lymph node type, the nodular type and

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TABLE 4

SUMMARY OF THE SIX DUST STUDIES BY THE U. S. PUBLIC HEALTH SERVICE SHOWING THE DUST CONCENTRATION, COMPOSITION, AND THE RESULTING HAZARD.

Study	*Average dust count in millions of particles per cubic foot of air*	*Average percentage of free silica (Quartz)*	*Other characteristics of dust*	*Degree of hazard (under conditions as observed in each study)*
Granite cutting:				
a. Hand-pneumatic tool operators	59	35	Balance mostly combined silica.	Great excess of pulmonary tuberculosis after 15 years or more exposure; silicosis in from 2 to 10 years.
b. Surface-machine operators, etc.	36			Silicosis after prolonged exposure; no excess of tuberculosis.
c. General air	20			
d. Less than general air	9			Negative except for occasional non-disabling silicosis.
Anthracite coal:				
Rock drillers	82	31	Siliceous rock	Data insufficient; other studies show severe hazard.

Miners and miners' helpers	232	1.5	Carbon and inorganic matter	Dyspnea and other signs of pneumoconiosis; excess sickness from respiratory conditions; excess mortality from influenza-pneumonia and possibly tuberculosis.
Bituminous coal:				
Rock drillers	78	54	Sandstone	Data insufficient; other studies indicate severe hazard.
Loaders and machine men	112	1.2	Carbon	Generalized fibrosis chiefly linear in character; excess mortality from influenza and pneumonia.
Cement	26	6–8	Primarily lime	Some early pneumoconiosis; excess of disease of upper respiratory tract and of influenza.
Cotton-cloth manufacturing	7	?	Vegetable and silica	Negative
Silverware manufacturing	5	1.7	Metal and other	"
Municipal	4	?	Not determined	"

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the interstitial type. Each of the two latter types may progress into an advanced interstitial predominance, rapidly or slowly progressive; and an advanced diffuse or terminal fibrosis, conglomerate nodular in type, interstitial in type or massively fibrotic in type.

In the early stages Irvine [89, p. 51] gives the following description of the clinical signs.

1. A certain lack of elasticity of the chest wall during the movements of respiration together with
2. A somewhat reduced air entry, and
3. A characteristic alteration of the inspiratory murmur from the normal "vesicular" character to a higher pitched or "harshened," thinned, and commonly somewhat shortened type, the expiratory murmur, although somewhat prolonged, remaining fainter than the inspiratory. This complex of physical signs is almost constantly present in cases of a slight degree of "simple" silicosis. The cough may be put down to the coincident bronchitis and bronchiolitis, the recurrent pains to slight intercurrent pleurisies.

The physical signs do not change greatly in later stages of the disease—there may be adventitious sounds as a result of intercurrent infection. "The percussion note is somewhat flattened without being definitely dull, especially posteriorly. Breath sounds have more definite, characteristic thinning and harshening of inspiration, the expiration being longer and fainter" [148]. There is no undue loss of weight unless there is a complicating tuberculosis when the definite and progressive loss of weight is noticeable.

The cardinal symptom of silicosis is dyspnea due to the restricted lung space left for breathing and to the compensatory emphysema. This is usually accompanied by a dry, irritating cough, more severe in the morning. There is anorexia, especially in the morning and the patient often complains of epigastric discomfort and sometimes pain.

The dyspnea gradually increases. If respiratory infection occurs the cough loosens and sputum, often discolored by dust particles, is raised. Broncho-pneumonia is a frequent complication, lobar pneumonia is not. About 75 per cent develop pulmonary tuberculosis with the

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accompanying symptoms. As the disease progresses there may be signs of heart enlargement, and ultimately heart failure. Few patients die of uncomplicated silicosis; most die of pulmonary tuberculosis or some other lung complication.

X-Ray

The most important evidence of pneumoconiosis is obtained by x-ray. An x-ray examination should never be omitted in any case suspected of having silicosis or silico-tuberculosis.

The examination consists first of a flat film taken in the posterior-anterior position in order to determine whether the chest shows signs needing further investigation. If the flat film is suggestive a stereoscopic set of films is taken. Frequently a picture which apparently shows pneumoconiosis in the flat film fails to show it stereoscopically.

A lateral flat film is also taken in questionable cases. Fluoroscopy is important to determine diaphragmatic adhesions.

Following the 1934 Saranac Symposium on

Silicosis a group of experts prepared the following tabulation (Table 5) of the roentgenological changes found in silicosis together with the underlying histological conditions. This tabulation follows the classification of Pancoast and Pendergrass already mentioned.

It should be noted that numbers two and three, (Figs. 14, 15) approximate stage one in the earlier classification, that number four (Fig. 16) approximates stage two and that number five (Fig. 17) approximates stage three.

TABLE 5

A Tabulation of the Roentgenological Changes Found in Silicosis Together with the Underlying Histological Conditions [123]

Prepared by Drs. Sayers, Pancoast, Pendergrass, McConnell, Sampson and Gardner

Healthy Lungs and Adnexa

Roentgenological Appearances	*Histological Appearances*
1. *Healthy Lungs*—as defined by the N.T.A. Committee Report (Nat. TB. Assn.)	1. Essentially the normal tissues of the vascular tree, the mediasternum, the bronchi and trachea.
2. Irregular exaggeration of the linear markings, with	2. Cellular connective tissue proliferation about lym-

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Roentgenological Appearances	*Histological Appearances*
possibly some beading, confined to the trunks.	phatic trunks in the walls of vessels and bronchi. Beading may be due to various causes as blood vessels seen end on, arteriosclerosis, minute areas of fibrosis in lymphoid tissues along the trunks.
3. Increased root shadow.	3. Cellular reaction in the tracheo-bronchial lymph nodes with extensions along afferent lymphatic trunks.

These changes come within normal variations when not accompanied by recognized organic disease.

Simple Silicosis

Roentgenological Appearances	*Histological Appearances*
4. *Nodulation*–Discrete shadows not exceeding 6 mm. in diameter tending to uniformity in size, density and bilateral distribution, with well defined borders surrounded by apparently normal lung shadow. The outer and lower lung fields characteristically show fewer nodules.	4. Circumscribed nodules of hyaline fibrosis located in the parenchyma of the lung. Occasionally some of these nodules may show microscopic foci of central necrosis.
5. *Conglomerate shadows* — that appear to result from a combination or consolidation of nodulation usually with associated emphysema manifested by:	5. The result of coalescence of discrete nodules; an area in which the nodules are closely packed and most of the intervening lung is replaced by more

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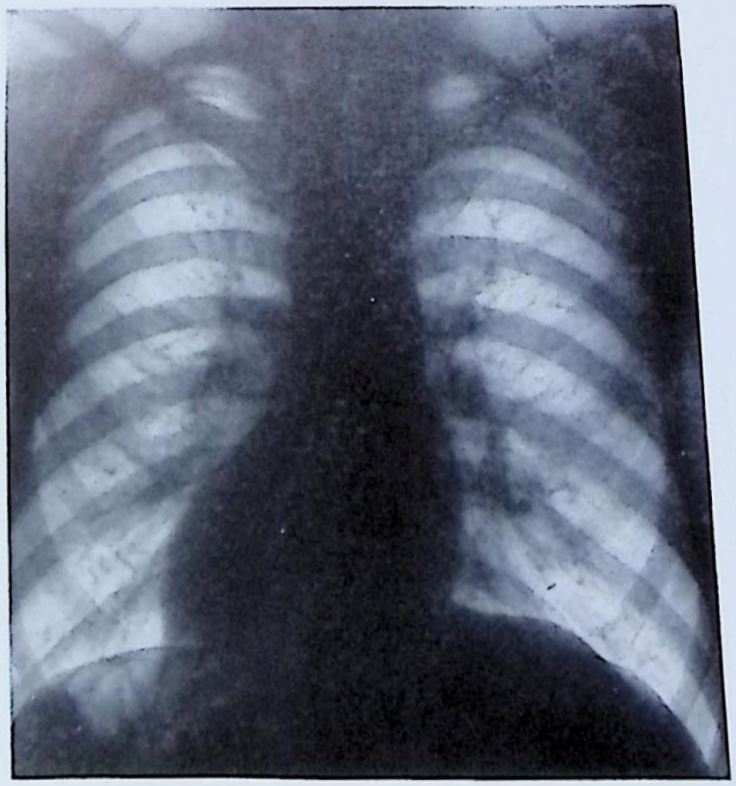


FIG. 14. Roentgenogram of the chest of a male, aged 29 years and a coal cutter for 8 years, showing the prominent trunk and hilum shadows of the so-called first stage of pneumoconiosis of the American classification, but without nodulation or a slight haze in the midlung fields, either or both of which would characterize the appearance as due to definite pneumoconiosis. (*Courtesy of Drs. Pancoast and Pendergrass* [136] *and J. Indust. Hyg.*)

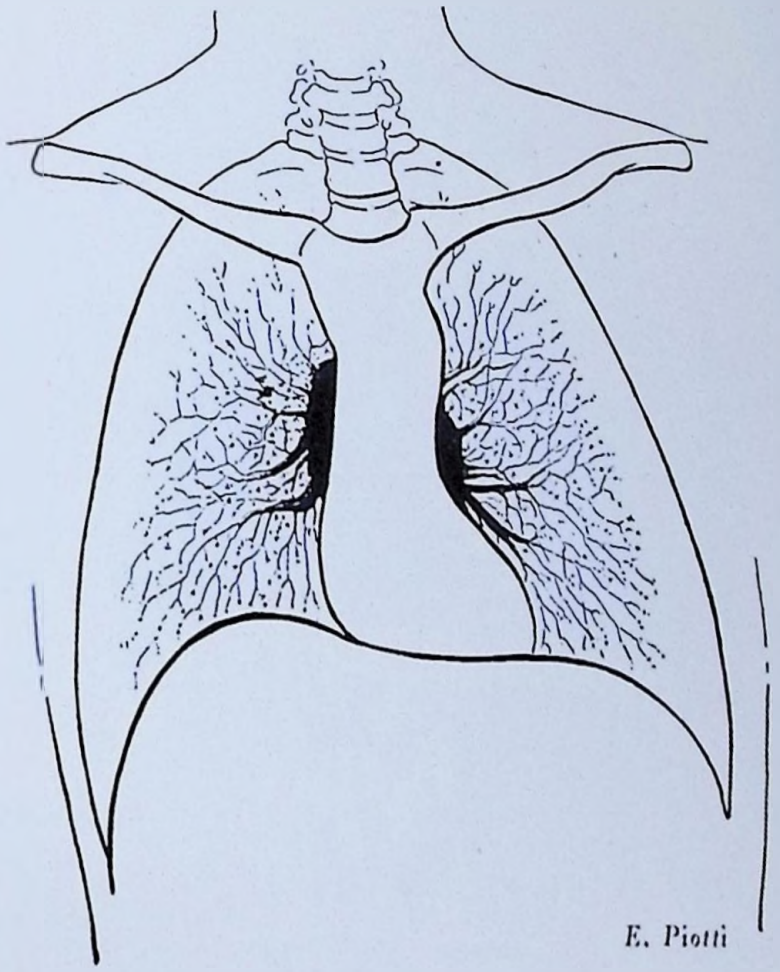


FIG. 15. Line drawing illustrating Stage 1, corresponding to Fig. 14.

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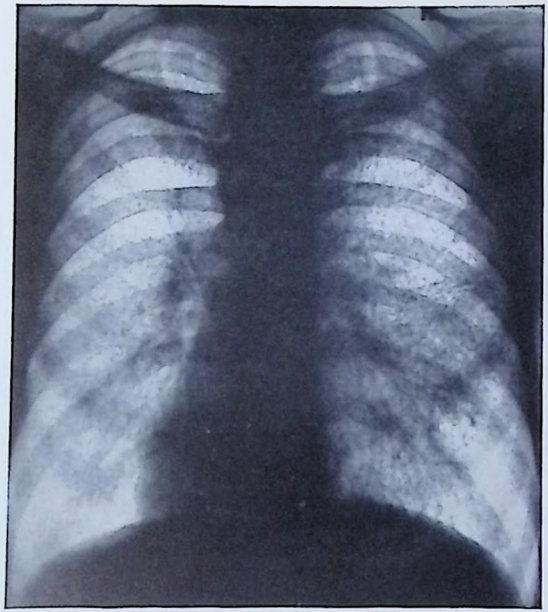


FIG. 16. Roentgenogram of the chest of a male, aged 58 years and a coal miner for 40 years. There is evidence of diffuse nodulation evenly distributed throughout both lungs. Diaphragmatic excursion was good. There is a moderate amount of emphysema. (*Courtesy of Drs. Pancoast and Pendergrass* [136] *and J. Indust. Hyg.*)

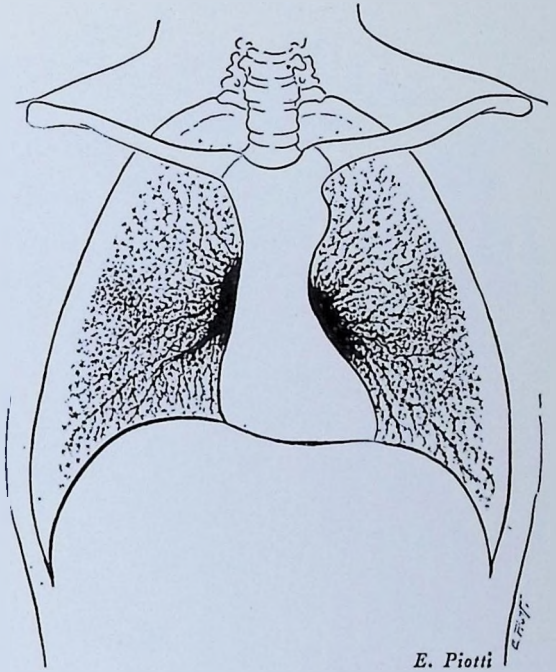


FIG. 17. Line drawing illustrating nodulation with beginning consolidation.

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Roentgenological Appearances	*Histological Appearances*
a. Localized increased transparency of the lung with loss of fine detail.	or less hyaline fibrous tissue. The lung architecture is partially obscured. No demonstrable evidence of infection. Emphysema is a compensatory dilatation of the air spaces with or without thickening of the septa.
b. Intensification of the trunk shadows by contrast.	
c. Depression of the domes with possible tendency toward individualization of the costal components of the diaphragm.	
d. Lateral view. Increase in the pre-aortic and retrocardiac spaces with exaggerated forward bowing of the sternum and backward curving of the spine. Widening of the spaces between the ribs may or may not be present.	

Silicosis with Infection

The characteristic appearances described under simple silicosis are modified by infection as follows:

Roentgenological Appearances	*Histological Appearances*
6. *Localized discrete densities* and/or string-like shadows accompanying those of simple silicosis described above.	6. Strands of fibrous tissue, often along trunks and septa, with or without areas of calcification; indicative of "healed" infection.
7. *Mottling*—shadows varying in size, with ill-defined	7. *a.*) Areas of bronchopneumonia with or

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Roentgenological Appearances	*Histological Appearances*
borders and lacking uniformity in density and distribution accompanying simple silicosis.	without caseation. (Acute infection) *b.*) Lobular areas of proliferative reaction with or without caseation. (Subacute or chronic infection)
8. *Soft nodulation*—the nodular shadows described under simple silicosis (4) have now assumed fuzzy borders and/or irregularities in distribution. This change may or may not accompany 7.	8. Perinodular cellular reaction either exudative or proliferative in character.
9. *Massive shadows* of homogeneous density not of pleural origin symmetrically or asymmetrically distributed.	9. Extensive areas of fibrosis probably due to organized pneumonia of tuberculous or non-tuberculous origin superimposed upon a coexistant silicotic process. Outlines of normal structures may be partially destroyed.

