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OCCUPATIONAL DISEASES DEFINITION, INTERPRETATION OF COMPENSATION LAWS IN THE VARIOUS STATES

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Introduction

The Workmen's Compensation Law is a modern conception of the employer's liability to his injured workman. The earliest compensation law in this country was enacted in 1910 (the first New York Act, which was held unconstitutional in 1911), and the present compensation law of the State of New York was enacted in 1914. Massachusetts, New Jersey and Wisconsin followed in 1911, and many states followed later. The compensation laws confer many benefits upon injured workers and the dependents of deceased workers of a monetary character. Unlike the system of employers' liability which it displaced, the compensation system also confers upon the injured employee the right to medical treatment at the expense of his employer, and it imposes upon the physician certain duties and obligations. While medical treatment is a benefit inherent in the system, the ascertainment of medical facts and the expression of medical opinions based upon such facts become essential parts of the administration of the law.

It follows, therefore, that knowledge of the compensation laws, their fundamental principles and their characteristics, is a necessity for the physician who would undertake the care of cases coming within the jurisdiction of those laws. Medical knowledge and training are not the whole armamentarium of the industrial doctor. There are certain lay and legal concepts of the system that must be the concern of the doctor who would devote some part of his practice to injured workmen coming under the compensation law. It is to some of those lay and legal concepts that we are presently to address ourselves.

At the outset we may concede that generally diagnosis and therapeutic procedures are no different in compensation work than they are in strictly private practice. Perhaps in considering surgical measures in cases where there is a surgical choice, consideration may be given to the provisions of the compensation law and their effect under the circumstances of the case, and the vocational use of an injured member, but always the interest of the injured man is paramount and his welfare comes first. But diagnosis and therapeutic measures, as well as etiological considerations, must all be brought into complete harmony with the compensation law and the somewhat complex system that is constituted thereunder. Accordingly, let us see what this compensation system is.

"Compensation" is a common term that denotes the system of extending medical treatment, together with money benefits on account of actual or potential wage loss, as the direct responsibility of industry to its injured and disabled workers, and the widows and children or other dependents of deceased workers. It may be said that compensation is universal in the United States. The compensation system prevails now in all but one of the states; namely, the State of Mississippi, and it likewise prevails in the District of Columbia and most of the territorial possessions of the United States. Compensation is also provided by Federal Law for all longshoremen and harbor workers who are employed under maritime contracts of employment, and for all civilian employees of the Federal Government.

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It is important that the industrial physician be familiar with the requirements and provisions of the compensation law of the state in which he resides and practices. If he is in a maritime state, he should also have some knowledge of the U. S. Longshoreman's Compensation Act, and the principal variances between that law and the local state compensation law.

Characteristics of Compensation

While differing in many particulars, such as the schedule of benefits, the periods during which benefits are payable, procedures for adjudication and payment of claims, and the provisions for medical treatment, there are certain common characteristics of the compensation systems that prevail in all states. It may be well to note some of these common characteristics.

1. Compensation is payable for all disabilities to employees resulting from accidents arising out of and in the course of their employment, except intentional self-injury or injury due solely to intoxication.

2. Negligence of the employee is no bar to the right of compensation. Neither is the negligence of a fellow worker nor the assumed risk of the business any defense. This is really the keystone of the whole compensation system.

3. The compensation rate is based upon a percentage of the worker's wages or his earning capacity, with certain maximum provisions.

4. Compensation is payable periodically during disability or during a definite period stated in the law for such disabilities. Lump sum payments are the exception to the rule in some states under given circumstances.

5. Medical treatment is afforded the injured worker at the expense of his employer for varying periods of time, and sometimes with limits of amounts. New York is one of the few states where medical treatment is unlimited as to time and amount.

6. Security for the payment of compensation is required. This may be effected by insurance in the manner provided by law, or by self-insurance under the conditions prescribed in state laws.

7. Enforcement of the law by boards or commissions rather than through the courts.

8. In a number of states compensation is payable for disability or death due to occupational disease, as well as disability due to accident.

Medical Care

The industrial physician must be familiar with the medical provisions of the law in his state. State laws vary widely on this subject; in the manner of the selection of the physician to treat injured workers, as to charges for such treatment, and as to the time during which the employer is responsible for treatment. Unlike private practice, in compensation the relationship is not

solely that between doctor and patient. This cannot be too greatly emphasized. The employer enters that relationship, as do also the public authorities administering the law. Either directly himself, or through his insurance carrier, the employer must pay the cost of medical treatment. Similarly he, or his insurance carrier, must pay the compensation for the entire period of disability, which may be shortened or may be prolonged as a result of good or indifferent treatment.

Another consideration, the obligation of the employer to pay or the right of the claimant to receive compensation in a disputed case, may to a great extent depend upon the medical opinion of the attending physician. Here come in questions of etiology, pathology and diagnosis, and often the doctor's early observations and the completeness of his records become determinative. The doctor has only one obligation, namely, to serve the truth. He should not be the employer's doctor nor the claimant's doctor, but he should be witness to the truth as he sees it. His opinion should not depend upon his interest in the payment of his bill for his services nor the fee that he is to receive for his testimony. He should be definite in his opinion, not vague, and he should state clearly his facts and observations and not be afraid to say he does not know, if they are the facts. And if he expresses an opinion and believes he is right, he should be willing to stand up to it and maintain it.

In New York, the period of time during which treatment may be rendered at the expense of the employer is not limited in the statute. The law provides that the employee is entitled to treatment "for as long a time as the nature of the injury and the process of healing may require." That, however, is not the prevailing rule outside of New York. In most states a definite period of time during which treatment may be required is set forth in the law, or a maximum cost in dollars may be prescribed. It is necessary that the industrial physician be familiar with those provisions of his local law.

Generally speaking, compensation laws cast upon employers the duty of furnishing and providing medical treatment for their injured employees, but the employer is relieved of that obligation when his employee refuses, without good cause, to accept the services of the employer's doctor. In some states the employee may transfer his treatment to a doctor other than the one furnished by the employer if he obtains the consent of the administrative authority. That consent will generally be granted only for good cause.

But here in New York, we have a unique system. The original law which placed on the employer the responsibility of designating (often upon the recommendation of his insurance carrier) the physician to treat his injured employee, if he was to be held liable for payment of the doctor's charges, was amended in 1935 to accord to the injured employee the right to choose his own doctor, without interference from his employer or insurance carrier, provided the doctor had received from the Industrial Commissioner authorization to treat cases under the Compensation Law. The authorization of physicians was made by the Commissioner upon recommendation of the County Medical Society of the county in which the doctor resided. By an amendment of the Law passed at the last session of the Legislature, the duty of qualifying physicians under this law was imposed upon a newly created Medical Practice Committee of three physicians, appointed by the Commissioner, in the four largest counties in the City of New York. Elsewhere in the State, that duty still rests upon the Medical Societies. Doctors qualified and authorized to treat compensation cases are arranged upon the panel by the Medical Practice Committee or on recommendation of their respective County Medical Societies, according to their particular qualifications and their fields of practice. Each doctor, when authorized,

is assigned a serial number which must follow his signature on all reports in compensation cases, and to the serial number is prefixed a code letter or letters designating whether the doctor is authorized as a general practitioner or a specialist in one or more fields. No doctor may treat a case, other than first aid or emergency treatment, that calls for qualifications outside of his assigned field. General practitioners, for instance, may not do operative surgery, nor may a specialist do a branch of medicine in which he has not given a medical report and opinion in the past. The law also provides that a doctor to which he has agreed to limit his practice.

The employer may not even recommend to his injured worker that he use the services of a particular doctor unless the employer has previously recommended a doctor, and even then the request must be in writing.

Medical Councils

Since contract and price do not enter into the relationship of the doctor and the employer, it was necessary to provide for the establishment of a fee schedule to which doctors must conform in compensation work. This schedule was established by the Industrial Commissioner after hearing all interested parties. The schedule established is a minimum fee schedule, below which no doctor may charge, but higher fees in a particular case of unusual difficulty may be arranged by agreement between the doctor and the employer or his insurance carrier.