It will be noted that the appearances in numbers 1, 2 and 3 are not considered as definite signs of silicosis, since there is a large number of pathological conditions which give similar pictures. Pancoast and Pendergrass [136] list these as follows (Figs. 18, 19):

1. Passive congestion of the lungs as a result of cardiac decompensation.

2. Passive congestion associated with coronary thrombosis.
3. Advanced bilateral bronchiectasis.
4. Asthma.
5. Infiltrating or permeating malignant disease.
6. Polycythemia or erythremia.

They further point out that:

Number 4 is characteristic of silicosis but must be differentiated from mycotic infections, miliary tuberculosis, and tuberculous broncho-pneumonia. It must also be differentiated from nodular metastatic malignant conditions of the lungs.

Number 5 must be differentiated from various forms of pulmonary tuberculosis.

It will be seen from this that the reading of a chest film involving the question of silicosis is confusing and difficult especially when tuberculosis or other infection is added to complicate the picture. It is in these cases that the x-ray appearance must be checked by a careful phys-

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ical examination and combined with laboratory findings before an accurate diagnosis can be made.

Gross Pathology of Simple Silicosis

The first effect noticeable in the affected lungs is an irregular exaggeration of the linear markings with an increased root shadow. This is owing to the proliferation of cellular connective tissue about the lymphatic trunks, in the walls of the vessels and of the bronchi. Beading is also noted along these markings owing to microscopic areas of fibrosis in the lymphoid tissues along the trunks. The increase of the root shadow is due to microscopic fibrosis in the tracheo-bronchial lymph nodes with extensions along afferent lymph vessels. This preliminary reaction slowly progresses into nodulation with or without a diffuse interstitial fibrosis. The connective tissue nodules invade the middle and lower parts of the parenchyma of the lung. Occasionally some of these nodules may show microscopic foci of central necrosis (Fig. 20).

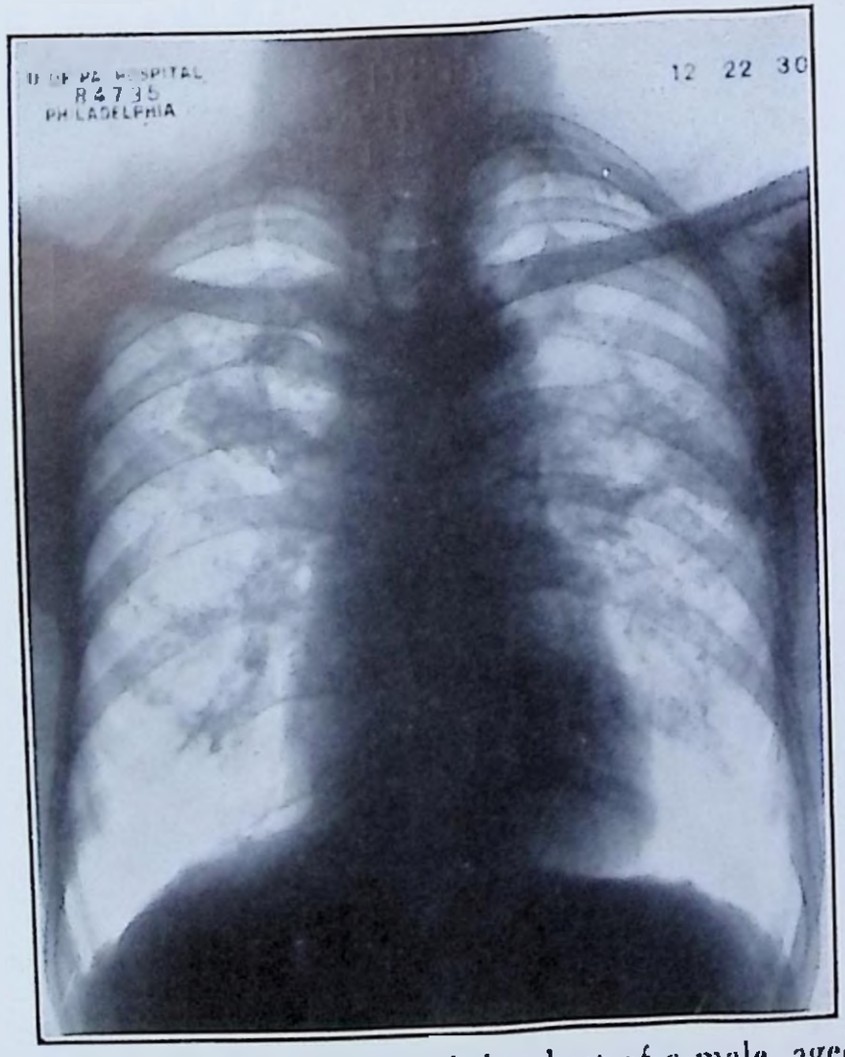


FIG. 18. Roentgenogram of the chest of a male, aged 49 years, who 18 or 20 years ago had worked for 15 years as a rock driller in a sulfur mine. As he has not worked in a dusty occupation since, we believe the condition has been progressive. The condition seen is a diffuse one resulting from a chronic interstitial type of pneumoconiosis. The appearance is that often seen in a case of chronic fibroid tuberculosis without cavitation. (*Courtesy of Drs. Pancoast and Pendergrass and J. Indust. Hyg.*)

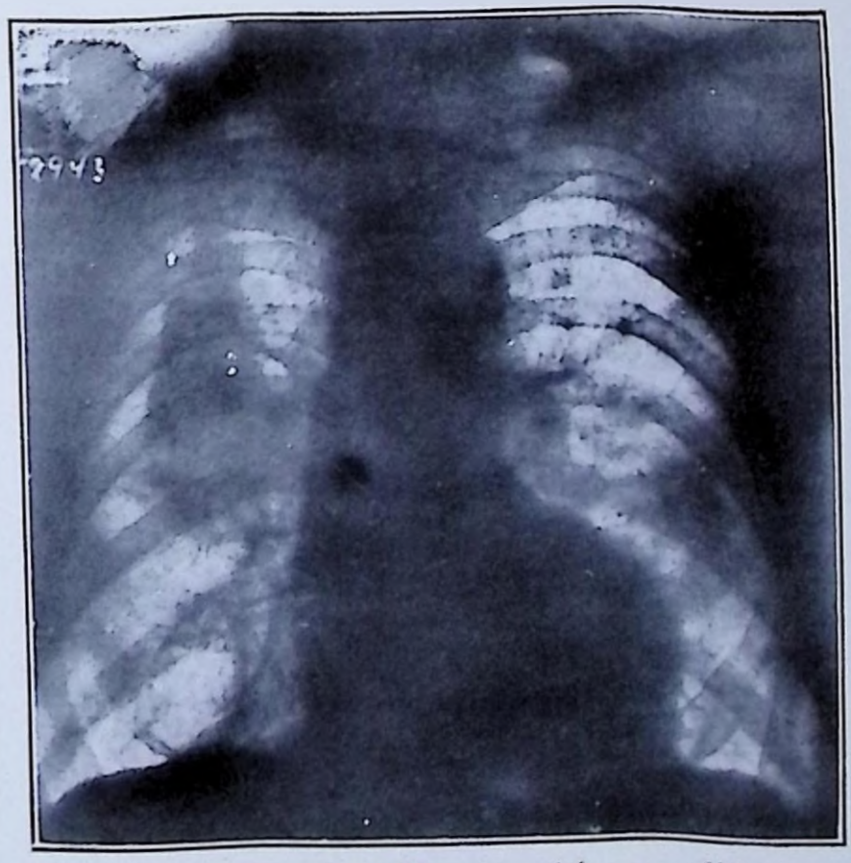


FIG. 19. Advanced silicosis with consolidation. (*Courtesy of Massachusetts Division of Occupational Hygiene, Department of Labor and Industries.*)

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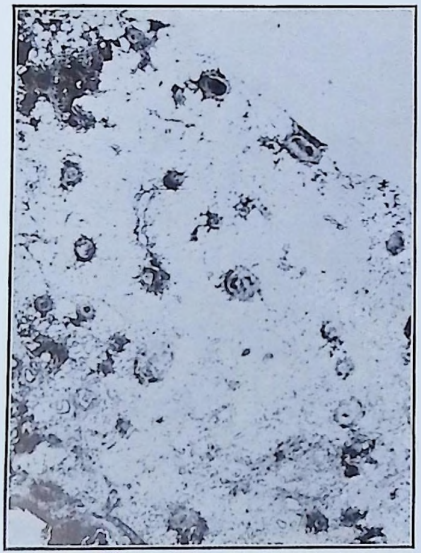


FIG. 20. Silicosis. Isolated silicotic nodules above, fine nodules below. (*Courtesy of Dr. Gardner and the U. S. Public Health Service* [145].)



FIG. 21. Silico-tuberculosis, Barre granite cutter. Diffuse and nodular fibrosis involving lung and pleura. (*Courtesy Dr. Gardner and the U. S. Public Health Service* [145].)

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The pleura is thickened and may show adhesions to the chest wall or diaphragm. As these nodules increase, a coalescence takes place which blocks off large areas, the intervening lung tissue being replaced by more or less hyaline fibrous tissue (Fig. 21). The parts of the lung which are not affected develop a compensatory emphysema.

Accompanying the later stages there may be hypertrophy of the heart and generalized arterio-sclerosis. Occasionally minute fibrous nodules appear in the liver and spleen, but this development is rare.

Pathology of Silicosis with Infection

Simple silicosis in the advanced stage is rare. The toxic effect of the silica dust and the accompanying fibrosis appear to render the lung peculiarly susceptible to respiratory disease of all types, but especially to tuberculosis. Thus in a recent survey by the Special Industrial Disease Commission in Massachusetts [112], 7.6 per cent of the granite workers examined were found to have silicosis and tuberculosis, while

it was noted that foundry workers were particularly susceptible to pneumonia, "about one fourth of all foundry men being found to succumb to this disease."

Tuberculosis may result from the activation of an old quiescent process, or it may be an infection superimposed upon the silicotic fibrosis. Thus according to Kettle [98] "harmful dusts if inhaled into the lungs may excite to activity a latent tuberculosis infection, they may exaggerate an active tuberculosis lesion or a coincident infection; and they may render the lung less able to cope with a superimposed infection."

The pathology resembles that of fibroid phthisis, the middle and basal areas of the lungs are more frequently involved and tubercular broncho-pneumonia more common. Cavity formation may occur and the whole picture resemble that of an advanced case of pulmonary tuberculosis [28]. Tubercle bacilli are usually not plentiful but may be found in the centers of areas of necrosis (Fig. 22). At any time there may, through a breaking down of a necrotic area containing tubercle bacilli, be a rapid spread to

other parts of the lung. The course of silicosis with a secondary tuberculosis is, however, usually slow, and "without the usual symptoms of intoxication and may be carried for years without serious impairment of working capacity" [147].

Respiratory infections other than tuberculosis are frequent. "On the Rand pneumonia is the most common cause of disability and death among native laborers." Proske and Sayers [139] have confirmed Cummings' discovery of fuso-spirochetal organisms as a cause of infection among miners in Picher. Of the pneumonias, broncho-pneumonia is the more frequent, lobar-pneumonia being relatively rare.

Asbestos and Asbestosis

The commercial article known as asbestos is not a distinct mineral but is a name applied to any mineral which can be separated readily into more or less flexible fibers. The most important variety is that obtained from chrysotile ($H_4Mg_3Si_2O_9$) which is mined extensively in Canada.

Sufficient exposure to dust of asbestos in any

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stage of its processing may cause asbestosis. It should be noted that there is no free silica in asbestos although the total silica in pure chrysotile is 44.1 per cent.

Symptoms and Physical Signs of Asbestosis

The symptoms and physical signs of uncomplicated asbestosis are suggested by the pathology. After 5 to 10 years' exposure, the patient usually complains of slight dyspnea and a cough which is dry and annoying. The expectoration if present is scanty. As the condition advances the dyspnea becomes more marked and may appear on the slightest exertion. With the dyspnea are pains in the chest. There is rapid loss of weight going on to emaciation. Cyanosis is frequently present and the complexion is "earthy." Inspection shows an emphysematous type of chest with poor nutrition. Expansion is limited frequently to less than 1 inch. The percussion note all over the chest is one of dull tympany reaching dullness at the bases. On aus-

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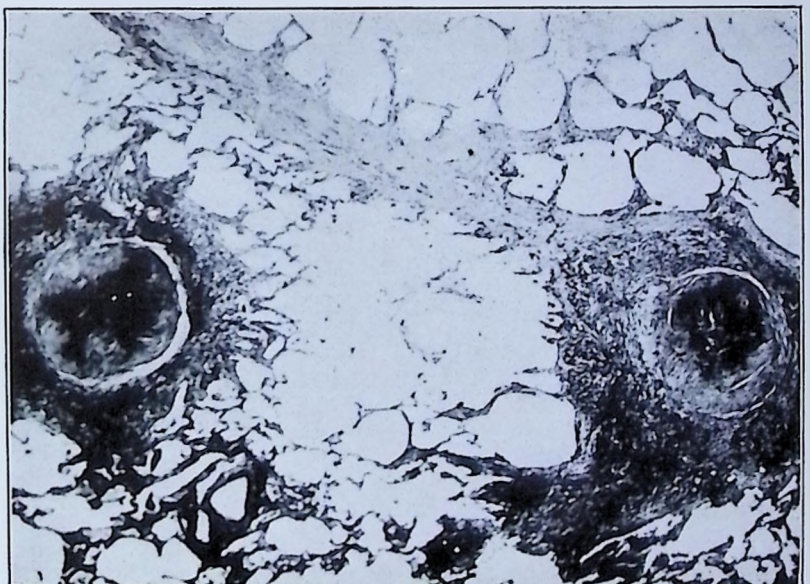


FIG. 22. Barre granite cutter. Two isolated silicotic nodules. Cellular connective tissue borders and hyaline fibrous centers. Thickened interlobular septum extending upward to the left of the right nodule. Note dilated lymph vessels in septum. (*Courtesy of Dr. Gardener and U. S. Public Health Service* [145].)

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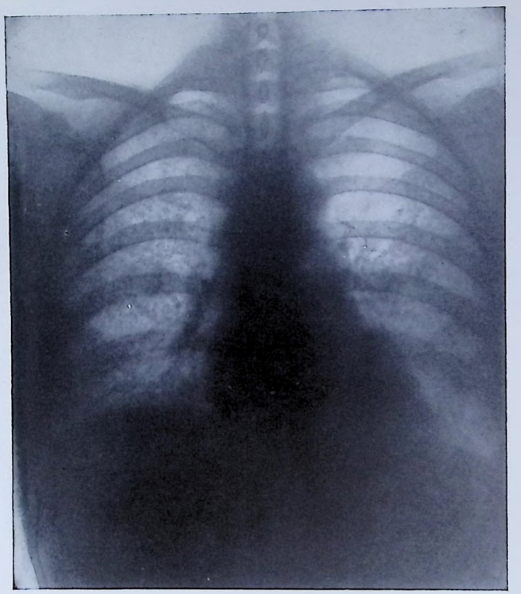


FIG. 23. Roentgenogram of female. Advanced asbestosis, involving mid-zones of both lungs, tubercle bacilli absent, asbestosis bodies in sputum. (*Courtesy of Dr. Ellman and J. Indust. Hyg.* [48].)

cultation fine, dry, crackling notes are usually heard over the lung bases posteriorly, and in the axillary regions.

There may be a fine pleural friction rub. The sputum is seldom streaked, hemoptysis is rare, and asbestosis bodies may be found. The x-ray examination shows a diffuse fine fibrosis with pleural thickening affecting the lower two-thirds of the lung. The diaphragm may appear irregular and the costophrenic angles are clouded. The film has a hazy appearance resembling ground-glass rather than showing the nodulation and trunk enlargement of silicosis. Tuberculosis may or may not be a complicating feature.