To protect the doctor and to provide a speedy and impartial means of adjusting medical bills, as to the reasonableness of which dispute has arisen, two methods of procedure have been provided to pass upon the matter of fairness and the necessity of treatment. Both of these procedures call for such determinations to be made by doctors. In the four large counties of New York City, the determinations are made by the newly created Medical Practice Committee, composed of three doctors appointed by the Industrial Commissioner. Elsewhere in the State, the determinations are made by an arbitration before five doctors, two of whom are appointed by the President of the local County Medical Society, and two appointed on behalf of the employer or his insurance carrier by the Compensation Insurance Rating Board and a fifth doctor appointed by the Industrial Commissioner. Over the years, arbitrated medical bills have numbered fewer than 1,000 a year, as contrasted with the estimated number of medical bills of more than a million a year, while the total amount in dispute has not exceeded \$75,000 a year out of total medical expenditures in the State in excess of \$15,000,000 a year.

The New York law provides in great detail for many of the procedures necessary in connection with medical treatment and the relations between doctor, patient, employer and insurance carrier. Time does not permit of discussion of these various details.

Basis of Compensation Benefits

Another characteristic of compensation laws, as distinguished from the old employers' liability laws, is the fact that all compensation is related directly to disability and loss of earning capacity. A percentage of wages, with generally some fixed maximum, is payable as compensation during actual or potential disability. For instance, in New York two-thirds of the average weekly wage is paid as compensation, with a maximum rate of \$28.00 a week and a minimum of \$12.00. These maximum and minimum rates were increased this past year by one

Legislature, effective June 1, from \$25.00 and \$8.00 respectively. The maximum rate is provided for a period of one year, as an emergency to meet the increased cost of living on account of the war. All disabilities may be divided into four classes, disregarding death benefits in this consideration, viz:

1. Permanent total disability
2. Permanent partial disability
3. Temporary total disability
4. Temporary partial disability

A disability in one class may progress from one classification to another, as for instance, a temporary total disability may become one of the permanent disabilities, or a total disability may become with improvement only a partial. It is important that the industrial physician keep in mind these classifications.

Permanent total disability is that condition which completely incapacitates a person from working and the condition is not amenable to improvement. As an illustration, the loss of two hands or two feet or two eyes, or any two of them, is declared to be permanent total disability. From a loss standpoint, this is usually the most costly type of disability. In adjudging disabilities the loss of use of a member, as by paralysis or deformity, is considered the same as the actual loss of the member by amputation. And the partial loss or loss of use of a member is proportioned to the total loss.

Permanent partial disability is that type of disability that, while permanent in character, is not wholly disabling. In this category are found the amputations of single members or parts of members, and the loss of use of such members or parts. These losses are commonly referred to as "schedule losses," since the evaluation of the disability is dependent upon a schedule of such losses set out in the law. The determination of these cases does not depend upon actual loss of wages so much as it does upon potential loss. If a man loses two fingers of his hand, his loss of time from work may be but a few weeks, perhaps three or four, but his potential loss is measured by the statutory schedule that sets up arbitrarily a number of weeks of compensation to pay him for his present loss and any future loss of earnings that he might sustain. These schedule losses run all the way from 15 weeks for the loss of a little finger to 312 weeks for the loss of an arm. These schedule values vary in different states, there being little uniformity of opinion as to the value of a thumb or an eye or any other member. Permanent partial disability also includes cases in the so-called "non-schedule" class, in which a condition is permanently disabling to some extent. In this class of cases may be included back injuries that are permanent, but only partly disabling, or head cases following fractures or concussions. These cases are settled not on the basis of a schedule, but on a reduction of the compensation rate, on the theory that the worker has some earning capacity.

Temporary total disability is by far the most numerous class of disabilities and is that condition that prevents the injured person from working, but the condition can be regarded at the time merely as temporary. This is generally the case with any injury that compels a man to stop work while he is undergoing medical treatment. During the time that medical treatment is indicated the condition is always classified as temporary, although it may later be classified as a permanent disability.

Temporary partial disability is that condition that, while not permanent, is not even totally disabling. That is illustrated by the type of case where the injured person is advised to return to light work, often receiving treatment outside of working hours. Of course, the compensation rate is properly adjusted to take account of the partial earning capacity.

The determination of the extent of disability within these classifications is the function of the administrative officer, but he must at all times be guided in making his findings by the informed and expert advice of the doctor.

Death benefits. Compensation in case of death is known as "death benefits" and is payable for various periods and at various rates, depending upon the individual state law. In New York, for instance, compensation to a widow is payable during her life, unless she remarries, in which case it terminates with the payment of two years of compensation from the date of remarriage. The widow receives 30% of the husband's wages, taken at a maximum of \$1950 a year. Compensation is also payable for each child under the age of 13 years, until he becomes 18, at the rate of 10% of the wages. In the case of the death of the mother or her remarriage the compensation rate for the children is increased to 15%. The aggregate of compensation in a death case may not exceed 66-2/3 per cent of the wages. In case the total compensation payable to a widow and children is less than the 66-2/3 per cent, benefits may be paid up to the maximum to brothers, or sister, father, or mother, or grandparents, if dependent for support in whole or in part upon the deceased workman. Dependency is usually conclusively presumed in the case of a widow and children. A husband may be awarded compensation for the death of his wife by an industrial accident, provided he was dependent upon her for support.

Medical Reports

A word on the subject of reports. Under the compensation systems of all states, the physician is an essential and integral part of the administrative machinery. No compensation case is adjudicated without a doctor somewhere in the picture. With the attending or consulting physician, careful observation and record are of the utmost importance. Therefore, reports of physicians take on a high degree of probative force and importance. Particularly under any free choice loss such as that in New York, prompt, full and periodic reports are necessary. A first report in considerable detail is essential not only to the administrative official, but even more importantly, it seems to me, to the employer or his insurance carrier. When an accident has occurred and is reported by the employer, both to his insurance carrier and to the administrative authority, and a prompt report by the attending physician is rendered indicating a disability related to the injury, prompt payment of compensation by the employer or his insurance carrier should, and usually does, follow. The doctor's report, therefore, is in the interest of the workman, the employer, the insurance company, and the general public. In the interest of truth and effectiveness, reports should be full, give the history as obtained from the injured person, a clear description of the injury, the diagnosis and where possible, the prognosis. If observation has been properly made, note should be made of any pre-existing abnormality or deformity and record and report should be made of that antecedent condition. Mention of it in the report may save time and trouble later on when some question with regard to such condition arises. Doctors are prone to complain of the paper work involved in compensation cases. It must be evident that the paper work is utterly important and a necessary part of the system. Careful reports frequently relieve the doctor of the necessity of attending hearings and testifying.

Continuing periodic reports, though much less detailed, are also necessary for the smooth operation of the law. The doctor should promptly report the discharge of a patient and state whether he is able to do his regular work or only light work, and whether there is any permanent condition. In New York, by a law enacted at the 1944 session of the Legislature, doctors are required to report directly to the Industrial Commissioner, and failure to do so may render the doctor subject to disciplinary procedure, even to the extent of revocation of his authorization to treat compensation cases. Formerly it was the practice of many doctors to send their reports in duplicate to the insurance company, which then filed one with the Commissioner. Now, however, the doctor must send one copy to the Commissioner and another to the employer or insurance carrier. It is utterly necessary for the insurance carrier to have copies of all reports for upon them the company depends to determine action to be taken relative to the injured person's compensation. But it is also obligatory for the doctor to file one directly with the Commissioner, even though the doctor be examining on behalf of, or at the request of the insurance company or the employer.

Occupational Diseases

In considering the coverage for occupational diseases, the extent of such coverage and the methods of coverage vary greatly in different states. For instance:

(a) Some state laws limit occupational diseases to certain specific industries or to certain specific diseases that predominate in the industries of the state. Illustrations of these are mining operations in certain states or the crushing, smelting and refining of metals and metal ores. These are limited laws enacted to meet certain well-known and recognized conditions.