The x-ray appearance of asbestosis (Fig. 23) is quite different from that of silicosis [48]. Here the outstanding features are a faint homogeneous interstitial haze over the lower lobes, combined with interlobular pleural thickening and an obliteration of the costophrenic angles. The lower lung areas have a "ground-glass" look owing to the fine interstitial fibrosis charac-

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teristic of the disease. There is usually little perivascular, peribronchial lymph node predominance.

In asbestosis the symptoms appear to be far in excess of the pathology shown by x-ray.

Pathology of Asbestosis

The reaction of the dust cells to asbestos dust is quite different from the cellular response to silica. Instead of migrating into the lymphatics, the cells cluster about the neck of the alveolus. As a reaction, a ring of connective tissue is formed and this in turn, by its contraction, closes the neck of the alveolus. The result is atelectasis of large parts of the lungs, developing into a widely disseminated interstitial fibrosis commencing at the bases. There is no transportation of the dust cells by the lymphatics and therefore no nodulation. Curious elongated, dumb-bell shaped bodies are found in the fibrosed portions of the lung. These were first described by Cooke [31], are known as asbestosis bodies (Fig. 24), and are pathognomonic of asbestosis, though bodies of analagous appearance are seen after exposure to other dusts [169].

These bodies differ in size and shape, varying in length from 20 to over 200μ and presenting a golden yellow elongated body with a bulbous enlargement at one or both ends. The core of the elongated body is generally the mineral chrysotile [8]. The colloidal golden yellow material is probably iron silicate [58].

While a secondary tubercular infection is not uncommon in asbestosis, it is far less frequent as a complication than in silicosis. The gross pathology is that of a massive fine interstitial fibrosis of both lungs, beginning at the bases and gradually involving the whole of both lungs. The apices are last affected and usually present a compensatory emphysema. The pleura is thickened and there may be adhesions to the chest wall and to the diaphragm. The heart is usually normal in size but it may be displaced if one side of the chest is more involved by fibrosis than the other.

Diagnosis of Silicosis and Asbestosis

Until complicating tuberculosis or some other respiratory disease intervenes the diagnosis of silicosis and asbestosis is made by x-ray examina-

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tion, the dyspnea and cough with the history of exposure being the indication for such an examination.

The x-ray should be taken stereoscopically for anterior-posterior films. Flat lateral, antero-lateral and postero-lateral films may also be of great assistance as pointed out by Pancoast and Pendergrass [135]. In examining films the important points for study are the thoracic cage, the tracheal position, the hilum, heart and blood vessels, diaphragm, and lungs. The examination must be made by a person having considerable experience in lung pathology and in reading x-rays as there are many conditions which simulate silicosis, especially in its earlier stages.

The diagnosis by x-ray of silico-tuberculosis as opposed to uncomplicated tuberculosis is particularly difficult. Pancoast and Pendergrass [136] classify these cases into six groups, based on occupational history and upon x-ray appearance.

1. Cases showing x-ray evidence of tuberculosis only, in spite of history of dust exposure.

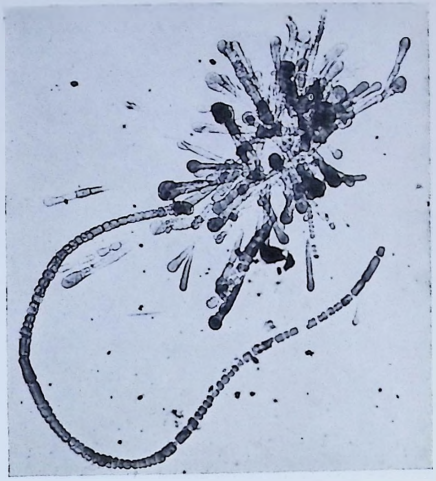


FIG. 24. Asbestosis bodies in sputum. (*Courtesy of Dr. Ellman* [45] *and J. Indust. Hyg.*)

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2. Cases showing obvious pulmonary tuberculosis with mild silicosis or pneumoconiosis.

   (Pancoast believes that these two groups should not be included in the x-ray diagnosis of silico-tuberculosis.)

3. Tuberculosis associated with silicosis (interstitial) of short duration but high exposure as sand pulverizers.
4. Obvious advanced tuberculosis with no evidence of silicosis because obscured by the extent of the tubercular infection (study of environment essential here).
5. Obvious tuberculosis with evidence of advanced silicosis.
6. Advanced silicosis in which there is doubt as to co-existence of tuberculosis.
7. Obvious tuberculosis occurring late in individuals with progressive and marked silicosis which has appeared many years after exposure to dust has ceased.

Because of its complicated picture, careful physical examination to determine areas of activity

is necessary before a diagnosis of active tuberculosis complicating the silicosis can be made. The x-ray may indicate tuberculosis but its activity can only be determined by the examining physician. Tubercle bacilli are difficult to find in many cases of silico-tuberculosis and the physician must depend upon the industrial history, the amount and the quality of dust exposure, physical examination (including afternoon temperature), loss of weight, and other signs of pulmonary tuberculosis, before he can make an accurate diagnosis.

The diagnosis of asbestosis likewise depends upon a combination of history, symptoms, physical signs, and the x-ray. The picture of a case having acute dyspnea, dry cough, no temperature, coupled with an x-ray showing pneumoconiosis of interstitial character invading the lower chest with some obliteration of the costophrenic angles is most suggestive. With asbestosis bodies in the sputum the diagnosis is complete. While active tuberculosis may complicate asbestosis, it is much less frequent than

in silicosis. Early asbestosis must be differentiated from acute emphysema.

Treatment of Silicosis and Asbestosis

The treatment of both of these conditions is preventive and symptomatic.

Early removal of the affected worker from the inciting cause of his disease is imperative. When the disease has progressed beyond the early stages, removal is useless. The fibrosis continues to build up whether the worker is exposed or not and eventually leads to incapacity. For this reason unless the silicotic worker has signs of respiratory infection, tubercular or otherwise, it is inadvisable to remove him from his work after nodular fibrosis is present. The man will gain nothing and will lose several additional years of productive work at tasks with which he is familiar and at which his earning capacity is highest. The same may be applied to asbestosis.

In the recent report of the Special Industrial Disease Commission of Massachusetts it was

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recommended that "no finding of physical unfitness shall be made except in evidence of active pulmonary tuberculosis." In the Picher Clinic, held in connection with the Picher mines, workers are not barred from employment unless they are infected.

CHAPTER VII

PNEUMOCONIOSIS (continued)

NON-DISABLING FIBROSES

THERE ARE NUMEROUS industrial processes today in which men are exposed to considerable dust concentrations seemingly without harm. Industry is at last becoming alert to the possible ill effects of dust inhalation so that we are able now to take some account of stock and to state with some assurance what dusts produce disabling fibroses and what do not.

Carbon dusts. Perhaps the commonest particulate impurity breathed is carbon. It occurs in air as unburned anthracite and bituminous coal dust and is an important solid constituent of black smoke from any carbonaceous fuel. Compared with industrial exposures the carbon dust concentrations met in ordinary life are negligi-

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ble. The lungs of city dwellers are, however, invariably blacker than those of persons who have lived in clean country air. Also silicotic lungs are generally blackened as if from carbon —hence the early term, anthracosis.

Studies by Collis and Gilchrist [29] indicated that a non-disabling fibrosis was experienced by men with a history of prolonged exposure to coal dust breathed in "trimming coal" in bunkers and in cargo holds. Hall and others [67] recently confirmed this view but none of these authors give any idea of the dust concentrations actually breathed. All of them emphasize the fact that quartz in any but trifling impurities is absent.

It is of interest that Gardner and Cummings [57] found the dust of the diamond, which is pure carbon and the hardest substance known, to be without injurious effect upon tissue. On the basis of his earlier work with Mavrogordato [114], Haldane [66] suggested that the Rand gold mines might be worked dry with the aid of dust traps and carbon dusts actually blown into the mine workings. Carbon, according to

Haldane, so increased phagocytosis that it might thereby reduce the potency of quartz and thus give a simple and effective preventive for silicosis.

Iron dusts. Exposures to excessive amounts of iron dusts are limited mainly to the mining and handling of hematite, the commonest iron ore. Chemically, hematite is Fe_2O_3 and is reddish in color. Zenker [174] described and illustrated the effects of breathing iron dust and gave to the condition produced the name siderosis. Since then several papers have appeared on the effects of breathing hematite dusts but nothing to show that the pure substance produces disabling fibrosis in the manner of silica [161]. As in coal mining, the rock mined is by no means pure hematite, but contains substances like free silica in varying amounts.

There is some evidence that iron oxide dusts not only are harmless of themselves but even may reduce the potency of free silica. It seems likely that work now in progress will settle this point within the next few years.

Calcium and magnesium carbonates. Calcium

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carbonate ($CaCO_3$) or calcite is the chief constituent of limestone rock, while marble and chalk contain calcium carbonate in percentages varying from 100 down to about 90. Few minerals have wider use in industry today and men have been exposed to dense clouds of these dusts for many years.

In the same mineral family belongs magnesite ($MgCO_3$) which is the source of all magnesium salts, and when calcined is made into magnesia bricks used for furnace linings, insulations, and the like.

The mineral dolomite ($CaCO_3 . MgCO_3$) is a common constituent of the southeastern Missouri rocks which contain lead. Certain varieties of dolomite are used as building stone.

Exposure to these calcium and magnesium carbonates has never shown the slightest evidence of disabling fibrosis. Laboratory [56] and practical [35] evidence all tends to show that an important reason these dusts are not harmful is their high solubility in water and in body fluids.

Clay. This word covers several minerals, chief

of which is kaolinite ($H_4Al_2Si_2O_9$). Commercially useful deposits are common in eastern United States. In the processing of clay and using it in ceramics excessive dustiness is very common. Carleton [20] and Kettle [97] are by no means sure that excessive amounts of clay dust can be breathed with impunity while Jones [97] has pointed out that sericite or quartz-free clay is unusual.

Talc. Large deposits of talc are mined, milled, and used in the United States. This mineral is $H_2Mg_3(SiO_3)_4$ and often occurs with or near asbestos ($H_4Mg_3Si_2O_9$) which might be said to be its near geological relative. Talc in the powdered form is used widely as a mineral filler and in the solid form for sinks, table tops, and the like. Exposures to dense clouds of talc dust are very common. In view of its close relationship to asbestos talc may be looked upon with suspicion but in its defense is the fact that talc dust exposures are by no means new. There are no especially incriminating data against talc at this time [34] but an industry with excessive talc

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dust exposure could ill afford to neglect precautionary measures.

Gypsum. This mineral ($CaSO_4.2H_2O$) is used in enormous quantities all over the world, especially in the form of the partly calcined material which is the essential constituent of plaster. Because of the affinity of the partly calcined material for water, one might expect the inhaled dust to be irritating but such apparently is not the case. Riddell [142] gives gypsum a rather clean bill of health.

Artificial Abrasives. The artificial abrasives, silicon carbide (SiC), and aluminium oxide (Al_2O_3), are products of the electric furnace and are used widely throughout the world. Their principal use is in wheels for tool sharpening, precision grinding, polishing, and snagging of metals and other substances, as well as a large number of other factory processes. They have supplemented the natural abrasives and are in very general use. Both materials are harder than quartz and are very insoluble. Of the common materials they are exceeded only by the diamond in hardness.

On experimental animals Gardner showed [55] [57] that neither of these materials produce a disabling fibrosis and that aluminium oxide does not compare with quartz in reactivating a healed tubercular lesion. Carbide of silicon did show an activating action. In a series of three papers covering 20 years' experience, Clark [26] has shown that men exposed to these dusts do not develop incapacity for work and that no undue amount of tuberculosis developed in a working force exposed continually to varying amounts of these dusts, the amount in most cases being greater than occurs in commercial use of these products (Fig. 25).

Pathology

The pneumoconioses of inactive dusts resemble the reaction of tissues to a foreign body. There is first an enlargement of the lymphatics along the broncho-arterial tree with increase in size of the tracheal lymph nodes. This peribronchial, perivascular, lymph node proliferation is slow. Later when there has been exposure for many years to large quantities of dust there

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may be in addition a moderate amount of interstitial thickening with occasional very small nodules. The pleura may be thickened and there may be diaphragmatic adhesions. Complicating respiratory disease occurs more frequently than in normal individuals in some types of dust, but the mortality does not seem greater than usual for the complicating disease. The danger of tuberculosis does not seem much greater than for the community at large. In the case of some dusts, as soft coal and marble, it seems to be less than for the community.

In discussing the differing effects of silica dust and the non-siliceous dusts upon the human being a recent note in the *Journal of the American Medical Association* [93] summarizes as follows:

"Only silica (SiO_2) is capable of inducing silicosis but any other mineral dust under conditions of prolonged and gross exposure may cause some increase in pulmonary fibrosis. If the relative potential harm of silica is rated as 100, these other non-toxic mineral dusts may be rated only on the order of 5 or 10. Moreover, the

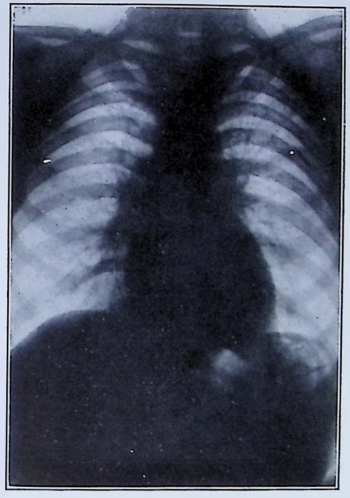


FIG. 25. Pneumoconiosis caused by artificial abrasives, non-disabling. Nineteen years' exposure. Man well and working.

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fibrosis is in itself not pathognomonic, since many other dusts, alkalies, acids, or vapors may induce somewhat similar if not identical x-ray markings."

Symptoms

The pneumoconioses of inert dusts produce no symptoms. Workers exposed to these dusts continue to work into old age. If exposure has been long and dust dosage abnormally heavy, there may be some dyspnea on moderate working exertion. This is usually not severe enough to interfere with the worker's normal activities or working capacity, nor is there evidence that these dusts, *free from quartz,* predispose to tuberculosis.

Physical Examination

The physical examination of those exposed to inert dusts is usually negative. In a few cases where very large quantities of dust have been inhaled for a long period of time there may be some restriction of chest expansion and slight signs of emphysema. It is not uncommon to find

men with long employment in moderately dusty jobs, in which the quartz dust content is negligible, showing perceptible x-ray changes but with no indication whatever of impaired physical functions (Fig. 25).

Chapter VIII

PNEUMOCONIOSIS (continued)

Functional Disability in Pneumoconiosis

The classical symptom of advanced pneumoconiosis, simple or infective, is dyspnea. But dyspnea from wholly different causes is rather common. Its extent may be gauged most simply by observing the vital capacity. Attempts in the past to correlate vital capacity with the degree of lung fibrosis have not given results of practical use in the diagnosis of early pneumoconiosis or in the estimation of questionable disability. A significant reduction in vital capacity, in pneumoconiosis, generally indicates a condition of fibrosis which can be seen readily by x-ray.

We need badly a technic which will be usable

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by compensation boards and medical examiners for estimating disability both before and after positive diagnoses of lung fibrosis are possible radiologically. This problem has received intensive study recently at the hands of McCann [115], Hurtado [85] and their colleagues. They have elaborated upon the technics developed by Christie [23] for studying lung volume and its subdivisions, and in addition they have devised several new technics applicable particularly to studying pneumoconioses.

Christie gives some simple definitions of lung volume and its subdivisions which are worth quoting:

Residual air is the amount remaining in the lungs after the fullest possible expiration.

Vital capacity is the amount of air that can be expired from the fullest inspiration to the fullest expiration.

Total capacity is the sum of residual air and vital capacity.

A normal healthy man of 150 lbs. and average height has a vital capacity of about 4500 cc. and takes an average breath at rest of about 500 cc. He uses, thus, about one-ninth of his available

lung capacity or about one-eleventh of his total capacity. In moderate exercise his pulmonary ventilation will increase two or three times that at rest, while in violent exercise the increase is still greater.

McCann determines vital capacity in the usual manner, a procedure which is commonplace in making physical examinations, but Hurtado has shown that vital capacity can be computed reliably from the area of the lung fields at maximum inspiration. This area is measured on the x-ray of the chest by means of a planimeter and can be done by any technician.

Taking the *vital capacity* as 78 per cent of the *total capacity,* one obtains, without gas analyses, the three lung volume measurements which McCann and Hurtado have shown to be of importance in estimating functional disability.

In interpreting the results of these simple measurements Hurtado points out that "the degree of respiratory disability has been found to be closely correlated with the ratio

$$\frac{\text{Residual air}}{\text{total capacity}} \times 100$$

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The higher this ratio, the more pronounced is the failure of respiratory adaptation to physical activity." McCann [115] adds that the higher the ratio the greater the tendency to anoxemia which, in turn, is gauged by the degree of oxygen saturation of the arterial blood.

In our opinion McCann's work promises to fill a significant and bothersome gap in the present methods now being used in examining patients with pneumoconiosis.

Determining the Comparative Harmfulness of Dusts

From the foregoing discussion it is clear that the cytocidal effects of dust are of prime importance in silicosis. Haldane and Mavrogordato both believe that a criterion of dust potency is the readiness with which the dust is removed from the body, since only those which are especially resistant to removal can do harm. Therefore the dust must be relatively insoluble and phagocytosed with difficulty—those easily phagocytosed being less dangerous. Fenn [51] found, for instance, that carbon was more easily

phagocytosed than quartz, indicating that the latter was more dangerous. Haldane, as we have pointed out, carried this idea so far as to suggest that the South African gold mines be carbon dusted as an antidote against the quartz, but the suggestion has apparently not received a practical trial.

Miller and Sayers [122] are studying the comparative effects of dusts injected into the peritoneal cavity of guinea pigs and looking particularly for signs of tissue proliferation as an index of dust potency.

Stüber [162] and C. K. Drinker [37] have described a technic for injecting dusts into the lymph circulation and then studying the dust cell necrology from biopsy specimens. The technic is more difficult than Sayers', but the results are obtained in a few days. In Drinker's preparations one follows the actual destruction of the dust cells by the dust particles.

The direct dusting of animals as carried out by Gardner, Mavrogordato, Carleton, Jötten, and others is slower but the results are more convincing. In criticism of animal dusting is the

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inevitable need of using as dense clouds as can be borne by the animals in order to shorten the time required for producing silicotic pathology. At best it takes a long time to carry out the direct dusting experiments and the expense is considerable.

The ash of dusted lungs. Analyses of the ash of lungs taken from fatal cases of pneumoconiosis have been published by McCrae [119], Riddell and Rothwell [143], Pancoast and Pendergrass [133], Sladden [156], Badham and Taylor [6], Jones [90], Beger [8], and Fowweather [52].

Fowweather suggests that "the materials which we should expect to be the most potent as silicosis producers would be the soluble alkali silicates. . . . " Jones makes a very strong and eloquent case for his sericite theory in the presentation of a few lung analyses, while Badham and Taylor add welcome analytical data but do not interpret them. Riddell and Rothwell's data indicated that a normal total silica content in the ash of dried lung was 2 to 3 per cent or less while severe cases of silicosis showed as much as 39

per cent total silica. Subsequent work is not, in our opinion, contradictory to the data given by Riddell and Rothwell but shows rather that the mineral content (especially free silica) of dusted lungs will vary greatly with the nature of the occupation—and particularly with the kind and duration of dust exposure.

As pointed out previously fatal silicosis uncomplicated by tuberculosis is very rare. Silicosis with superimposed tuberculosis may result in a fatality in which the victim's lungs are not especially high in silica. A high free silica content, however, in any autopsied lung can only have been caused by silica dust inhalation. The evidence is overwhelming that with heavy dusting, by quartz, rapidly developing microscopic nodules of silicotic tissue are to be expected and fatal tuberculosis is very likely. Fowweather states that "there is no obvious connection between the amount of silica in the lungs and the amount of fibrosis which is present."

In this we agree and have yet to see any convincing data from lung analyses on the basis of which one might predict or forecast the likeli-

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hood of silicosis or asbestosis from a specific working environment. Furthermore, the analyses cited show very convincingly that a positive diagnosis of silicosis, for example, cannot be made simply on the basis of the mineral content of lungs.

The Solubility of Dusts

In searching for an explanation of the peculiar action of free silica upon dust cells and especially for one of the exceptionally poisonous nature of colloidal silica [64], advantage has been taken of the brief paper by Myers [125] on the solubility of quartz dust in water, alcohol, and kerosene. Myers found some slight quartz solubility, to be sure, but he neither made nor implied some of the sweeping generalities since attributed to him. There has been no real proof that the solubility of quartz, in the biochemical sense, is the basis of the specific cellular necrosis which quartz causes.

On the other hand, Gardner and Dworski [56] showed biologically that marble dust ($CaCO_3$) was very soluble and was removed

from the lung tissue rapidly. Titus [165] recently has shown that dolomite dust ($CaCO_3$. $MgCO_3$) is about thirty times more soluble than quartz and if the solvent (water) is saturated with carbon dioxide gas, the solubility increases to more than seventy times that of quartz. Dusts like marble and dolomite apparently do not cause disabling fibroses.

To the geologist quartz is one of the most insoluble of minerals. The geologist doubts the solubility explanation of the harmful action of the inhaled quartz and, from the evidence available, he cannot be blamed. If solubility is the explanation rather than some surface phenomenon a chemical or physico-chemical proof of its validity should be forthcoming. Also, if quartz produces its effect by virtue of its trifling solubility, why is it not toxic in larger particle sizes, such as 10 to 20μ?

Permissible Dustiness

There have appeared in the literature over the last 20 years various suggested figures for permissible dustiness in various industries—figures

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which the engineer welcomes as an objective of cleanliness for which to strive. Some of these figures are founded upon reliable data and some are frankly estimates.

Legge and Duckering [104], [46] suggested 0.5 mg. per cu.m. for lead dust which Russell and his colleagues [146] lowered to 0.15 mg. for litharge indicating that this figure might be scaled up or down with changes in the solubility of the lead compound in serum.

The South African authorities, for their high quartz rock in the Witwatersrand gold mines, have gradually arrived at a figure of about 1 mg. per cu.m. and silicosis, under their conditions, has not been eliminated (although greatly reduced). Higgins, Lanza, Laney and Rice [81] suggested 10 mgs. per cu.m. for the high quartz rock of the Joplin, Missouri, zinc and lead district. In both of these examples the standards suggested are both practicable and attainable.

Russell et al [145] in their granite study in Barre, Vermont, suggested 9 to 20 million particles per cubic foot by the impinger sampling

technic and light field counting. Here the dust contained about 35 per cent quartz. In sandstone working, where there is a high percentage of silicates, Badham, in Australia [5] advocated 200 particles per cubic centimeter by Owens' jet dust counter, but hinted that 100 would be better. In Great Britain, perhaps wisely, no figures for permissible dustiness in connection with silicosis have been suggested.

In their cement dust study Thompson and his associates [164] investigated one of the most harmless inorganic dusts of modern industry. The effects upon the chests of workers with abundant dust exposure were in no way comparable to the chests of the granite workers depicted by Russell. Nonetheless, Thompson advocated 10 to 20 million particles per cubic foot as compared with 9 to 20 million for granite.

In the case of asbestos dust no figures have as yet been suggested but on the basis of abundant experience McConnell [116] of the Metropolitan Life Insurance prefers to see the dust count below 5 million and welcomes still

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lower figures. Merewether [120] gives figures on asbestos dust concentrations but no indication of permissible dustiness.

In studying metal fume fever caused by breathing freshly formed zinc oxide (Fig. 10), Drinker, Thomson, and Finn [43] found that concentrations of 14 mgs. of zinc per cu.m. for 8 hour exposures or 45 mgs. for short exposure could be tolerated without causing the "shakes" to the average worker. Drinker believed that figures could easily be obtained for other metallic fumes but the required experiments have never been done. Prodan [138] found that cadmium oxide fume, which is close to zinc oxide metallurgically, was far too toxic to permit breathing measurable amounts.

To insure the avoidance of trouble from chromic acid mist Bloomfield and Blum [15] suggest that continuous daily exposures be kept below 1 mg. per cubic meter and show in their report that this figure is readily attainable by proper transverse ventilation.

Cummings [32] sees no need to require the manufacturer of materials with well established

harmless effects to maintain air as clean as that demanded in granite cutting. However, there is no longer any excuse for excessive dustiness in any process—it merely represents slovenly practice. A few years ago it was not uncommon to see workmen employed in an atmosphere so thick with dust that a 50 watt lamp was obscured at a distance of 10 feet. In a recent lawsuit a witness testified that he had to sweep up every hour because if he waited longer it was necessary to use a shovel! Industrial physicians and engineers have seen these conditions but they cannot defend them.

The only justifiable conclusion to be drawn from such apparent inconsistencies is that generally we lack data for defining rigidly permissible dustiness. The application of medical and engineering knowledge and common sense to dust control is highly commendable but the point has not been reached where a manufacturer can be told with certainty that his plant will have no silicosis, no asbestosis, or no lead poisoning if he keeps dustiness down to some definite figure. He can be told only that the maintenance of certain

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degrees of air cleanliness represents the best present practice.

The question is asked frequently whether 10 million quartz particles per cubic foot breathed for 8 hours is equivalent to 70 million for 1 hour plus 1 million for 7 hours, the total amount entering the respiratory system in both cases being 80 million. On this point Mavrogordato [113] in discussing his "dust floods" says: "Lesions of silicosis in a mild degree can be produced in an animal by 30 hours' exposure to intense dust clouds, and one is inclined to suspect that it is intermittent exposure to relatively dense clouds that is the deciding factor in producing the disease in susceptible human subjects." All medical experience is in accordance with Mavrogordato's statement and after all, it is only physiological common sense—a subthreshold stimulus for a long time produces no reaction whereas a relatively brief super-

TABLE 6

AVERAGE DUST COUNT IN CERTAIN DUSTY TRADES

Industry	*Dust count in millions of particles per cubic foot of air*
Slate finishing mills	
Floormen	1598.0
Loaders	1276.0
Disc crusher operators	312.8
Talc mining	
Jackhamer drillers	2159.8
Muckers	44.8
Talc finishing mills	
Crushers and cylindermen	14.0
Packers	50.1

Industry	*Dust count in millions of particles per cubic foot of air*
Marble carvers	19.1
Marble cutters	32.8
Granite quarrying	
Leyner drillers	144.4
Jackhamer drillers	112.1
Plug drillers	36.9
Cement mill, average of all operations	26.0
Granite cutting	
Hand pneumatic tool operatives	59.2
Machine pneumatic tool operatives	35.9
Attendant labor	17.0
Anthracite coal mining	
Mining and miners' helpers	231.5
Attendant labor	31.1
Bituminous coal mining	
Coal cutters and coal loaders	112.3
Attendant labor	3.9
Silverware manufacturing	
Dusty processes	5.2
Non-dusty processes	1.7
Municipal dust (street cleaners)	
Congested district	4.1
Residential district	1.8
Cotton industry	
Carding room	8.6
Weaving and spinning room	4.5

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threshold stimulus may cause a reaction. It is not enough to find that the *average* dust concentration is below a desired figure: occasional exposure to excessive dustiness should be avoided.

Industrial Dust Concentrations

Granting the advisability of maintaining the concentrations indicated, it is just as well to know something of the concentrations one actually finds in sampling. In this country Bloomfield [14] of the U. S. Public Health Service, and Fehnel [117] of the Metropolitan Life Insurance Company, have assembled and personally determined dustiness in a large number of industries. Some of Bloomfield's data are given in Table 6. Harrington [72], [73] gives figures of dustiness in mines and the early mining investigations by Higgins et al [81] are still useful.

Chapter IX

LEAD AND METAL FUME FEVER

Lead

Lead in small quantities is being ingested daily by most people. Kehoe [95] has shown that even in isolated non-industrial communities normal individuals excrete a certain amount of lead daily.

Workers in lead or where the process involves the use of lead are exposed to abnormal amounts of the metal and may suffer from lead poisoning. While in the case of exposure to tetraethyl lead, absorption is through the skin [70], in the majority of cases lead is inspired as a dust or fume [71]. Although a small amount of lead may be taken in by mouth from the worker's unwashed fingers, the amount in most cases is too small to be of importance [71].

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When lead fume or lead dust is inhaled in sufficient quantity there is a body reaction. What this sufficient quantity is, is disputed. As little as 2 mg. daily is said to be poisonous [104] [46].

Poisonous dosage occurs when an amount is absorbed by the worker which is slightly above the threshold of excretion. It also depends upon personal idiosyncrasy. It is, therefore, important for the physician to consider the possibility of lead poisoning in patients whose work involves exposure to lead dust or fumes.

Pathology

Lead entering the lungs through the inspired air enters the alveoli and is absorbed through the capillaries surrounding them. On reaching the blood it becomes a dibasic lead phosphate and circulates as such. While circulating it has a specific effect on many organs in the body. A small amount is deposited in the liver but the principle site of deposit is in the long bones [4]. Here the deposit is in the trabeculae and hard parts of the bone, not in the marrow [71] and is greatest in the region of the epiphyses. This is

shown in x-rays of children suffering from lead absorption but may not show in x-rays of adult bones. When lead is ingested in large amounts it is stored in the liver to a much greater extent than when inhaled [11].

While circulating the lead acts on the blood cells and upon the vessel walls. The red cells are rendered more fragile and new forms are induced. Thus we find a reduction of hemoglobin to about 70 per cent and the appearance of poikilocytes and basophilic red cells in the stained smear. The basophilic or stippled cell while it appears in a number of common diseases, when found in any number in the blood of a worker, is strongly suggestive of lead absorption. The number of white cells may not be affected but there may be a leukocytosis of moderate degree, rarely over 20,000 [71]. Legge and Goadby [104] consider a mononucleosis characteristic of plumbism.

The presence of lead in the blood of the capillaries appears to cause microscopic hemorrhages and vaso-constriction [131]. The former may be the cause of the neurological symptoms, the lat-

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ter of the characteristic facies and possibly of abdominal colic.

The effect of lead upon the walls of the larger vessels has been thought to produce the arteriosclerosis frequently found in lead workers [131]. Lead is excreted in the bile and in the urine. Lead poisoning may be expected when lead is excreted at a mean rate above 1.1 mg. per day in the feces and 0.21 mg. per liter of urine [70].

The storage and excretion of lead in the bones follows the calcium metabolism. If the diet is rich in calcium, lead is deposited in the bones. If a low calcium diet is maintained lead is excreted from the bones. This fact is made use of in treating plumbism.

Symptoms

The symptoms of plumbism are usually divided into gastro-intestinal and neurological. The symptoms may be single or multiple and may present a confused picture.

Kessler [96] has found gastro-intestinal symptoms in the following order of frequency:

(*a*) general gastric discomfort,

(*b*) sweetish metallic taste in mouth,

(*c*) loss of appetite,

(*d*) constipation,

(*e*) nausea,

(*f*) vomiting,

(*g*) colic.