(b) Some state laws provide coverage by lists of named diseases and by schedules of conditions in which they are found in the industries. Such was the law of New York for many years, and such are the laws of many states today, including among others New Jersey, Maryland, Minnesota and Rhode Island. The so-called schedule method of coverage has the great merit of certainty... informative to the employee as to the conditions for which he may make claim, and also to the employer as to those conditions for which he may be held liable.

The schedule method of coverage has also the merit of simplicity. Objection is made to that method, however, that in these days of changing industrial processes and methods, the schedule may not include some diseases that are characteristic of new substances that were not commonly used when the schedule was written and that, therefore, some person may be deprived of compensation for a condition that is truly occupational but not named in the law. This, of course, may happen even though the law be amended and enlarged from time to time to keep it abreast of changing industrial conditions.

It is suggested that a schedule law could be kept up with current conditions by providing that some administrative board or body shall have authority, when the Legislature is not in session, to add diseases to the schedule after a hearing and proof that some disease or condition not named in the law is actually a hazard of workers in some industry. When, therefore, some new chemical or substance comes into industrial use and creates a disease hazard it could, by prompt action of the authorities, be made an occupational disease.

(c) Some states provide in their compensation laws for the inclusion of all occupational disease by vague and uncertain language in the definitions in the statute. The evident purpose and intent of such definitions are to make the law all-inclusive of occupational diseases, but without any attempt to be specific as to the conditions covered thereby. An illustration of this sort of un-

certain language is contained in the Massachusetts law which is declared to cover all "injuries," whereas most laws cover all "accidental injuries." The omission of the word "accidental" has been interpreted by the courts to make the law applicable both to injuries by accident and to injuries by disease, but the very broad possibilities of the definition have seemed to cause the administrative authorities of that State to exercise extreme care lest compensation be extended to diseases and conditions not definitely and certainly the responsibility of the employer.

In such cases it would seem desirable to have some sort of limiting definition in the statute. Such a limitation is found in the compensation laws of some states, which declare that the term "occupational disease" shall mean only a "disease that arises out of conditions that are characteristic of and peculiar to the employment or process in which the worker is employed." This, while not a satisfactory definition, is at least an intelligent limitation upon any tendency to make awards for all manner of diseases that may not be characteristic in any way of the employment, but that may be asserted to have arisen in the employment by reason of some merely incidental condition.

The very difficulty of defining "occupational disease" is perhaps best illustrated by the definition contained in the statutes of Illinois and Indiana. Both states employ the same exact wording. That definition reads as follows:

"Sec. 6. In this Act the term "occupational disease" means a disease arising out of and in the course of the employment. Ordinary diseases of life to which the general public is exposed outside of the employment shall not be compensable, except where the said diseases follow as an incident of an occupational disease as defined in this section.

"A disease shall be deemed to arise out of the employment, only if there is apparent to the rational mind upon consideration of all the circumstances, a direct causal connection between the conditions under which the work is performed and the occupational disease, and which can be seen to have followed as a natural incident of the work as a result of the exposure occasioned by the nature of the employment and which can be fairly traced to the employment as the proximate cause, and which does not come from a hazard to which workmen would have been equally exposed outside of the employment. The disease must be incidental to the character of the business and not independent of the relation of employer and employee. The disease need not to have been foreseen or expected but after its contraction it must appear to have had its origin in a risk connected with the employment and to have flowed from that source as a rational consequence."

You will note that his definition appears to be all-inclusive in the first sentence, but then it is qualified by language that makes an exception of "ordinary diseases of life." It might be pertinent to inquire, what are the ordinary diseases of life that are excepted? Certainly no one, be he doctor or lawyer, is so wise as to be able to say with certainty what the definition means in certain cases.

(d) Some compensation laws, by explicit declaration, are intended to cover "any and all occupational diseases." That is the rule in New York that has been effective since 1935. The first New York law including occupational diseases was enacted in 1920, such occupational diseases being listed by name and description of industrial process. To the very extensive schedule of diseases in the law, the Legislature added an additional item known as Paragraph 28, that reads "any and all occupational diseases." This has raised many nice legal

problems and the status of diseases under the New York law is far from certain or settled.

In the leading case on the subject of occupational disease, the New York Court of Appeals declared in the language of Judge Finch:

"The contention is made that his paragraph 28 extends the Workmen's Compensation Law so as to make it applicable to any disease arising out of and in the course of the employment, just as it is applicable to any accident arising out of and in the course of employment. This in substance would make workmen's compensation the equivalent of life and health insurance. ***

"Manifestly, it was not the intention of the Legislature to change the Workmen's Compensation Law from a statute affording protection to employees against the hazards of their occupation to a law insuring against diseases which might happen to be contracted while the employee was engaged in his occupation. ***

"Thus an occupational disease is one which results from the nature of the employment, and by nature is meant, not those conditions brought about by the failure of the employer to furnish a safe place to work, but conditions to which all employees of a class are subject, and which produce the disease as a natural incident of a particular occupation, and attach to that occupation a hazard which distinguishes it from the usual run of occupations and is in excess of the hazard attending employment in general. Thus compensation is restricted to disease resulting from the ordinary and generally recognized risks incident to a particular employment, and usually from working therein over a somewhat extended period. Such disease is not the equivalent of a disease resulting from the general risks and hazards common to every individual regardless of the employment in which he is engaged."

(Goldberg vs. 954 Marcy Corporation, 276 N.Y. 313)

It is clear, I think, from this declaration of the court that not all diseases occurring to workmen are occupational diseases within the meaning of the law, and the court has explicitly stated that the occupational disease provisions of the Compensation Law are not to be construed as providing life and health insurance for the workers at the expense of their employer.

Time does not permit of extensive discussion of court decisions on occupational diseases nor of the various ramifications of the administration of that law. It is important, however, that the industrial doctor familiarize himself with the laws of his state on the subject of occupational diseases. As we have indicated, there may be wide variances in the laws of the different states. In this field perhaps is demonstrated the greatest difference between the private practitioner and the industrial doctor. Upon correct diagnosis much depends. Etiology plays in this field a most important part. Differentiation between conditions produced by industrial processes or by toxic substances used in or produced by the industry and those conditions wholly unrelated to the industry, is essential. This requires a knowledge of the industrial processes surrounding the worker and the substances with which he comes in contact. Upon the doctor's knowledge and skill may depend the right of a disabled worker to obtain the compensation intended by the law or the obligation of his employer to pay the benefits called for under the law. This is indeed a heavy responsibility.

So complex are the medical questions that arise from time to time in occupational disease cases that it has long been urged by many that a fair and intelligent administration of the law requires an expert medical board to pass

upon them. It is interesting to note that the law of Maryland makes provision for such a board of three physicians, appointed by the Governor from names recommended by the Council of the State Medical Society and by the deans of the two medical colleges in that State. That board has now been functioning for several years and it is making an enviable record, marked by utmost fairness, intelligence and courage. Such boards may well point the way to the administration of such laws in the future.

Silicosis

Another phase of occupational diseases has to do with silicosis and asbestosis, or other lung disease due to the inhalation of harmful dust. This is a subject for the complete understanding of which an entire course would scarcely suffice.