Constipation and colic when present are severe, the clinical course being as follows:

1. Constipation with abdominal discomfort increasing and culminating in an intense agonizing pain,
2. Colic—pain spasmodic owing to violent contractions and slight relaxations of the intestinal musculature,
3. Severity—may be severe enough to produce collapse,
4. Site—pain is referred to the umbilical region. The patient doubles up clutching his rigid abdomen. Pressure usually relieves the pain,
5. Position of patient—likely to be lying on his face,
6. Clinical picture—pallor without sweat, thirst, pulse slow, small, and of increased

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tension. Blood pressure usually raised during attack. Respirations are rapid and labored. Nausea is common but vomiting not frequent. Occasionally lancinating pains in the limbs,

7. Duration—a few to 24 hours,
8. Diagnosis—made on
   a. sudden onset in previously healthy man,
   b. history of occupation,
   c. physical and laboratory signs of plumbism,
   d. may be mistaken for intestinal obstruction or acute appendicitis.

The symptoms of nerve involvement include muscular weakness and palsy, the former frequently preceding the latter. Such symptoms are a late manifestation of plumbism.

The theoretical sequence of the effect of lead absorption according to Melliére, quoted by Legge [104] is as follows:

1. Impregnation of the body tissues by lead salts, the nerve tissue being the chief tissue affected,



FIG. 26. Chronic lead poisoning, gouty arthritis and edema of lower extremities of eighteen years' duration. Wrist-drop for two years. (*Courtesy of Dr. Kessler* [96].)

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2. Retardation of the general oxidation changes of the body resulting in malnutrition and general loss of tone,
3. Establishment of intoxication with general affections, paralyses, and in some cases arthralgia about the large joints which is myalgic in character.

Sudden large dosage of lead may precipitate an outburst of symptoms as may a sudden checking of elimination as a result of intercurrent disease or alcohol.

There may be prodromata consisting of lassitude, loss of weight, and weakness, accompanied by a frequent fine intention tremor, but the onset is frequently sudden.

The nerve symptoms appear most often in the upper limbs. The commonest type of paralysis is one affecting the muscles of the hands. There is first diminution of extensor power followed by the onset of paralysis. The first muscles affected are the extensor communis digitorum and the extensor indicis (Fig. 26).

The second group of muscles affected are those

about the shoulder joint, mainly the deltoid. The third group in order of frequency is the peroneal or leg group, in which the peronei muscles and occasionally the interossei of the foot are affected. Legge [104] believes the muscle weakness is owing to the direct toxic action of lead on muscles and this view is supported by the experimental work of Aub [3] who points out that fatigue as well as lead is an important factor. Therefore, one may consider the paralysis as due partly to the effect of lead upon the specific nerve apparently affected, and partly to the effect of the lead upon the weakest group of muscles in frequent use. In considering the paralyses, the Dejerine-Klempke classification is generally used:

1. Anti-brachial, paralysis involving extensors of wrist and fingers, musculo-spiral innervation except that supinator longus not attacked. Important symptom, wrist drop;
2. Brachial type—deltoid, biceps, brachialis anticus, and supinator longus. Important symptom, shoulder and elbow weakness;
3. Aran-Duchenne type—small muscles of the hand involved generally following ulnar innervation. Chief symptom—finger weakness;
4. The peroneal type with involvement of the peroneal muscles, the extensor communis digitorum and extensor of the great toe. Chief symptom—foot drop and steppage gait.

In obscure cases lead poisoning may simulate almost every other disease of the central nervous system [92].

Encephalopathy. Plumbism in some cases produces a variety of mental symptoms. These may be headache, dizziness, insomnia, melancholia, and nervousness or more serious symptoms such as delirium, hallucinations, dementia, and blindness. Women appear to be somewhat more susceptible to plumbism than men and among married lead workers, abortion is fairly frequent.

Physical Findings

The following evidences of lead absorption should be looked for when examining lead workers.

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1. Blue line of gums.
   This is a stippling of the papillae at the gum edge with blue-black spots of lead sulfide. It can be best found by wiping the gums clean and placing a piece of white paper over the teeth to the gum edge, and using a magnifying glass. It is present in only a small proportion of cases of plumbism.
2. Anemic appearance.
   Cases of plumbism have a characteristic pallor, described as ashen gray, involving especially the lower half of the face. It is probably due to vascular spasm, not to changes in the blood.
3. Neurological changes.
   Weakness of extensor muscles of the fingers may be detected by extension against pressure. "The earliest sign of wrist drop is the dropping of the two middle fingers when the hands are stretched out, with fingers separated and extended" [70]. Other neurological findings consist in weakness of the groups of muscles involved.

4. Laboratory findings.
   Blood: The number of red cells and percentage of hemoglobin is not significant of plumbism. A finding of polychromatophilia and anisocytosis accompanied by marked basophilia as evidenced by stippled red blood cells is of definite significance. The presence of symptoms of plumbism is often related to the extent of basophilia present. At the Leipsic Clinic it is held that the stippled cells must exceed 100 per million red cells to be diagnostic of lead poisoning. Others feel that the count should be as high as 500 per million [70].
   Urine: Urine positive for lead is only significant when more than 0.21 mg. per liter is excreted during a day [95]. Hematoporphyrinuria is regarded by many as diagnostic of plumbism.
   The urine in chronic cases shows the changes present in the urine secreted by a small contracted (arterio-sclerotic) kidney.

Disability. In mild cases of lead poisoning there may be no permanent disability. In the

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gastro-intestinal type of lead poisoning the symptoms are temporary in character and except during attacks of colic are not disabling. The cerebro-spinal form of lead poisoning is destructive and may result in permanent disability. Chronic lead poisoning produces a lowered resistance to disease and especially to alcohol which may precipitate any of the symptoms [101].

Kessler [96] gives the following criteria for the termination of temporary disability:

1. When all symptoms disappear,
2. When the employee returns to work,
3. When symptoms are no longer corroborated.

Treatment

The treatment is preventive and curative. Preventive treatment consists of the protection of the lead worker by mechanical devices which prevent his exposure to lead dust and fumes.

Instruction as to personal cleanliness and provision of proper locker and wash rooms is necessary and advisable. When mechanical removal

of dusts and fumes is impossible, air masks or respirators must be worn by the workers.

In spite of all care occasional cases of lead poisoning may occur unless the work and workers are under good medical supervision and there is regulation of hours of exposure. The patient should be removed from lead work at the first symptom.

The curative treatment consists of diet and drugs. The diet should be calcium rich as long as symptoms are present in order to maintain the storage of lead in the bones. Calcium may be given freely in milk (2 quarts a day) and as calcium lactate (60 grains, or 4 gm. daily).

The very slow intravenous administration of 20 cc. of a sterile solution of 5 per cent calcium chloride has been shown by Bauer, Salter, and Aub [7] to relieve the pain of lead colic more rapidly and more constantly than any other form of treatment they have employed. Hot applications are helpful, morphia may be indicated. When it is felt that the acute symptoms have abated and that it is safe to remove the lead slowly from storage, a diet low in calcium

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should be instituted (meat, potatoes, rice, macaroni, milk-free bread, sugar, apples and bananas. Milk, eggs, and green vegetables should be avoided). "Ammonium chloride 3 ii-iii (8 to 12 gm.) a day, sodium bicarbonate gr. 30–60 (2 to 4 gm. a day) or dilute phosphoric acid in doses of 3 v. (20 cc.) may be given to aid excretion." Potassium iodide in doses of from 5 to 10 grains three times a day also aids in excretion [131].

The anemia and other symptoms are treated in the usual way. For catharsis Carlsbad salts or magnesium sulfate are indicated.

The nervous symptoms require neurological treatment, such as massage and electricity. Legge speaks highly of the electrical treatment of the paralysis by galvanism. The treatment should be started early and consists in arm or foot baths through which the current passes [104]. The paralyzed limb should have necessary splints for support (Fig. 27).

Metal Fume Fever

The febrile reaction which follows exposure to a thick cloud of zinc oxide fumes from molten



Fig. 27. Cock-up molded leather splint for use in cases of double wrist-drop. (*Courtesy of Dr. Kessler* [96].)

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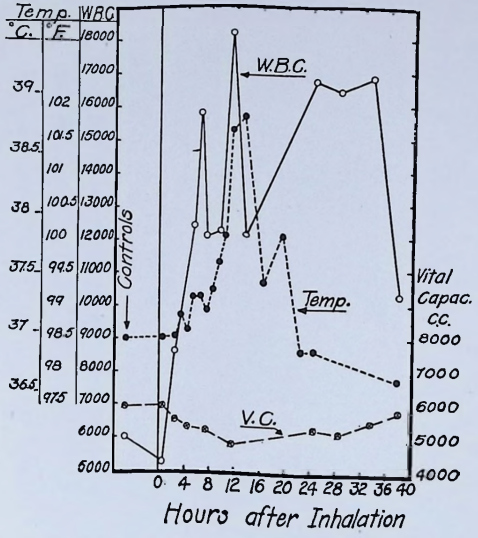


FIG. 28. A typical attack of metal fume fever, showing increase in leukocyte count, body temperature, with drop in vital capacity. Note that fever abates before the white count returns to normal. (*Courtesy of J. Indust. Hyg.*)

brass has been very well described by Hamilton [70] to whose account we add but little. The malady is purely an industrial one and has been known for many years [40]. The cause is always the inhalation of very finely divided particles of metallic oxides like zinc oxide, magnesium oxide, and probably others, which would ordinarily be considered wholly inert. The fever never is caused by swallowing the oxides, and only by fumes consisting of particles of the order of 0.1 to 1μ (Fig. 10).

The fever is much like that caused by a typhoid inoculation or by any other foreign protein. There is no reason, therefore, why it should be caused by any particular metal oxide—any very fine, inert, insoluble and well dispersed metallic powder or oxide might do it. The effects cannot be predicted from untried substances; cadmium oxide is definitely dangerous [138] while magnesium oxide [42] is much less likely to produce the fever than is zinc oxide. We would anticipate trouble from non-metallic fumes from burning iron welding rods with high silicon content, from burning ferrosilicon or

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pure silicon. Yet we have reports on exposures to condensed silica vapor from boiling off quartz in making quartz apparatus. Trouble was expected but the men were watched with especial care, the ventilation was unusually good, and so far no symptoms whatever have appeared.

K. B. Lehmann [106], whose work in this field is classic, thought the fume fever to be the direct result of absorbing protein from the epithelial cells lining the respiratory tract. Schmidt-Kehl [153] claims to have proven the validity of Lehmann's hypothesis by exposing germ-free rabbit serum to zinc oxide, centrifuging off the zinc oxide, and then injecting the centrifuged serum subcutaneously and intravenously into rabbits. Control experiments, without zinc oxide, were negative but the serum exposed to zinc oxide caused febrile reactions.

There is no sure way of aborting an attack of fume fever if the subject has had sufficient exposure. It must run its course which is always unpleasant but apparently never serious; there are no cumulative effects and the fevers are apparently never fatal. A definite resistance is acquirable from exposure and the extent of this resistance can be gauged by the leukocyte count —when the count is high the subject may breathe the fumes and not get the fever [41]. Consequently it is commonest on days following holidays when the leukocytosis has subsided.

In Figure 28 is shown graphically a typical attack of the fever. Note that the body temperature reached its peak some 8 to 12 hours after inhaling freshly burned, well dispersed zinc oxide, and that the leukocyte count was still well above normal on the next morning. Body temperatures may rise as high as 104° although the usual rise is to about 102°. Severity of the reaction varies considerably with the individual.

The substance breathed must be both finely divided and well dispersed (Fig. 10). Clumps and aggregates of particles cannot be kept in suspension in the air. In many processes, therefore, where finely divided and well-dispersed metal powders and metallic oxide powders are breathed by humans, a reaction of some sort is to be expected. It may be severe, such as that from cadmium oxide, mild like that from mag-

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nesium oxide, or doubtful like that from iron oxide fume from welding.

These reactions should be distinguished sharply from chronic cumulative effects such as may result from repeated breathing of lead fumes or manganese dioxide. In these, fume fever reactions occur, if at all, a few hours after exposure, but true lead or manganese poisoning occurs only after repeated exposures.

Metal fume fever is easy to prevent, first by local exhausts and second by dust respirators. Exposures are generally intermittent—during a pouring of molten metal or during the welding of some particular piece of metal. Processes of this sort are ideally adapted to the use of simple but effective respirators as described on pages 215–16. Drinker and others [43] showed that permissible doses of zinc oxide, and probably of other fever producers, were fairly definite and rather high compared to allowable dustiness for pure silica.

Generally we find attacks of fever today as a result of welding or cutting galvanized iron sheets or pipes. It has become rather rare in brass

foundries and in zinc smelting operations where it is well known to all engaged, particularly to the older employers. Lipkowitsch [108] does not believe that the modern electric welding processes for iron and steel offer a health hazard of any consequence. New steels with varying percentages of metals other than iron are, however, rapidly coming into use. It is advisable, therefore, to make certain that in new processes neither the fumes from welding nor from the coatings of the welding rods are harmful.

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CHAPTER X

INDUSTRIAL DERMATOSES

THE INDUSTRIAL DERMATOSES are very confusing as many substances with which workers come in contact may produce a skin reaction. This reaction may be rather general, occurring to most of those exposed, or it may be confined to workers whose skins appear to be particularly sensitive.

In order to simplify the subject the dermatoses will be divided into (*a*) those which appear as an acute reaction to chemical irritation (dermatitis venenata), (*b*) those in which the reaction is a slow one and frequently takes the form of an eczema, (*c*) those in which the reaction takes the form of a localized infection, and (*d*) those which are or may become malignant. Of these four types the "trade eczema" is by far the

most common form of dermatitis. It is usually slow of onset, affects exposed parts, and resists treatment unless and until the worker is removed from exposure to the skin irritant.

The eczema is often complicated by a fungous infection which maintains the irritation and prevents healing. The eczema itself is not characteristic and follows the usual non-industrial types, either spreading, moist and acute, or slow, dry and chronic.

In a susceptible skin almost any repeated mechanical or chemical irritation will produce an eczematous reaction. Even such a mild combination as soap and water by its continued use, as among washerwomen, will frequently cause eczema [100].

The U. S. Department of Labor [45] lists the following twelve groups of chemicals as being particularly apt to produce eczema:

Aniline and other amino compounds of benzol and its homologues,
Antimony and its compounds,
Formaldehyde,
Mercury and its compounds,
Nitroglycerin,
Phenol,

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Arsenic and its compounds,
Bromine,
Chromium compounds,
Tar,
Tellurium,
Turpentine.

Herxheimer [100, p. 909] has listed 74 different "trade eczemas" and there are new ones constantly appearing as a result of new manufacturing processes and new chemicals.

Why some individuals are susceptible and others not has been discussed at length by dermatologists, but no conclusion has been reached. It is recognized however that skins which sweat easily or are seborrheic are particularly susceptible [167, p.33]. The tendency of these "trade eczemas" is to persist for some time after the patient has been withdrawn from exposure, and comparatively slight irritation or mild reexposure causes a prompt return, suggesting sensitization of the skin. In some cases, as in chrome and in arsenic exposure, ulceration may develop in eczematous cracks.

In contradistinction to the chronic trade eczema is the acute reaction to chemical irritation.

This usually begins as a general redness and swelling of the parts affected, and goes on to the formation of vesicles and possibly pustules. The last is usually owing to secondary infection of the vesicles either from the skin itself or from without, through scratching or contact with dirty material. It resembles the dermatitis venenata of poison ivy, causing the same type of itching, burning, and discomfort.

According to White [168, p.36] [167, pp. 47–51] there are "distinguishing features of the occupational dermatoses: they crop up in series rather than single, and are characterized by their intense pruritis, grouping, situation, mode of appearance, spread and evolution from and around the stomata." White believes that most occupational dermatoses originate in the stomata, in hair follicles, or in folds of the skin, spreading from these sites.

When the skin comes in contact with strong chemicals, there may be a chemical burn with the usual picture of a burn and in severe cases a slough.

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The third and very common form of dermatitis comes from contact with oil. This is termed oil folliculitis.