Silicosis and asbestosis are the recognized and demonstrable conditions of fibrosis in the lungs, resulting from long years of exposure to the dust of free silica or asbestos in very finely divided particles. While disabling cases of silicosis resulting from a relatively short exposure - say two or three years - have been known, they are the rare exceptions, and it is the rule that silicosis does not result in disability without exposure of ten years to twenty-five years, unless complicated with tubercular infection. These long exposures are rarely in employment by the same employer. They commonly result from exposures in several different employments. When disability occurs, however, the liability for compensation is placed on the last employer by whom the worker was employed in such exposure. This was in the beginning felt to be very unfair, since the employer made liable for compensation benefits might have been the employer in whose employment the man was the least exposed. Moreover, at the time of the enactment of any law bringing silicosis under the act, a tremendous amount of exposure with its resultant effects has already taken place before the effective date of the new law, and to place that already accrued burden on the employers on that date would be more than many of them could carry financially. To meet the situation of the already accrued liability and permit industry to "catch up," so to speak, New York and several other states enacted laws limiting the recovery for silicosis or other dust disease to an absolute minimum, as of the date of the statute, and then increasing the maximum benefits on a sliding scale as time passes and the workers continue in dusty occupations. In New York, for instance, a maximum limit of \$5,000 in any silicosis case was set. That maximum was reached a few months ago, and thereupon the Legislature amended the law fixing a maximum of \$6,500 on all cases arising after April 1944. The progression from an original maximum benefit of \$500 to the present limit has been accomplished without too great a disruption of industry and without driving employers in the dusty trades out of business, which at one time seemed likely to happen and which would have resulted in widespread unemployment.

The relief benefits in silicosis are not the greatest part of the law. Making the dusty trades subject to compensation has had a very marked effect upon preventive measures, and has greatly stimulated the engineering and safety programs of this branch of industry. Already the effect of dust control is having a most beneficial effect on the health of the workers in dust. In New York, because of the peculiar medical problems involved in dust disease determinations, a special medical board was set up of three experts in this field, and to this expert medical board are referred all medical questions involved in particular cases.

Security for Payments

Another characteristic of compensation laws is the fact that they all require security for the payment of compensation. As we have seen, compensation is payable periodically as it becomes due. Many cases of long-term disabilities or death benefits call for periodic payments spread out over years. It is not sufficient to leave the claimant or beneficiary at the risk of the continued business life or solvency of the employer. His security must be supplemented by the assets of a recognized insurance carrier. Through the medium of insurance, the claimant is assured that the money to pay his claim to maturity is set aside and reserved, and will always be available to him when due. The employer in effect really pays for his accidents in advance, and, therefore, is not so immediately concerned with the ultimate cost. Also, he is relieved of the devastating impact of a serious loss or catastrophe.

Moreover, through this medium, the cost of accidents is spread out over the whole of industry and can be accurately measured. Every dollar paid in compensation and every dollar paid in medical and hospital charges enters into the statistics of the cost of compensation and has, therefore, a very direct bearing upon insurance rates, and the cost to industry. Insurance, in a very real sense, is the agent of industry and the trustee of its moneys paid for compensation. Whatever losses are incurred and paid by insurance must eventually be made good by industry through the medium of premium rates. It is not my purpose to discuss the problem of insurance or the principles underlying it at any length, but it is so completely identified with the compensation system that some understanding of its function is necessary for the physician practicing in the industrial field.

The method of securing the compensation obligation varies in different states. Insurance carriers are of three general types, namely, state funds, stock insurance companies and mutual insurance associations or companies. In addition, in most states employers who are able to prove their financial ability, may be permitted to assume their own compensation obligations under a system known as "self-insurance." Generally, self-insurance is conditioned upon the employer complying with certain rules and requirements as, for instance, in New York, where in addition to his agreement to pay the compensation benefits and medical cost, the employer must deposit securities with the Industrial Commissioner in an amount sufficient to guarantee the payment of all claims should the employer default in any payments.

State funds are of two types--one the competitive fund and the other, the exclusive or monopolistic type. The competitive fund, as in New York, exists along with stock and mutual insurance, and the employer has the right to choose whether he will insure in the State Fund or with a private carrier. The exclusive or monopolistic state fund exists in seven states. In those states stock and mutual insurance is excluded and employers have no choice but to insure in the state fund. In such cases the State Fund makes its own premium rates, and may act arbitrarily in many matters, because of the lack of any competition.

Administration

Another feature of the compensation system is the fact that the law is administered and claims adjudicated by administrative officials rather than by the courts. In two or three states the law is enforced through courts, but the overwhelming rule is administration by boards and commissions. Payment of uncontroverted claims is made the immediate concern of the employer or his insurance carrier, while controverted claims are heard before and decided by the

administrative authority. This makes for speedy hearing and determination, for informality of procedure with the very minimum of technicality. From the administrative decision, an appeal is usually allowed to the courts. Only serious cases are likely to come before the courts on appeal, and generally the courts have jurisdiction to review decisions only on questions of law, the findings of fact by the administrative officers being final and conclusive.

Conclusion

In conclusion, it should be evident from the foregoing that the role of the physician in compensation is most important, and his records and reports highly significant. While it is evident that an injury will be treated the same and heal the same, whether the injury occurred in the employment or out of it, and the same procedures and skill will be required in either case, still there is a wide difference in the manner in which the case will be observed and recorded. Any physician who would seek to do industrial work, must learn and observe much that he will not learn in the curriculum of his medical school. The subjects that I have here sketched in outline should be given intensive study by the doctor, and will greatly aid him as he goes forward in this most important, useful, and interesting field.

CRITERIA FOR DETERMINING THE PRESENCE OF AN OCCUPATIONAL DISEASE

Irving Gray, M. D., F.A.C.P.

The historical background of industrial and occupational diseases (1) can be traced back to the time of Hippocrates. However, it was not until the year 1700 when Ramazzini (2) published his memorable treatise on the effects of occupation in relation to the health of the worker that the medical profession had vividly called to their attention the importance of industrial hazards in relation to health and disease. Since then, there has been an interesting literature on the subject of occupational diseases. Terms such as "disease arising out of employment," "peculiar to the occupation" and "due to the nature of the work," have all been used to define the term occupational disease.

The most commonly encountered occupational disease is dermatitis. It has been estimated that the proportion of dermatitis to all other occupational disease is 69% (3). The criteria for establishing a diagnosis of occupational dermatitis is excluded from consideration in view of the fact that this subject will be discussed by another speaker in this symposium.

As physicians, we recognize the fact that certain criteria must be present before a diagnosis of occupational disease can be established. These criteria may be listed as follows:

- I. History of exposure (usually prolonged) to a substance that has been proven to be injurious.
- II. The presence of distinctive clinical symptoms and objective findings.
- III. Corroborative laboratory data.

- I. History of exposure (usually prolonged) to a substance that has been proven to be injurious:

The symptoms of occupational disease may be insidious in onset. It is generally recognized that exposure over weeks, months, and years, is necessary before there are either clinical symptoms, physical findings or corroborative laboratory data to justify a diagnosis of occupational disease. Experimental work among animals usually forms the basis as to whether or not a substance is injurious. Clinical experience has been, and is, however, the important guide in determining whether or not any chemical is injurious to the health of the worker. Poisoning by the absorption of lead is probably the best example of an occupational disease and can be illustrated by this case report:

A male adult, 42 years of age who had been an iron worker and "burner" for about fifteen years, was employed in the construction of a bridge. The steel girders used in the bridge were painted with red lead. While driving the rivets thru the girders, the red lead was melted and the fumes escaped. In the construction of the tower, a situation was created comparable to an enclosed chamber. In previous years of employment as a riveter and burner, this individual was not exposed to the absorption of lead in any enclosed areas. Approximately one month after the onset of work in his present occupation, the patient began to complain of a metallic taste in the mouth, nausea, vomiting and increasing abdominal colic and constipation. The essential findings on physical examination

were the pallor and pigmentation of the gums. The essential findings on laboratory examination were an abnormal amount of lead in the urine (0.22 mgm. per liter of urine) and the presence of 40 to 50 stippled cells per fifty microscopic fields. Approximately one hundred men were employed in the same occupation and doing the same job under like circumstances, and thirty-five (4) developed signs and symptoms of lead intoxication, corroborated by laboratory findings. The diagnosis of occupational disease in this group is relatively simple. The criteria as stated above were present. All of the patients had

1. A history of exposure to a substance that has been proven to be injurious.
2. There were characteristic clinical symptoms and objective findings.
3. Corroborative laboratory data (abnormal lead metabolism) was present.

The symptoms of acute lead poisoning are distinctive. In acute lead poisoning, there is always evidence of an abnormal lead absorption and lead metabolism which can be verified by quantitative examination of the urine and of the whole blood. There may or may not be a varying degree of anemia, but there is usually an abnormal amount of stippling of the red blood cells.

Individuals exposed to the absorption of toxic materials may develop signs and symptoms suggestive of an occupational disease and nevertheless have an illness which is entirely unrelated to the occupational hazard. The following case citation will illustrate such an instance:

A male adult, 40 years of age, was employed as a garage worker for a period of ten years when he developed unilateral convulsive seizures affecting the right upper and lower extremity. Examination of the spinal fluid and of the oculi fundi revealed no abnormalities. A diagnosis of tumor of the brain was suspected. There were no symptoms referable to any other organs or systems other than the central nervous system. Because of the fact that the patient worked in a garage and handled gasoline containing lead (tetra ethyl), a diagnosis of lead poisoning was suspected. Quantitative studies of the whole blood urine for lead were reported as containing amounts of lead that were distinctly abnormal. A diagnosis of lead encephalopathy was made, based upon the history of occupational exposure and on the finding of abnormal amounts of lead in biological materials. There investigation and subsequent studies revealed the fact that the laboratory reports of abnormal amounts of lead in the urine and blood were incorrect. The normal amount of lead in a liter of urine may vary from zero to 0.1 mgm. of lead. The normal amount of lead in the whole blood may vary from zero to 0.06 or 0.1 mgm. per 100 grams of whole blood. Inasmuch as we are dealing with infinitesimal amounts of lead in the biological materials, the greatest care must be exercised in the chemical determinations. The chemicals used in estimating the amount of lead in any given specimen may contain more lead than was present in the original specimen to be examined. In the case under discussion, it was conclusively proven after numerous control studies and other investigations, that the lead in the urine and whole blood was well within normal limits. The diagnosis of lead poisoning as a cause of the convulsive seizures was challenged. We are cognizant of the fact that there may be pathological lesions in the brain causing numerous symptoms including convulsive seizures that cannot be recognized or proven to be present even after the most meticulous and scientific study. In the case under discussion, subsequent events indicated the necessity for surgery. The patient was operated on and a brain tumor removed which was followed by complete recovery.

The lesson to be learned from this case is quite obvious. Although lead may

affect the nervous system, it does not necessarily follow that neurological symptoms that cannot be explained on any other basis are necessarily due to the occupational hazard. At no time in this case in question was there a history of abdominal colic, nausea, vomiting, arthralgia, etc. The clinical symptoms were not characteristic of the occupational disease in question. The patient had a history of prolonged exposure to the absorption and there were no abnormal amounts of lead but as proven subsequently, there was no anemia, no stippling of the red cells, and there were no abnormal findings of lead in the biological materials. Even if we assume that this patient had had abnormal lead absorption, certainly, neither the lead absorption nor any leading poisoning played a part in the origin of the brain tumor, its development or subsequent clinical course which caused the patient to stop working and seek medical care.

The importance of obtaining a complete and detailed family and past history, especially of previous occupations, cannot be overemphasized. The symptoms of occupational disease may be insidious in their onset and run a chronic course. The need for a differential diagnosis especially in any bizarre case may require all laboratory aids and special studies. We know there is no parallelism between symptoms and disease. There are instances where psychosomatic or functional disorders may produce symptoms and after complete physical examination and laboratory examination, patients are found to be entirely free of disease. It is well recognized that organic disease may exist without clinical symptoms. Hypertension, nephritis, diabetes, gastro-duodenal ulcer, disorders of the hemopoietic system, broncho-pulmonary system, skeletal system, etc., may exist without producing clinical symptoms. In the course of a routine preemployment or pre-placement or health examination, disease may be discovered of which the individual is entirely ignorant. It becomes clearly manifest that since there is no parallelism between disease and symptoms, that before arriving at a diagnosis of occupational disease, thorough and careful medical examination is necessary in every instance. These "silent" diseases enumerated above may exist for many months or years until some incident brings that patient to the office of his physician. An industrial accident or the question of an occupational disease causes the patient to seek medical care and then for the first time, some constitutional or underlying disease unrelated to occupation, is discovered. The necessity of an adequate pre-placement examination becomes evident and its importance can be readily appreciated when we are dealing with individuals who may in the course of their work be exposed to the absorption of injurious substances that may produce disease. The examining physician has the responsibility to determine whether the patient's illness is due solely to the occupation or only partially to the occupation or whether the illness is entirely unrelated. If an occupational hazard is known to exist, the symptoms and abnormal findings in any given case can be properly evaluated when the physical condition of the patient prior to employment is known. Thorough physical examination, X-ray examination of the chest and a blood Wassermann should be a routine procedure. Where indicated, blood studies or other laboratory examinations may be essential both prior to employment and during the regular employment. Individuals exposed to the absorption of inorganic dust containing silica or asbestos require periodic X-ray examination of the lungs. The hazard of a specific occupation is the determining factor as to the type of medical survey necessary in order to recognize the earliest manifestations of industrial illness. The incidence and severity of illness in any type of industry depends not only upon the control and prevention of disease but also upon its early recognition.

In reviewing the special bulletins of the New York State Department of Labor of the years 1932 to 1939 (5), that section which deals with occupational disease reveals the fact that most common occupational diseases are Dermatitis and those due to the absorption of metals (lead, arsenic, mercury, etc.) and the absorption of solvents (derivatives of benzol and petroleum). To these substances, in recent years, may be added the absorption of the non-organic dusts such as silica

and asbestos. It is clearly evident that only after prolonged exposure to the absorption of these substances that signs and symptoms of occupational disease become evident. In those cases where compensation has been paid for lead poisoning, the absorption of this heavy metal has occurred over a period of years before the specific occupational disease manifested itself. However, the inhalation of lead dust and lead fumes may cause lead poisoning within several weeks to several months. Experience has shown that as with lead dust and lead fumes, so too, with the solvents, a poisoning may occur within a period of several weeks or month. The mode of absorption is frequently the determining factor as to the appearance of toxic symptoms. When there is absorption of injurious substances thru the lungs or thru the skin, manifestations of toxicity may become apparent within several weeks after contact. The degree of concentration of the injurious substance in the working atmosphere is the determining factor as to the time interval between the onset of work and the occurrence of the occupational disease. In silicosis and asbestosis, it is well recognized the period of exposure to the inhalation of the dust may take several years before clinical symptoms arise. The common denominator in all occupational diseases aside from the question of the specific hazard is the period of time or employment.

II. The presence of distinctive clinical symptoms and objective findings.

It is reasonable to expect that when an occupational disease is under consideration that certain clinical symptoms and objective finding will be present. By virtue of accumulated medical experience the physician today is cognizant of the clinical symptoms that are usually associated with occupational diseases. While there may not be all of the signs and symptoms present in any given case, it is reasonable to expect that a patient having an occupational disease will have the symptoms and signs that are usual to the disease to a varying degree. By reason of the fact that occupational disease is associated with distinctive clinical symptoms and objective findings, most definitions of occupational disease include the term, "peculiar to" or "characteristic of" the employment.

As an illustration of distinctive symptoms and findings in an occupational disease, the following instance of benzol poisoning is cited:

A male adult, 43 years of age, had worked as a pressman on black and white presses for a period of 25 years. There was no hazard in this type of employment. Some three to four months after he began working on multicolored presses where he was exposed to a variety of colored inks and the inhalation of fumes of benzene (benzol), the patient began to complain of increasing weakness, black and blue spots of the skin in various parts of the body, loss of weight and strength, with bleeding from the nose and gums. The concentration of benzene vapors in the work-room air ranged from 50 to 70 parts per million parts of air. The patient was admitted to the hospital with a diagnosis of benzol poisoning. Blood studies revealed a pronounced anemia with a marked leukopenia and a thrombocytopenia. The white blood cells when the patient was first examined were 1,600 per cu. mm with a pronounced lymphocytosis (83%). There was progressive leukopenia and increasing anemia with diminution of the platelets. Despite numerous blood transfusions, the patient expired. Post mortem examination revealed a complete aplasia of the bone marrow.