While it is known that certain petroleum products have a definite irritating effect on the skin, in the case of oil folliculitis the dermatitis is caused by the effect of a mixture of oil and dirt plugging the hair follicles. This first produces a blocking of the gland secretion and folliculitis, which may be followed by a secondary infection, usually of staphylococcus aureus. The crop of papules covers the exposed forearms and backs of the hands. In the early stage these show a black spot in the center. Later they may become typical furuncles.

A second type of oil dermatitis is multiple furuncles due to the infection of minute abrasions and cuts received from the metallic dust mixed in the oil or from the fine steel chips clinging to the dirty waste with which the worker wipes his hands and arms. Other forms of infection of the skin may occur as anthrax, erysipeloid, and tuberculosis, but these are comparatively rare and not confined to industry.

In the fourth form of dermatitis, we find those reactions of the skin which occur as the result of prolonged mechanical irritation. Some of these occur with enough frequency to have received trade names as in miners' "beat hand, beat elbow, and beat knee." In beat hand there is thickening of the palmar skin with inflammation and swelling of the subcutaneous tissue. This is owing to the effect of the pick handle upon the hand. Occasionally suppuration occurs in the deeper layers of the skin. Beat elbow and beat knee are similar in character and arise from pressure and friction against the walls of the mine [144, pp. 1270–1271]. Similar but milder pressure calluses occur in a number of occupations which call for the continuous use of tools. Glassblowers develop heavy calluses of the hands, nature's method of protection [96, p. 643].

The tars and petroleum products have a definite irritation effect on the skin and in addition appear to have carcinogenetic properties [70, pp.250–257]. The reaction is at first a dermatitis with thick reddened skin over the exposed area. Later red papules form as in oil folliculitis.

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Some time later there is general thickening of the skin and numerous papillomata appear. The papilloma ulcerate at their bases and drop off, leaving ulcers. Epitheliomata may develop on the edges of these ulcers. Such conditions are rare in the United States.

The diagnosis as to whether a skin condition is or is not owing to some substance with which the worker comes in contact during his work is very perplexing. In most cases there is nothing to distinguish the occupational from the non-occupational dermatitis. It must also be remembered that there are many contacts with irritating substances which occur outside of working hours. Thus the worker may have photography as a hobby or use lacquers or varnishes in decorating his home. The hands of a woman who does housework as well as industrial work are exposed to a multitude of chemical substances, such as mixed alkalies, metal polish, and similar substances. The reaction to these may easily be considered as coming from the materials with which the worker comes in contact.

Schwartz and Pool have recently shown how

difficult it is to determine the exact cause of an irritating dermatitis affecting a number of workers in a cotton mill [154].

A method of diagnosis which is suggestive but by no means conclusive is the patch test. This is usually performed as follows:

A very weak solution of the suspected substance is made, and a circular piece of absorbent material about the size of a dime is saturated in the solution. This small patch is applied to the arm or the leg and covered by a piece of cellophane approximately the size of a fifty cent piece. The cellophane is held in place with adhesive plaster. A similar patch without solution is placed on the opposite arm or leg for control. The object of having the cellophane larger than the circular piece of material is to allow observation of any redness as the skin reacts to the solution. The patch test is left in place for 24 to 36 hours, being removed at once if reaction appears. Care must be taken in persons who have sensitive skin conditions to remove the dressing early if discomfort is complained of as the reaction is occasionally very severe.

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The prevention of industrial dermatosis is obtained by:

1. Recognition of danger from the skin irritant,
2. Proper washing facilities,
3. Instruction of workers in methods of preventing skin irritant from coming in contact with exposed parts, and the treatment of the normal skin with lanolin,
4. Wearing long sleeves to protect arms, handkerchief or gauze about neck; gloves to protect hands; gauntlets buttoned under sleeve of overall. Where gloves are impractical, use General Electric Hand Cream No. 406 after washing with soap and water;
5. Thorough washing of exposed parts after work with a non-irritating soap; shower bath if much exposure;
6. Engineering methods which keep irritating substances from circulating in the air or coming in contact with the skin of the worker.

The general treatment consists in removing the worker from contact with the offending substance, or by protecting the affected parts from such contact. The treatment of the skin disease is similar to that of the condition as found in general practice.

Arsenic

The U. S. Department of Labor lists thirty occupations in which there is some hazard from contact with arsenic. In New York State there were but six occupational disease claims in 1931 and but eight in 1932 in which arsenic was named as the cause of the disease [157].

Industrial arsenic poisoning, according to Legge [103], occurs in two ways—first, from the dust of salts of arsenic and second from arseniuretted hydrogen gas (arsine).

The salt causing most trouble is white arsenic or arsenious acid (As_2O_3). The dust of arsenic salts in industry has local, not constitutional effects. The dust which settles on the skin and which is inhaled produces a local dermatitis in

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the first instance and a chronic catarrhal condition in the second.

The dermatitis may occur anywhere on the body but is most frequent where there are folds, as around the nose, at the corners of the mouth, and in the groin. The arsenic salts also attack moist surfaces as the axilla or scrotum.

The inhaled dust produces coryza, dryness of the throat, and hoarseness with an irritating cough. These symptoms are not severe but are troublesome. The dermatitis is very irritating; it starts as an inflammation which may go on to ulceration forming "arsenic pocks." The eruption is itching and burning in character and causes a great deal of discomfort and irritation. If the worker neglects the condition a variety of skin lesions may develop such as scleroderma, melanoderma, warts, and a slowly developing cancerous degeneration which usually has a fairly benign course and does not give rise to metastases [68]. Loss of hair is common.

Just as the dust of arsenic salts causes ulceration on moist body surfaces so it may cause ulceration of the cartilaginous septum of the nose

leading to perforation, "varying in extent from a circular hole ⅛ to 1 inch in diameter" [100]. In this lesion the destruction is confined to the upper and posterior portion of the cartilage so that there is no deformity. The perforation is painless.

Arsenic absorption may cause parasthesia and peripheral neuritis but in industry these symptoms are rare.

Arseniuretted hydrogen or arsine is an extremely dangerous gas and always occurs in the form of a by-product in some industrial process where nascent hydrogen is formed in the presence of arsenic. The most frequent cause of poisoning from arsenic in this country, according to Hamilton [70] "is the cleaning out of tanks and tank cars that have contained a heavy acid, either hydrochloric or sulfuric." The weak acid left as sludge after cleaning the tank with water acts on the arsenic in the iron, producing arsine.

Arsine acts by causing hemolysis of the red blood cells. This action gives no symptoms for a few hours after exposure, then there is a pro-

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found reaction due to the destruction of the red blood cells. The early symptoms are nausea and vomiting followed by edema of the face and eyelids and the passage of dark or bloody urine. The kidneys are clogged in their efforts to dispose of the broken down blood cells. Later symptoms are jaundice and anuria. If the patient recovers there is a persistent anemia. Death occurs in about 20 per cent of the cases [100]. Arsenic can be detected in the urine during the acute attack and for some time afterwards.

The only treatment according to Legge [103] is oxygen inhalation and transfusions.

Anthrax

The one industrial disease caused by a special bacterium is anthrax. There are a number of common parasites, such as hook-worm which infect workers in the South, but these are in no way peculiar to industry. Bacteria other than the anthrax bacillus may attack workers but none are specific to a special industry or to industry alone, as they occur among the general population.

Anthrax in industry is confined largely to two occupations, wool sorters and tanners. Thus the records of the Massachusetts Department of Labor and Industries from 1929 to 1933 show that of a total of 43 cases investigated, 36 occurred in tanneries [111], while in England the majority of cases over a longer period were found among wool handlers [83, pp. 326–27].

The disease is caused by the anthrax bacillus present in animal hides and wool which are infected. It is usually encountered in one of two forms: as a skin infection (malignant pustule) or as a lung infection (wool-sorter's disease). In the former the infection comes from direct contact of the infected hide with the broken skin of the worker; in the latter it is inhaled in the form of dust from infected wool.

Symptoms of malignant pustule. After an incubation period of from 1 to 3 days, a small, hard, painless papule appears at the site of infection. This papule becomes vesicular, discharging a bloody serum, and is surrounded by a spreading area of redness and induration. Within 36 hours it is covered by a hard black

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tenacious crust. At this time the induration may show small vesicles containing brown fluid on the protruding edge of the central pustule. The whole inflammatory area tends to become edematous and the glands draining the affected area are swollen and painful [53].

With the local manifestations, there are systemic symptoms; there is a sharp rise of temperature which later falls, frequently becoming subnormal. The pulse is rapid, weak, and irregular. Chills with vomiting and signs of collapse follow the onset. The patient complains of headache and muscular or arthritic pains. In fatal cases death occurs on the ninth or tenth day [155].

Pulmonary form of anthrax (wool-sorter's disease). When the anthrax bacilli or spores are inhaled with dust there is sudden onset with chills, fever, vomiting, dyspnea and cyanosis. Cough is not pronounced, respiration is rapid, and the patient complains of pain in the chest. Collapse occurs early and death may ensue in 24 hours. Anthrax bacilli may be present in the sputum.

Intestinal form of anthrax. This form is rare and follows the ingestion of food which has been contaminated by dirt on the hands or nails of workers which contain anthrax bacilli or its spores. It may also occur from eating infected meat. The symptoms are similar to those of the pulmonary form except that chest symptoms are lacking and there may be diarrhea. The spleen may be enlarged and there may be hemorrhages of the mucous membrane.

Diagnosis. In the early stages the malignant pustule may be differentiated from carbuncle by its lack of pain and its more severe symptoms coupled with the occupational history. Anthrax bacilli may be found in the discharge of the pustule or the surrounding vesicles.

Prevention. Workers who handle hides should have two locker rooms and adequate washing facilities. In one locker room the street clothes are removed, in the other the work clothes are put on. When the worker leaves work he takes off his working clothes in one locker room, washes all exposed parts of his body in the lavatory and then puts on his street clothes.

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While working, exposed parts should be protected by gloves, long-sleeved overalls, and suitable protection about the neck.

All minor injuries should receive prompt attention and anti-anthrax serum given at the slightest indication of pustule formation.

Workers employed sorting wool should wear respirators. An excellent description of preventive measures is published by the Massachusetts Department of Labor and Industries [111].

Treatment. The treatment of malignant pustule as given at the Cook County Hospital, Chicago, is as follows:

1. Bed with affected part elevated and covered with warm boric acid compresses.
2. Local injection of 10 to 12 cc. of anti-anthrax serum by three punctures 1 to 1½ inches deep into the tissues just external to line of edema in such a way as to circumscribe the lesion.
3. Injections repeated at intervals of 24, 12, or, in severe cases, of 6 hours, until from four to six injections have been given or, rather, until the inflammation, which is

increased after the first few injections, shows definite evidence of subsiding.

4. Intravenous injection in non-septicemic cases of 50 to 200 cc. of antiserum every 12 to 6 hours according to severity of case followed by intramuscular injections when the disease is under control.
6. In cases with positive blood culture (septicemic cases) 200 cc. of antiserum is injected intravenously every 3 hours until blood culture is negative [50].

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Chapter XI

GASES, BENZOL, ASPHYXIA AND ARTIFICIAL RESPIRATION

The Toxic Effect of Gases

The physiological classification of gases suggested by Henderson and Haggard [79] enables one to understand and often to predict the effects of some particular gas. Hamilton [70] has covered well the systemic effects from the various chemicals and their vapors. We shall borrow freely but briefly from both these sources.

Group I. Asphyxiants. Asphyxia may result from oxygen lack as in the ascent to very high altitudes or it may be caused by some peculiar property a gas possesses. An excess of the wholly inert gas, nitrogen, depresses the oxygen concentration proportionally and as a result the atmosphere may become dangerously deficient in

oxygen. Carbon monoxide acts specifically upon the hemoglobin and displaces oxygen with great avidity and therefore acts as a "chemical asphyxiant." Likewise hydrogen sulfide and hydrocyanic acid (prussic acid) gas have specific and prompt asphyxial action and should be classed as chemical asphyxiants and not as simple asphyxiants.

Group II. Irritant gases. Ammonia, hydrochloric acid, chlorine, "nitrous gases," and some of the lethal war gases belong in this group. Some of them are very soluble in water and in body fluids while others, like chlorine, are rather insoluble.

All of them are irrespirable in any but very low concentrations and many cause immediate irritation resulting in coughing, sneezing, and the like. The soluble gases, such as ammonia, cause an immediate edema in the upper respiratory passages, whereas the insoluble gases, like chlorine, are more likely to produce their ill effects in the form of a dangerous lung edema which may not be apparent for some 12 to 36 hours after exposure.

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In the disaster in the Cleveland Clinic which resulted from the burning of x-ray films, the immediate deaths were owing mostly to carbon monoxide poisoning, whereas those which followed a pneumonia on the second or third day were owing mainly to the nitrous fumes. The carbon monoxide is a "chemical asphyxiant" and the nitrous fumes are "insoluble irritants."

Group III. Anesthetics, volatile drugs, and drug-like substances. This group includes the typical surgical anesthetics like ether and chloroform, the vapors from the common organic industrial solvents like gasolene, carbon tetrachloride, benzol, and many of the newly developed compounds used in household refrigerators. Some of these substances, like benzol and tetrachlorethane, exhibit distinct cumulative or chronic action in addition to their purely anesthetic properties.

Group IV. Inorganic and organometallic gases. A typical example of this group is tetraethyl lead, the much advertised anti-knock ingredient of present day motor car gasoline. Another example is mercury vapor. Most of the

gases falling in this classification are extremely toxic. The dangers are very well known in industry and troubles from breathing the gases in question are generally the result of an accident, like a broken pipe line, and are rarely owing to toxicological ignorance on the part of industry.

Treatment of Gas Poisoning

Obviously the treatment of victims gassed by the substances described in the several groups will vary somewhat with the gas. However, the physician will not err if he removes the victim immediately from the source of poisoning, keeps him quiet and warm, and administers oxygen-carbon dioxide if respiratory difficulty is apparent. It is important, often, to keep the victims quiet, particularly if the offending gas is one of the irritant group like nitrous fumes or chlorine, until it is certain that a pneumonia is not going to follow the exposure. This sequel was, of course, a familiar occurrence in the World War following gassing by chlorine.

Chronic effects, like those from benzol, re-

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quire specific treatment which again varies with the individual substance.

Benzol

As an instance of one of the more important chemical poisons, benzol will be briefly discussed. Benzene or benzol is a coal tar distillate which is largely used in industry. The U. S. Department of Labor [45] lists 75 occupations in which there is exposure to benzol or one of its homologues (toluol and xylol).

Benzol is widely used in the chemical industries. Though it has been in use only a comparatively short time [62], it is one of the most important of the industrial poisons [87]. The concentrated vapor of benzol is very poisonous and there are a number of recorded deaths from its inhalation [130] [70].

In diluted form it is present in the work rooms of certain industries, although because of its poisonous qualities its use is declining [69]. In mild concentration it produces a chronic form of poisoning with injury to the blood-

forming organs and to the central nervous system.

Acute poisoning is usually accidental. A worker is unexpectedly exposed to benzol vapor from a leak at some part of a chemical process, or without proper protection he enters a tank which had previously contained benzol. Shortly after exposure begins, the worker's mind becomes confused, he staggers and falls unconscious with anesthetic and neurotoxic symptoms. Workers who are unexpectedly stricken apparently are less liable to die than their would-be rescuers, who enter an enclosed space, do hard physical work, and finally collapse [130].

The patient is found unconscious. He should be removed from exposure as quickly as possible and immediately given artificial respiration by the manual method, followed as soon as possible by treatment with an oxygen and carbon dioxide inhalator. Those engaged in the rescue should wear an air mask and should not rely upon a gas mask.

The chronic form of poisoning is more com-

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mon. Workers are often exposed in rubber processes, artificial leather, sanitary can manufacture, dry cleaning, and in making of paints, varnishes, and stains.