In this case cited, there was a history of proven exposure to a substance that is injurious. We also note here the characteristic clinical symptoms and objective findings as a result of the aplasia of the bone marrow due to benzol.

There is a depression of the red cells, the white cells and the megakaryocytes or platelets. As a result of the depression of all of these three elements of the blood, there is anemia with weakness and loss of strength and weight, and as a result of the decrease to absence of platelets, there is bleeding into the skin and all of the cavities of the body.

At this point, it seems advisable to stress the importance of the fact that the industrial physician should be cognizant of the hazards of any materials used in a specific industry. It seems wise that a program of education relative to the hazards in specific industries be instituted so that the employer group may realize the dangers inherent in occupation. The speaker has had the opportunity of examining a dozen or more men who for many years has been engaged in the rubberizing of cloth. Various solvents were used including such chemicals as ethyl ketone, toluol, gasoline, and ethylene dichloride. With the speeding up of activities during the war, the concentration of these materials in rubberizing balloon cloth was increased with the result that signs and symptoms of intoxication developed among the workers. When attention was called to the employer that possible substitutes could be used that were harmless, these substitutes were introduced and ventilation and other safety measures were improved with the result that there was no further illness. The speaker further had the opportunity of seeing half a dozen cases of methyl bromide poisoning some two years ago. The employees were filling ampules with methyl bromide (which was to be used as a delousing agent among the soldiers). The employer had no knowledge whatsoever of the extreme toxicity of this solution which readily vaporizes and becomes a gas and may have irreversible action on the central nervous system. One of the workers died as a result of methyl bromide intoxication. There were approximately twelve people engaged in the work and the employer knew absolutely nothing about the toxicity of the chemicals which the workers handled. It would certainly seem advisable for the chemical concerns that manufacture and distribute these various products, who are undoubtedly familiar with the hazards, should have the employer groups or the workmen educated as to the possible potential dangers when working with substances that may prove injurious.

The solvents that are derivatives either of petroleum or coal tar when absorbed may produce toxic symptoms by virtue of their affinity for certain organs in the body. Benzol, a derivative of coal tar, when absorbed, produces irritative phenomena of the bone marrow to the end that aplasia with death may occur as evidenced by the case presented above. Carbon tetrachloride, the most frequently used solvent, is a derivative of petroleum. When absorbed, carbon tetrachloride causes gastrointestinal disturbances and may produce injury to the liver and to the kidneys. In most of the cases observed, uncomplicated recovery has been the rule after carbon tetrachloride absorption. In two instances, death occurred as a result of acute hepatitis and acute renal insufficiency following irritative phenomena in the kidneys with suppression of urination. Both of these patients were known alcoholics. Because of the changes in the liver parenchyma that may be present in chronic alcoholics, it is advisable to exclude these individuals from working with any of the solvents.

The clinical symptoms and objective findings that occur in the patients with silicosis and asbestosis and asbestosis will be discussed in another lecture at greater length. The speaker, however, desires to call attention to the fact that pulmonary tuberculosis in its X-ray appearance may mimic any and all types of dust diseases, as may be illustrated by the following case citation.

A patient 32 years of age, was employed for a period of two years as a

machinist's helper. His work consisted in "cutting brass, shaping iron and steel". After this two year period, the patient complained of "increasing shortness of breath, weakness and pains in the chest". Examination was essentially negative except for the exaggeration of the bronchovesicular breath sounds and a slight degree of hyperresonance thruout both chests. X-Ray examination revealed findings commonly present in silicosis. Scattered thruout both lung fields, there were distinct military nodulations, symmetrical in distribution. In the right infraclavicular region, there was an area of coalescence of these nodules. In the second and third interspaces of the left chest, there was conglomeration of these nodules with some honeycombing, but no definite cavitation was noted. The patient was markedly dyspneic and at-thapasic with a pronounced decrease in the Vital Capacity. A visit to the plant where the patient was working revealed the fact that there was no silica hazard either immediate or in the vicinity where the patient was working. A review of the past history excluded any possible contact with silica. Despite the fact that the X-Ray had all the characteristic appearances of silicosis, it was reasonable to conclude after thoro investigation that the patient had a healed, military tuberculosis. This case citation is further proof of the necessity of thoro studies in the differential diagnosis when the question of occupational disease arises.

III. Corroborative laboratory data.

In many instances where occupational disease is suspected, laboratory aids are of importance and value in arriving at a diagnosis. The laboratory investigations that can be of value are:-

- a. Studies of the working environment (dust sampling, air analysis, etc.)
- b. Examination of the biological materials (urine, whole blood, etc.)
- c. Complete blood count and blood chemistry studies (for liver and renal injury, etc.)
- d. X-Ray examination.

A detailed discussion of the evaluation of any or all of these various procedures as laboratory aids does not seem essential to this presentation. It is apparent that abnormal concentrations of silica dust, benzol, etc. in the atmosphere can be considered as proof of the presence of working conditions that are harmful. The examination of biological materials is of value in determining the presence or absence of abnormal amounts of metals such as lead. When solvents are suspected of having caused injury to the liver, special blood chemistry studies as the Icteric Index and the Van den Bergh are of importance. X-Ray examination of the lungs is of course essential in determining whether or not there have been parenchymal changes as a result of the inhalation of any dust that may be considered harmful.

In discussing the criteria for determining the presence of occupational disease, consideration has been given to disease that may develop after exposure to contact with an injurious substance over a period of many months or years. There can be no disagreement as to the classification of lead poisoning, silicosis, benzol poisoning, mercury poisoning, etc. as occupational diseases. The medical aspects of occupational disease if based upon definite criteria, become simpler to define and interpret. Since 1935, in the State of New York, Paragraph 23 has been added to the occupational

disease list to include compensation for "any and all occupational diseases". Clarity and distinctness has not resulted from this type of all-inclusive law.

As a result of the failure of specific definition, claim for compensation on the basis of occupational disease has arisen in instances of rheumatic fever, bronchitis, pneumonia, asthma, pulmonary tuberculosis, arthritis and actinomycosis, etc. These diseases are common to the general population and are not specific to any one industry. Disease may occasionally be contracted as a result of occupation, as may be illustrated by the following case:

A nurse 26 years of age, was employed in a tuberculosis hospital. Physical examination and X-Ray examination of the lungs revealed normal findings. Tuberculin skin test was negative prior to employment. Eight months after the onset of the patient's work in this tuberculosis hospital, she developed cough, fever in the afternoon and X-ray evidence of pulmonary tuberculosis. Contact with an active case of pulmonary tuberculosis may have occurred outside of routine hospital duties. However, one cannot deny that the disease may have been contracted as a result of occupation. One should not classify the rare instance of pulmonary tuberculosis which develops in an individual working in a tuberculosis hospital as an occupational disease. The necessity for formulating criteria to determine when tuberculosis is the result of an occupational contact has been stressed by Brahday. (6)

From the medical point of view, infectious diseases such as pulmonary tuberculosis and constitutional diseases such as asthma, should not be classified as occupational diseases. Asthma, like many other forms of allergy is due to underlying constitutional factors and is inherent in the individual. The occupational factor may act in the nature of precipitating clinical symptoms as may be illustrated by the following case citation:

The patient 31 years of age, had a pre-existing history of hay fever, and a familiar history of various types of allergy. He was employed in dyeing and blending furs. After eighteen months, he developed attacks of wheezing relieved by adrenalin. In this instance, an individual with a known pre-existing allergy was employed in an industry known to produce sensitization. The industry was responsible for the attack of asthma, but certainly not for the underlying allergy. Bronchial asthma is a hereditary disease which occurs only in predisposed individuals. These potentially sensitive individuals may become specifically sensitized to various proteins or non-protein substances with which they may come in contact in the course of their occupation. The period of exposure required before sensitization takes place may vary from a few days to several years and differs with the individual (7).

In another individual, 30 years of age, clinical signs and symptoms of bronchial asthma developed after six months work with an insecticide containing derris root and pyrethrum. A diagnosis was made shortly after the onset of symptoms. Following a change of occupation, the symptoms of asthma immediately subsided and there has been no recurrence since. Pre-asthmatic symptoms such as sneezing and rhinorrhea are usually present before the attack of asthma occurs and it is advisable to remove the patient from his environment contact when these early symptoms of sensitivity are evident.

In the State of New York, "accidental injuries due to harmful substances" are properly differentiated from "occupational diseases from exposure to harmful substances and occupational activity". Acute poisoning such as may follow exposure to carbon monoxide, carbon dioxide, and clinical symptoms arising

out of a single exposure is considered an accidental injury due to harmful substances. These acute illnesses that are considered occupational accidents should not be classified under the heading of occupational diseases. The concept of disease in general as a physiological adjustment or occupation has been stressed by Garrison (8). Disease that can occur amongst all groups of people irrespective of station and occupation and does not occur in excess in any one occupation should not be classified as an occupational disease.

In this discussion of the criteria of occupational disease, it seems apparent that the diagnosis of an occupational disease can be established in any given case. If medical opinion were to prevail, there should be very little difficulty in the classification of what is and what is not an occupational disease. For the sake of clarity, it seems advisable to have clearly stated those diseases which are to be considered occupational. The criteria for determining when such illnesses as asthma, infectious diseases, etc. may be occupational, can likewise be defined. With clear definitions and criteria established and accepted, the problems that arise in any discussion of occupational disease are nearer solution.

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LECTURE D-III

TOXIC GASES, FUMES AND VAPORS

Leonard Greenburg, M.D.

It is a rather difficult task for a speaker to present the subject of toxic gases, fumes and vapors in a course such as the present one in the thirty minutes which have been allocated to this lecture. The speaker must decide in his own mind how he can present the subject so as to give the students a maximum of valuable information. Because of this limitation, it has been decided to present the basic underlying principles of the subject with very little detail concerning the toxic properties of any single material, in the hope that these basic principles will serve as a framework on which the student may, in the future, add his accumulated knowledge.

In order to maintain life, the animal body obtains oxygen from the inspired air and in turn exhales carbon dioxide resulting from the metabolism of fats, proteins and carbohydrates within the body. This exchange of gases takes place in the lungs, between the air in the terminal alveoli and the blood, in the smallest of the blood vessels, namely the capillaries.

The surface area of the alveoli is extremely large, approximately 90 square meters, which is approximately the area of a standard size tennis court.

The amount of air which a person breathes in order to satisfy these metabolic requirements varies with the degree of physical work. Obviously, the heavier the physical work the more air and oxygen is required. The following is the amount of air in approximate figures required for various degrees of activity.

Resting bed	6 liters per minute
Walking	14 - 26 liters per minute
Running slowly	43 liters per minute
Heavy Exertion	65 - 100 liters per minute
Heaviest exertion	Up to 150 liters per minute

If there are contaminants in the atmosphere being breathed, a large amount may be taken in in a relatively short time because of the large gaseous exchange and it is obvious that if a man is engaged in hard labor, his gaseous exchange will increase and for this reason he takes in more noxious gas. Therefore, the total amount of noxious material taken into the lungs is greater than that in the case of a man engaged in sedentary work. This simple fact explains why heavy exertion produces susceptibility to poisoning. In practice, we sometimes find that a man compelled to enter a closed tank, say for example for the purpose of cleansing, may succumb to the gases, fumes or vapors in the tank and fall unconscious to its floor. Subsequently, when rescue is attempted by a second worker without adequate protection, the second worker will also succumb to the effects of the toxic material. Frequently on rescue of both men from the tank by proper and suitable means and with the inauguration of resuscitation techniques, the first man will be brought back to consciousness whereas the second man, the attempted rescuer of the first, will often succumb and fail to be revived. The second man clearly has, by virtue of his exertion in attempting to lift the first man, absorbed a larger amount of the noxious material which brings about his death.

The large surface of the capillaries, the large surface of the alveoli, and the large amount of inhaled air which takes place particularly with physical effort, make it obvious that even small percentages of contaminating substances in the atmosphere breathed may lead to the absorption of relatively large amounts of the toxic material by the blood of the exposed person.

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The blood carries gases by virtue of two mechanisms:

- (1) the hemoglobin mechanism which plays a role in the transport of oxygen and carbon dioxide; and
- (2) the solution mechanism by virtue of which a gas is absorbed by liquid such as the blood, depending on the partial pressure of the gas and its solubility in the blood. This is usually referred to as the law of partial pressures or the law of Henry and Dalton.

In passing, it may be pointed out that the solubility mechanism is of great importance when men are exposed to atmospheres under increased pressure, such as compressed air work, in the case of divers, or caisson workers or when men are exposed to low atmospheric pressures as in the case of aviators ascending to great heights in a very short period of time.

Just for illustrative purposes, I have made two computations with reference to two important substances; namely benzol and methanol. These computations are very approximate and do not take account of certain important factors which we need not discuss in the short time available but they will serve to indicate very approximately the amount of absorption which may take place in the case of a man engaged at ordinary labor in the usual type of workshop.

Let us assume that a man breathing 25 liters of air per minute is working in an atmosphere of benzol vapor of approximately 75 parts per million for one hour, a concentration of benzol vapor not uncommonly encountered in industrial establishments. The man would absorb 0.36 grams of benzol and in an eight-hour working day he would absorb 2.9 grams of this material. The fatal dose by mouth is usually given as 15 grams.

In the case of methanol, a worker in one hour would absorb 0.26 grams and in eight hours, 2.05 grams when the atmosphere breathed contained 100 parts per million. A dose of approximately 16 grams taken by mouth at one time is considered dangerous.

Please bear in mind that these two examples are not to be considered accurate but merely indicative of the amount of toxic material which may be absorbed in a given working period.

It is important to bear in mind that absorption by the pulmonary route is more important than absorption through the gastro-intestinal tract because of one other basic reason. When a toxic material is absorbed through the gastro-intestinal tract, the blood which carries this material must first pass through the liver before going to the body tissues. In the liver, the material may be detoxified. On the other hand, the blood which absorbs toxic material from the lungs does not pass through the liver first but passes directly to the body tissues by means of the arterial circulation. This fact is of great importance in the case of toxic dusts. Years ago it was a general belief that lead poisoning was chiefly produced by the ingestion of lead and many industrial establishments expended considerable effort in compelling workers to cleanse their hands, teeth and mouth prior to eating lunch. Now we know that most of the lead which is ingested is excreted rather directly through the intestinal tract, whereas the lead dust which is breathed into the lungs must remain there in situ until it is dissolved and carried away by the blood stream. Only a small portion of such lead dust is expectorated or removed from the lungs by the natural cleansing mechanism of this organ.

The classification of atmospheric contaminants is a somewhat arbitrary procedure but is nevertheless a valuable one for students of this subject since it makes it possible to readily keep in mind the general scope of the problem with which we are dealing. The following classification is suggested as a useful one for this purpose:

- a) Dust - Solid particles mechanically generated; in sizes from say 1 micron to 10 microns and larger.
- b) Mists are gases in which liquids in the form of droplets or bubbles are dispersed.
- c) Fumes are gases in which amorphous or crystalline particles are dispersed, and usually resulting from the sublimation or condensation of vapors of various metals. The particle size here is usually from .001 to 1 micron in size.
- d) Vapors and gases, particles below .001 microns in size having vigorous Brownian movement of the single particles.

We must now pass on to a consideration of the nature of toxic gases, fumes and vapors. For your purposes, I should like to present these in four groups, as follows:

- (1) Irritants
- (2) Asphyxiants
- (3) Volatile drug and drug-like substances
- (4) Inorganic and organo-metallic substances

Let us now consider the nature of each of these groups of substances.

Irritants

The most important characteristic of the group of irritant gases is the inflammation of the tissues which they produce.