The Benzol Committee of the National Safety Council [130] and the Smyths [160] have set the limit of 100 parts of benzol per million of air as the standard of permissible concentration for continuous exposure. However, control of the amount is very difficult because of the irregularity of distribution of the vapor through the workroom.

Personal idiosyncrasy is an important factor in this as in other forms of industrial poisoning, and one worker out of a roomful may be affected by a comparatively low concentration which has no effect on the other workers.

The symptoms of chronic poisoning may be divided into those resulting from irritation of the nervous system, those from injury to the blood-producing organs, and those from skin contact.

Of these the second is most important. The characteristic symptom of benzol poisoning is

anemia, which is of the aplastic variety in which all elements of the blood show degenerative change. There is reduction in the number of white blood cells, particularly the granulocytes, reduction in the number of red cells, in the percentage of hemoglobin and in the number of blood platelets. These changes result from the effect of benzol on the bone marrow, on the spleen, and on the lymph glands.

While aplasia of the bone marrow is the usual finding at autopsy, Hamilton [70] has pointed out that hyperplasia may be present.

The effect of the continuous inhalation of benzol vapor is evidenced by those vague symptoms usually grouped under malaise, such as headache, dizziness, weakness, loss of appetite, and loss of weight. With these symptoms is found the pallor of a true anemia. Accompanying the anemia are sore, spongy gums which often bleed, complaints of burning eyes and dry throat, and the presence of purpuric spots, which last is an important symptom. Sometimes there are signs of internal bleeding, while bleeding from the nose or prolonged menstruation is

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fairly frequent. Because of the destruction of the protective cells in the blood, suppurative infections are common and may be serious.

Nerve irritation is shown by slight tremors, complaint of visual disturbances, abnormal sensitiveness to touch, and headache.

While skin erythema, papules, or eczema may develop from contact with benzol, such reactions are rare.

Considerable animal experimentation has been done to determine the pathology of chronic benzol poisoning. These experiments [130] show that there is first a slight increase in the number of leukocytes followed by a rapid fall. The form most affected is the polynuclear leukocyte which is frequently reduced to one-half its usual percentage in the blood. While the lymphocytes are also reduced, their proportion to the polynuclears remains high. There may or may not be a marked diminution in the number of red cells. This phase, the reduction in number of red cells is earlier and more important in human benzol poisoning than in the experimental poisoning of animals [70].

While there are degenerative changes in the blood-forming tissues, these are not permanent except in extreme cases, as patients suffering from benzol poisoning tend to recover on removal from exposure. In this respect the picture differs from idiopathic aplastic anemia, where the degeneration of the blood-forming tissues is apparently complete and permanent. The condition of the bone marrow in aplastic anemia is, however, still under discussion [173].

While there is a reduction in the number of all cellular elements in the blood in benzol poisoning, no unusual or irregular forms are usually present though Hamilton [70] reports cases in which they have appeared.

The diagnosis finally appears to rest on the proved exposure to benzol vapor over a sufficient period. Anemia of any type, especially with leukopenia, occurring in a worker with a history of a long period of exposure to benzol vapor in the air of the workroom, must be considered a probable case of benzol poisoning and treated accordingly.

The treatment may be divided into preventive

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and curative. The former measures consist in a pre-employment examination including a careful blood examination to prevent workers with disease of the heart, blood, lungs, or kidney from exposure to benzol vapors. Following this there should be routine examination of the blood of those in contact with benzol, in order to detect anemia or leukopenia in its earlier stages.

Workrooms should be well ventilated and hoods with exhausts should be used where possible to trap the vapor at its source. Unfortunately this is frequently impossible as has been pointed out by Hamilton [69]. The number of workers exposed to the vapor is decreasing steadily as a result of the substitution of homologues of benzol which are less toxic, and by eliminating its use entirely.

The curative measures consist in removing the worker from benzol exposure and in endeavoring to stimulate the blood-forming organs to renewed activity. This is attempted in severe cases by transfusions. Usually removal from work, combined with general hygienic measures are sufficient to promote cure. It must be remembered, however, that cases of chronic benzol poisoning may develop an increasing anemia after they have stopped work, and must be watched and treated for a considerable period after the exposure has ceased. Workers who have suffered from chronic benzol poisoning should be kept from future exposure.

The State of Massachusetts has in effect a law requiring all receptacles of benzol, except tanks of automobiles to be marked with the words "Benzol, beware of poisonous fumes," and to be clearly labeled showing the benzol content.

Asphyxia and Artificial Respiration

In ordinary life asphyxia [36] occurs most commonly as the result of drowning, gas poisoning, and electric shock. The first is the commonest and the last rarest. Drowning, as an industrial accident, can be dismissed as it belongs to the hazards of every-day life, rather than to industry. Gas poisoning frequently is a hazard of industrial life and occurs usually in connection with the distribution and use of artificial illuminating gas, the toxic ingredient of which is

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carbon monoxide. It has also become a well recognized hazard in the form of automobile exhaust gases. Since the most dangerous voltages and currents are handled by a small specialized group in industry, electric shock is apt to be industrial in origin. Fatal accidents from the ordinary household electrical appliances are, however, by no means unknown.

The asphyxia of drowning results from flooding the lungs with water—the heart is beating strongly at the moment water is breathed in and may continue to beat for a few minutes after respiration stops. Sometimes, therefore, the victim of drowning can be aided if promptly lifted at the waist and the head hung down so that water runs out of the mouth. Artificial respiration then must be applied immediately.

Asphyxia from carbon monoxide poisoning is owing to the property this gas possesses of combining with the hemoglobin of the blood about 300 times more easily than does oxygen. The effect of breathing air containing carbon monoxide is, therefore, similar to the anoxemia one experiences in ascending to a very high altitude.

Death results promptly if the gas concentration is high, or slowly if the victim is gassed but slightly and not removed from the contaminated air. Deaths from high concentrations are owing to the immediate effects of oxygen deprivation while the slow death, which may not occur for several days, is owing to the cumulative decerebrating effects of oxygen lack.

The asphyxia resulting from electric shock may follow cardiac fibrillation for which there is, at present, no treatment. Prolonged contact with high voltage current may cause permanent tissue destruction as a result of burns or of heat. Then of course, artificial respiration is unavailing. Again, asphyxia may be the result of the severity or violence attending the shock itself; the victim may recover spontaneously, or he can often be treated successfully by simple artificial respiration. Maclachlan [109] describes a case of electric shock in which artificial respiration was applied intermittently for 8 hours before the victim could breathe unaided.

The Schäfer method of artificial respiration. The men (including physicians) with most ex-

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perience in applying artificial respiration today are the employees of gas and electric companies in our large cities. In addition to their regular duties their "trouble men" respond to all fire alarms and to reports of accidents from illuminating gas or from any other kind of gas poisoning such as from motor cars.

The physician probably does not realize that the application of the Schäfer method to an average sized man is hard work. The employees of the utility companies generally work in teams of three, each man working for 20 minutes, and the team continuing for as long as 4 hours or until rigor mortis has obviously commenced. A person in any but the best of physical condition will find this ordeal extremely tiring.

The Schäfer method has gradually superseded all others in this country and Canada. A pamphlet used by the Consolidated Gas Company of New York gives the following directions for applying this method.

1. Lay the patient on his belly, one arm extended directly overhead, the other arm bent at elbow and with the face turned outward and resting on hand

or forearm so that the nose and mouth are free for breathing.

There are two reasons for the face-down position. First of all, it clears the upper air passages more adequately than can be attained in any other way. Not only does the prone position tend to open the upper air passages but if, as frequently happens, the victim vomits or if fluid comes up into the mouth from the lungs, it runs out freely and no chance is given to hindrance of entrance of air.

The second important reason for the prone position consists in the fact that pressure, bringing about reduction in the size of the chest and so expelling air, can be applied more safely and effectively from behind than from in front. With the patient upon his back, one might readily press the contents of the belly forward and move the diaphragm with them, but the pressure would be applied directly to the intestines and contents of the belly, and there is no doubt that it would prove dangerous to do this repeatedly over a period of hours. When the pressure is applied from behind, the force is distributed more safely, and results in a movement forward of the abdominal contents and a compression of the chest from back to front which has a maximum effect in expelling air.

Place the palms of the hands on the small of the back with fingers resting on the ribs, the little finger just touching the lowest rib, with the thumb and fingers in a natural position, and the tips of the fingers just out of sight. The position of your hands is important. You apply pressure to the lowest ribs which

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are free to move and are consequently not in danger of breaking when the pressure is given properly (Fig. 29). If your hands are too high you operate upon the more thoroughly supported upper ribs, and at times these may readily be broken by even a moderate pressure.

With arms held straight, swing forward slowly so that the weight of your body is gradually brought to bear upon the patient. The shoulder should be directly over the heel of the hand at the forward swing. Do not bend your elbows. This operation should take about two seconds.

When you make the motion in this direction as illustrated in the diagram, you compress the chest and push the diaphragm upwards. Air is driven out of the lungs. Your active movement accomplishes expulsion of air. In natural breathing the active part of the process enlarges the chest and lets air into the lungs, and in its turn, breathing out is passive. The prone method of artificial respiration reverses the procedure, air being expelled by pressure and entering the lungs as a result of the passive expansion which follows release of pressure. The effect is shown in Fig. 30.

Now immediately swing backward so as to remove completely the pressure.

When you release your pressure you obviously allow those parts you have compressed to spring back to their normal position, and this in its turn accomplishes enlargement of the chest and inrush of air.

After two seconds, swing forward again. Thus

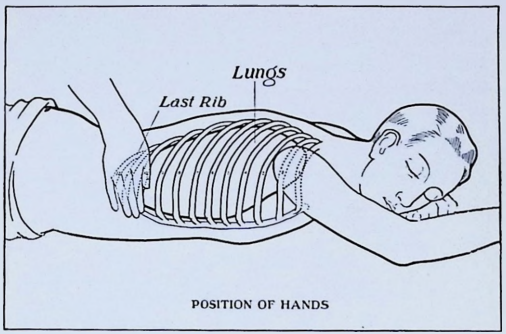


FIG. 29. The position of the hands in relation to the ribs. (*Courtesy of the Consolidated Gas Company of New York.*)

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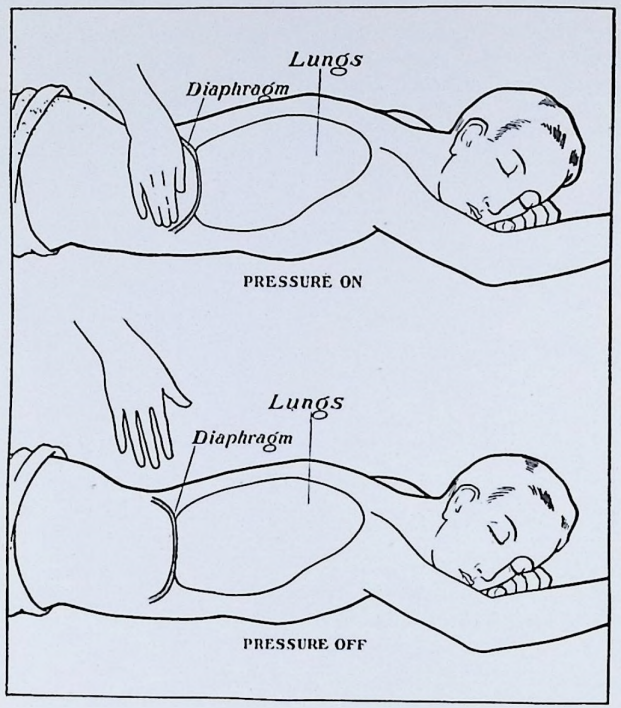


FIG. 30. The effect of pressure and release of pressure in changing the size of the chest. (*Courtesy of the Consolidated Gas Company of New York.*)

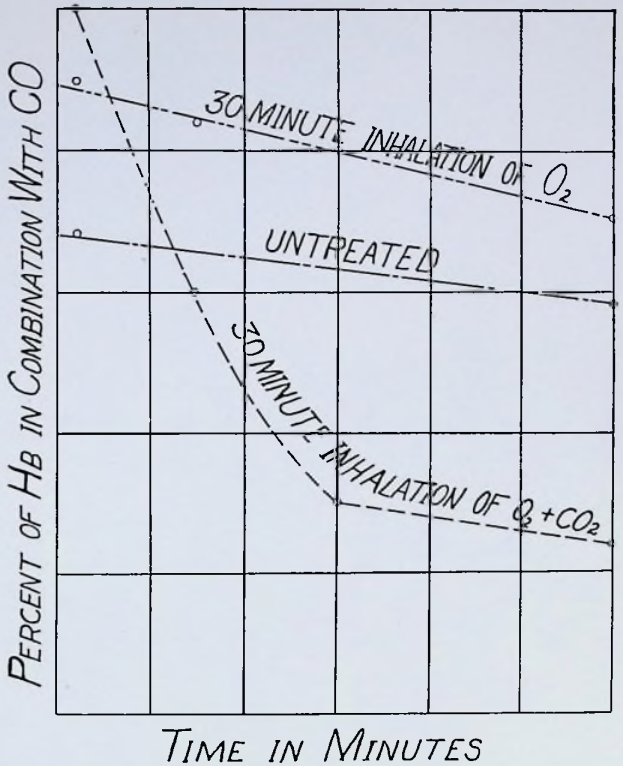


FIG. 31. Curve showing the beneficial effects of oxygen-carbon dioxide on patients seriously gassed with carbon monoxide. (*Courtesy of Drs. Henderson and Haggard* [80] *and the J. A. M. A.*)

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repeat deliberately twelve to fifteen times a minute the double movement of compression and release, a complete respiration in four or five seconds.

A partially asphyxiated person loses heat readily and special care must be taken to keep him warm. This point is sometimes ignored by laymen and the physician called to treat an asphyxiated patient should see that the necessary precautions are taken.

Oxygen-carbon dioxide inhalation. The oxygen-carbon dioxide inhalation devices, for the origination of which Henderson and Haggard [80] are responsible, offer a simple and safe means of giving a gassed victim a mixture of 7 per cent carbon dioxide and 93 per cent oxygen. Henderson and Haggard showed (Fig. 31) that the blood of a man gassed with carbon monoxide up to 35 per cent and to 50 per cent hemoglobin saturation could be brought down to 15 per cent saturation in 30 minutes when the patient inhaled the oxygen-carbon dioxide mixture. The same men, gassed to comparable saturations were relieved very slowly when oxygen alone was inhaled.

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C. K. Drinker and Shaughnessy [38] showed that 7 per cent carbon dioxide with 93 per cent oxygen was generally better than the 5 and 95 per cent mixture originally advocated by Henderson and Haggard. In actual practice administration of this gas mixture with the "inhalators," is an invaluable aid in the treatment of carbon monoxide poisoning. The inhalators have displaced entirely the pulmotor although devices which crudely imitate the latter keep cropping up from time to time. Occasionally ill-informed physicians come upon men using an inhalator on a gassed victim and, thinking it is a pulmotor, tell the men to stop.

It is not certain whether the oxygen-carbon dioxide inhalations are of any real help in the treatment of asphyxia from drowning or electric shock. If the victim has been gassed from some substance, the effects of which are essentially anesthetic, treatment with the inhalator is definitely indicated and is beneficial just as it is in de-etherization after surgical operations.

The physician who anticipates contact with workmen of any large company today would do well to examine and try personally an oxygen-carbon dioxide inhalator and especially to practice the Schäfer method and to have it practiced upon him. Otherwise he may find himself in the embarrassing position of receiving instruction, in an emergency, in a life-saving procedure which has been developed by the medical profession but in which the most proficient teachers are now laymen and not physicians.

After-effects of asphyxia. Serious cases of asphyxia requiring long treatment should, if possible, be taken to a hospital, artificial respiration being given en route. The most dangerous sequel to serious asphyxia, particularly by gas poisoning, is pneumonia, and the dangerously gassed patient should be treated with all the care given a patient coming out of ether after a long operation.

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Chapter XII

THE PREVENTION OF INDUSTRIAL DISEASES

Elsewhere we defined an industrial disease as a toxemia resulting from too great exposure to a toxic substance or to an unfavorable working environment. Obviously, then, the avoidance of such an exposure furnishes the first and most important defense against industrial disease. Generally it is unnecessary to prohibit *all* exposure to a toxic substance—most substances have threshold doses. In Chapter VIII we have given figures of permissible concentrations for certain dusts, fumes, and for one mist, chromic acid. Hamilton [70] and Henderson and Haggard [79] and publications of the U. S. Public Health Service give the available data on gases [151].

Perhaps the second most obvious preventive

measure is the limiting of working hours so that the exposure will be sub-threshold. Usually such limitation is developed by practical experience and later is put upon a sound scientific basis by appropriate laboratory experimentation. Notable examples of limitation of exposure are shown in the rules for working in compressed air and again for working in hot deep mines (Chapter IV). In both of these examples industry would welcome a normal full working day if the resulting exposure were physiologically safe for man.

A third method which is always sought by an industry which finds itself in toxicological trouble, is the development or substitution of a safe process for that which is causing trouble. A familiar example is the almost total elimination of benzol poisoning [69] by the use of less toxic solvents in place of benzol—toluol and xylol. In dusty trades it is often feasible to change a process so that it becomes non-dusty. Examples can be seen in any plant doing sand-blasting; a few years ago the abrasive was sand and the blasting frequently was done on the open

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floor of the foundry. Today a great deal of blasting is done with steel shot entirely free from sand, while automatic, enclosed equipment, requiring no manual control, is in common use. Many industries now using finely ground minerals containing quartz could use instead products from which most of the particles below 20μ have been eliminated. It is the part of common sense to avoid the small dust particles which are the cause of silicosis.

A fourth preventive method, which has been widely applied in the mining, metallurgical, and ceramic industries, is the substitution of wet methods for the original dry screening, grinding, milling, and mining processes. Dry drilling [157] is the cause of most dustiness in mining and while wet drilling does not insure a dust free atmosphere [72] it reduces dustiness very greatly. It is true that silicosis has not been eliminated in South Africa, where wet drilling is enforced, but if the rock contains much quartz, wet drilling and the use of water blasts are thoroughly practical dust prevention measures.

Grinder's rot is one of the oldest known forms of silicosis and is caused by the inhalation of silica particles from sandstone grinding wheels. Drury [44] showed that wet grinding reduces the severity of the dust exposure but does not, by any means, eliminate silicosis.

A fifth method for preventing the escape of undesired substances into the working atmosphere is the local exhaust hood which is placed as close to the source of pollution as possible. Generally a number of such hoods are connected up with a fan or exhauster to a central filtering or recovering plant which removes the impurities and discharges air which frequently is clean enough to be blown back into the working place. Sometimes the impurities drawn off through the local exhausts are toxic gases which are best discharged through a tall chimney or stack. A rather new application of local exhaust hoods is the Kelley trap [74] and dust collector now being applied in rock drilling. The dusty air is drawn off as it emerges from the hole in the rock, the dust is filtered out and clean air is discharged at the work place.

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A sixth method of preventing industrial disease is the routine recording of conditions and the payment of a bonus to one or more persons to maintain the desired conditions. In processes where control of temperature or of quality of product is required the measurement and recording of conditions is carried out routinely. It requires no great ingenuity to devise similar control measurements for the benefit of the workers. We discuss later the sampling and determination of dusts and gases.

A seventh preventive measure is the application of general as distinguished from local exhaust ventilation. A good example is in the ventilation of the modern multi-storied garage or the Holland automobile tunnel. In these two instances it is obviously impracticable to apply local exhausts direct to the automobiles and recourse must be had to the more expensive general ventilation—more expensive because more air must be handled than if only the gases exhausted from each car were removed.

A final and eighth method is the wearing of special protective equipment by each worker.

Although this step is often a last resort, it is nonetheless very important and will be treated in more detail.

Personal or Individual Protection

Equipment to be worn by the individual for his protection can be divided into two broad classes, that for preventing injuries from accidents, and that for preventing injuries of a chronic or cumulative sort.

Protection against the effects of accidents. (*a*) Various kinds of goggles are required in many states for protecting the eyes against foreign bodies such as chips of iron or steel thrown off in cleaning up rough castings with pneumatic chippers, or stones and fine gravel in stone cutting, dusts and metallic sparks from high speed grinding wheels, and the like.

(*b*) The "hard boiled hat" has become a fixture in the mining industry and has reduced enormously both minor and major head injuries from falling rock and from the effects of bumping into objects overhead.

(*c*) Special types of shoes, particularly

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"safety shoes" with very strong, stiff toes, are available in a wide assortment.

(*d*) Rubber aprons, coats, shin guards, gloves, and the like are standard articles used widely in handling chemicals, in sandblast work and in other jobs where the unprotected skin or clothing may mean an immediate injury.

As new processes are introduced, new kinds of protective equipment are developed and the old are improved upon. Illustrations become out-of-date and out-of-style rapidly. The best sources of current information are advertisements in the trade periodicals.

Protection against injuries of a cumulative kind. (*a*) Goggles and shields held in the hand are an essential part of the equipment of the man welding with oxy-acetylene or with the electric arc. The electric welder generally prefers the hand shields to the goggles as does the man using the newer "atomic hydrogen" welding apparatus. The ultra-violet rays as well as the intense light from welding cannot be tolerated by the unprotected eye.

Goggles for protection against intense light

rich in infra-red are used in steel mills and in examining the furnaces and hearths in modern power plants.

Specifications for goggles of all kinds are obtainable from the U. S. Bureau of Standards, Washington, and from the American Standards Association, New York.

(*b*) Respirators for protection against dusts of all sorts are available and have been improved immensely in quality in the last few years. The U. S. Bureau of Mines, Pittsburgh, has recently published a rigid testing schedule [166] which none but effective and comfortable respirators can pass.

A respirator is a filter and not a chemical remover of impurities. The filter has no effect upon gaseous impurities like carbon monoxide or benzol. Respirators passing the Bureau of Mines' test are not 100 per cent effective against fine dusts and it is unlikely that any so effective will be used in industry—they are too hard to breathe through. But they will clean moderately dusty air to the point where it is safely respirable. Though they are not and never will be a

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substitute for adequate dust control, they are applicable particularly to jobs with intermittent dust exposure and not to jobs where the dust exposure is more or less continuous.

(*c*) Gas masks include a gas-tight face piece and canister which serves to remove gaseous impurities in amounts which are of the order of 3 per cent or less. They do not effect the oxygen concentration and if that is below about 14 or 15 per cent, the gas mask should not be used. Masks and canisters are available today for protection against a combination of all gases or against any particular gas. Rigid specifications for approval have long been offered by the U. S. Bureau of Mines and masks without such approval are a foolish risk.

(*d*) Gas mask face pieces to which one blows clean compressed air have been in use for a long time by the gas companies in all large cities, by chemical manufacturing plants, and by others whose men are required to enter atmospheres of uncertain gaseous content. The general arrangement is usually termed a *hose mask* and the hose itself is especially heavy, is oil and gasoline

proof, and strong enough to be used as a life line in emergencies.

(*e*) Men doing spray painting, lead burning, and the like generally should wear *air masks,* or respirator face pieces to which a light air line is attached and through which clean air is fed at a rate controlled by the wearer. In general air masks give better protection than respirators and for severe hazards, like lead burning, are to be preferred.

(*f*) Sandblasters have one of the severest dust exposures in industry and should always be protected by rugged helmets through which sufficient air is blown to insure dust free air to the wearer. It is a common practice for the sandblaster to take off his helmet when his blasting is done and blow the loose sand off his casting, thus getting his hair full of fine sand which he blows off and breathes as soon as he puts his helmet on again. Obviously such carelessness should be prevented [16].

(*g*) Small cartridges fitted with special gas absorbents like activated charcoal, soda lime, and silica gel can be used for protection against

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very low concentrations of many objectionable gases. The cartridge is attached to an ordinary respirator face piece thus simulating a gas mask. Its effectiveness, however, is much less than that of the gas mask.

Determination of Dusts and Gases

Dusts. The physician whose patients work in dusty places should know the general technic of dust sampling but need not be expert himself. In this country dust sampling is generally done with the impinger apparatus [77], Fig. 32, developed at the Bureau of Mines, Pittsburgh, where it was tested thoroughly, and later simplified. Air is drawn into the top of the vertical glass tube and dust trapped in the water at the bottom tip of the tube where the air velocity is about 22,000 feet per minute. The actual sampling rate is 1 cu. ft. per min. as this is supposed to represent the rate at which the average man breathes, and is probably the outgrowth of Lanza's early work in which man was the suction pump. With the mechanical pumps

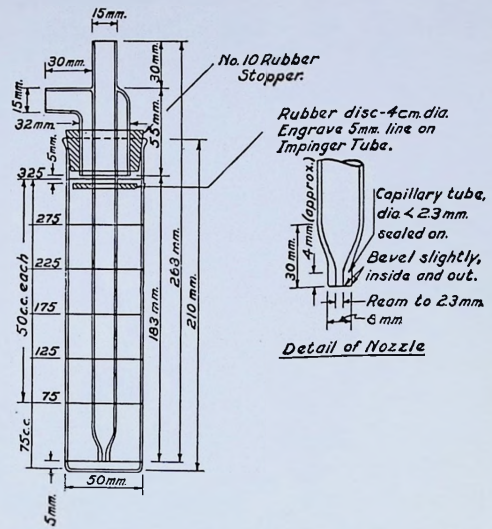


Fig. 32. Modified impinger assembly and detail of nozzle, with specifications for construction. (*Courtesy of Mr. Hatch et al.* [77] *and the J. Indust. Hyg.*)

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used today we might just as well use 1 liter or 20 cu. ft. per minute as 1 cu. ft.

The actual time of sampling in air which the fastidious might call doubtfully dusty, is about 40 to 60 minutes; in air which the plant superintendent thinks is clean but the dust expert questions, the sampling time is around 20 minutes; and in air which all admit is too dusty the sampling time is about 5 minutes and less. The sample, which will appear as a dusty emulsion, is transferred to a suitable flask, diluted to a known volume, and a known fraction is counted in a Sedgwick-Rafter cell, using the 16 mm. objective and ordinary lighting [63]. Recently Fehnel [117], Cummings [32], and Hatch and Pool [75] have reduced the labor of counting by substituting dark ground for the light ground lighting, Hatch increasing the magnification. The indirect lighting procedure greatly increases the sensitivity of the whole technic and enables one to include particles below the 3μ size which Gardner and Cummings [57] have shown to be so important.

If the dust under examination is rather soluble like limestone, marble, and dolomite, it is advisable to make the estimates by chemical or gravimetric methods and not to attempt counting or turbidimetric methods. The addition of liquids like alcohol reduces the solubility of some dusts in water but tends to increase the difficulty from foaming. Alcohol, however, may be required to prevent freezing.

Drinker suggested shortening the time for routine dust sampling by shining a beam of light through the impinger samples and measuring their turbidity in terms of the reflected light. Hatch and Thompson [76] have described a very practical routine plant method for comparing small samples taken by the Owens counter against standards of known dustiness.

Hazard and Drinker [78] have devised a recorder for use against all types of dusts and fumes. The particles are caught on a transparent moving picture film base and a light shone through the dusty film. The light transmitted is recorded and is proportional to the dust caught. This instrument, in addition to its recording

feature, can be set to ring alarms, light warning lamps, start motors and the like at any desired dust concentrations.

With any of the existing dust sampling methods it is difficult for the skilled technician to interpret or quantitate in the laboratory more than a dozen samples a day. For routine control of a dusty process quicker methods are greatly to be desired and for that reason the short-cut procedures and the recording device are suggested.

The great fault in all present dust sampling methods is that they represent conditions only for a comparatively brief time. They may miss entirely what Mavrogordato calls "dust floods" [113]—momentary excesses of dustiness—which are of great importance in the causation of silicosis. The experienced technician will, however, observe from the process, the general condition of the building, and the appearance of the men whether dust floods are likely. His report certainly should cover an estimate of the extremes to which the dustiness can go.

In interpreting dust samples the composition

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of the dust and not simply the quantity must be known. This composition, when possible, should include individual compounds or minerals and should not be simply a chemical analysis—a dust with 50 per cent total SiO_2 is not the same, physiologically, as one with 50 per cent quartz, which is pure SiO_2.

The physician can rarely diagnose silicosis without the complete occupational picture before him—nature of employment, physical examinations, x-ray, and dust analysis. The dust analyst, in turn, uses all possible short cuts to his answer, especially composition of the original rock or source of the dust. But he must base his findings finally upon the composition of dust which is air floated and respirable and not on the composition of the original source. For instance, Hurlbut and Beyer [84] showed that the percentage of sericite in foundry dusts increased rapidly on air settlement of dust samples because the sericite constituent was slowest to settle. The U. S. Public Health Service in their reports, always require petrographic or mineralogical analyses of impinger dust samples before stating

the nature of the dust breathed. Jones [90] has shown beyond argument that petrographic analyses of dust breathed is imperative before one can say whether or not the dust is harmful.

The present technic followed in making petrographic analyses is not easy and is made much more difficult by the fact that particle size is well below that ordinarily used in petrographic work. Knopf [99] has set forth a usable technic but the methods available are by no means finished and further work on this phase of the problem is badly needed.

Gases. The determination of gas concentrations in work places is distinctly the chemist's task and generally offers no specially new problem. Concentrations of hygienic significance, however, are usually measured in parts per million by volume and not in percentages, thus showing that the absolute concentrations generally are very low.

Gases most commonly sampled and recorded in connection with industrial medical problems are carbon monoxide [152], carbon dioxide, oxygen, hydrogen sulfide [150], hydrogen cy-

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anide [94], vapors from organic solvents like benzol [159], gasoline, or carbon tetrachloride [47]. Ocasionally chlorine, ozone, or oxides of nitrogen must be determined but the methods to be used are well established. Gases, vapors, and mists from heavy chemicals like sulfur dioxide [158], sulfur trioxide, hydrochloric, sulfuric, and nitric acids, and ammonia require no elaborate equipment or technic.

The heavy chemicals are determined conveniently in the impinger apparatus using two bubblers in series and appropriate solutions and indicators as absorbing media instead of water [15].

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nesium oxide, or doubtful like that from iron oxide fume from welding.

These reactions should be distinguished sharply from chronic cumulative effects such as may result from repeated breathing of lead fumes or manganese dioxide. In these, fume fever reactions occur, if at all, a few hours after exposure, but true lead or manganese poisoning occurs only after repeated exposures.

Metal fume fever is easy to prevent, first by local exhausts and second by dust respirators. Exposures are generally intermittent—during a pouring of molten metal or during the welding of some particular piece of metal. Processes of this sort are ideally adapted to the use of simple but effective respirators as described on pages 215–16. Drinker and others [43] showed that permissible doses of zinc oxide, and probably of other fever producers, were fairly definite and rather high compared to allowable dustiness for pure silica.

Generally we find attacks of fever today as a result of welding or cutting galvanized iron sheets or pipes. It has become rather rare in brass

foundries and in zinc smelting operations where it is well known to all engaged, particularly to the older employers. Lipkowitsch [108] does not believe that the modern electric welding processes for iron and steel offer a health hazard of any consequence. New steels with varying percentages of metals other than iron are, however, rapidly coming into use. It is advisable, therefore, to make certain that in new processes neither the fumes from welding nor from the coatings of the welding rods are harmful.

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