The members of this group naturally vary to some extent in the relative degree of irritation they produce as compared with the systemic toxic effects resulting from their inhalation. This group may be divided into two sub-groups; the first known as primary irritants which possess little or no systemic toxic effects; and the second group, the secondary irritants which produce systemic effects of some importance.

The primary irritants vary among themselves in that some have their most important effects on the upper portions of the respiratory tract whereas others exert their most profound effect on the bronchi and still others on the deeper pulmonary tissues. For example, ammonia, hydrochloric acid, formaldehyde and sulphuric acid, in general, affect the upper portions of the respiratory tract. Sulphur dioxide and chlorine affect the upper respiratory tract and also the bronchi whereas phosgene and nitrous oxide chiefly affect the lung tissues. For example, in the case of nitrous oxide, it not infrequently happens that a worker, after exposure to this gas which may be slightly irritating so that he is conscious of the irritating effects to the upper respiratory tract, may some six or twelve hours later develop acute edema of the lungs to which he succumbs.

The most important of the secondary irritants is hydrogen sulphide which is really a systemic poison but has nevertheless marked irritant action on the cornea and conjunctivae and certain volatile hydrocarbons which possess irritant action for the upper respiratory tract and bronchi but whose most important effects are systemic in nature.

It not infrequently happens that with exposure to low concentrations of hydrogen sulphide, say for example in building excavations, that the workers may

complain only of conjunctival irritation for which they seek medical relief. Several years ago, in the construction of a large sewer project in the vicinity of Buffalo, the excavation passed through an area in which hydrogen sulphide was liberated in small amounts. Here many men developed conjunctival irritation but no systemic effects.

Asphyxiants

The general characteristic of this group of substances is that for reasons which we will discuss later, the body tissues are deprived of oxygen and naturally, when this occurs, asphyxiation results.

This group is composed of two sub-groups:

- (1) Simple asphyxiants
- (2) Chemical asphyxiants

Simple Asphyxiants

The simple asphyxiants are inert or physiologically neutral gases which exclude oxygen from the atmosphere which the worker is compelled to respire, the gases being present in concentrations so high that the necessary amount of oxygen to support animal life is not present. In this group we may place such gases as nitrogen, hydrogen, helium, methane and carbon dioxide.

As you know, fermenting silage kept in silos on farms and used for cattle fodder produces carbon dioxide in large amounts. It not infrequently happens that a farmer is compelled to descend into such a silo for the purpose of repair or some other reason and there becomes asphyxiated. Methane similarly affects workers in mines and other places underground where this gas accumulates in high concentrations.

Chemical Asphyxiants

The chemical asphyxiants are different than the group of simple asphyxiants in that the exposed person breathes an atmosphere sufficiently high in oxygen to support life but the chemical asphyxiants prevent the utilization of this oxygen by the tissues. Two examples of this group may be cited; namely, carbon monoxide and the cyanides, for example, hydrogen cyanide (HCN).

Carbon monoxide readily combines with the hemoglobin of the blood and in effecting this union, prevents the oxygen of the respired air from entering its normal combination with the hemoglobin. In other words, the carbon monoxide decreases the capacity of the hemoglobin for carrying oxygen. Naturally, with small concentrations of carbon monoxide, the decrease in oxygen carrying capacity may only be relatively slightly decreased but with higher concentrations of carbon monoxide in the atmosphere breathed and over a longer period of time, the body may actually be deprived of the necessary quota of oxygen for the support of life and asphyxiation will result.

Hydrogen cyanide is somewhat different in its action. It combines with the oxidative ferment in the tissues and in this way deprives the tissues of their capacity to utilize the oxygen for the maintenance of the vital processes. The end result, however, is essentially the same and if continued for a sufficient period of time, asphyxiation will result.

Volatile Drug and Drug-like Substances

The third group of materials which must be considered is a wide variety of chemicals which are extensively used in industry, particularly since the advent of the war. This group includes the organic solvents and certain petroleum derivatives.

The chief characteristics of this group taken as a whole is the fact that all of these substances have, to some extent, the property of inducing anesthesia when inhaled in sufficient quantities over a sufficient period of time. Some have little or no other physiological action on the body than the production of anesthesia while in the case of others, the anesthetic action is rather mild, whereas the systemic effects are extensive.

a. Primary Anesthetics

The most marked effect of the inhalation of these substances is the production of an anesthesia. As examples of the substances in this group, we might cite nitrous oxide, hydrocarbons of the paraffin series, such as methane, ethane, propane, butane; various fractions of petroleum, such as petroleum ether, petroleum benzene, gasoline, etc.; certain of the ketones, such as methyl ethyl ketone; and certain hydrocarbons of the olefin series, such as ethylene, propylene and butylene.

b. Anesthetics which may cause changes in visceral organs

This group of substances produces organic changes in the body even in the absence of marked anesthesia although the anesthetic changes are present if the exposure is sufficient and these substances may also produce visceral changes from prolonged and repeated exposures to concentrations which do not produce anesthesia. In other words, in this second group, while the substances are anesthetic in nature, the anesthetic action is overshadowed, particularly in industrial exposures, by the changes produced in the body organs. In this group of substances, we may include methyl chloride, methyl bromide, carbon tetrachloride, trichloroethylene, vinyl chloride.

c. Anesthetic gases and vapors which injure the hematopoietic system

These substances also produce anesthetic effects but as in the previous group, the anesthetic effect is usually overshadowed by the injury which these substances produce in the hematopoietic system, particularly in industrial exposures. There are two outstanding examples in this group; namely, benzene and toluene.

d. Anesthetic gases and vapors which chiefly injure the nervous system

This group of substances is characterized by its high degree of solubility and in low concentration in the respired atmosphere the concentration of these materials may mount up in the blood stream to rather high levels. They appear to have a particular predilection for the nervous system on which marked changes may be seen as a result of exposure. In this group we may include such substances as methyl alcohol, propyl, butyl, amyl and ethyl alcohol, carbon disulphide and methyl bromide.

e. Volatile drugs acting mainly on the circulation

In this group there are but two or three important substances; namely nitrobenzene and aniline and the organic nitro compounds. Nitrobenzene forms an isomer of oxyhemoglobin when it comes in contact with whole blood. This isomer is called methemoglobin. Methemoglobin has a property of binding oxygen more firmly than does hemoglobin. It is thus relatively difficult to dissociate the oxygen from methemoglobin. In this way the normal function of the hemoglobin of the body is altered. Poisoning from these substances is now relatively uncommon in industry since industrial processes which use nitrobenzene and aniline must often use these materials in closed pipe lines, in this way preventing exposure of workers to the absorption of this material.

Inorganic and Organo-Metallic Volatile Substances and Miscellaneous Compounds

This group of substances, somewhat miscellaneously related, includes those materials which are characterized chiefly by their effects on organs of the body and have relatively little other effects of importance. In this group we may include such substances as mercury, phosgene, lead, nickel and iron carbonyl and arsine. In general, most of the heavy metals which are toxic should be placed in this latter group.

Protection against Toxic Gases, Fumes and Vapors

I believe it is important for the industrial physician to know a great deal about protection against toxic gases, vapors and fumes. Unfortunately, many industrial physicians do not have an appreciation of the basic principles involved in the protection of the worker against these toxic materials.

The first approach to protection consists in the substitution of non-toxic or relatively non-toxic for toxic materials; for example, in the use of petroleum fractions as solvents instead of benzol; or, for example, in the use of toluol in place of benzol under controlled conditions.

The second approach consists in the use of material which may be toxic but utilizing the material in closed vessels and closed pipe line systems only. This technique is by no means possible in all cases but where it may be used, it is a great aid in the conduct of industry on a safe basis.

Where these first two techniques cannot be used, the third technique may be utilized; namely an enclosure surrounding the processes insofar as possible and the provision of an exhaust ventilation system so that there may be no degrees of toxic material from the enclosure into the workroom air.

Where these approaches fail or cannot be used, it is necessary to resort to the use of respiratory protection devices. Such devices are of many different kinds, depending upon the nature of the material against which the worker is to be protected. In the case of dusts of relatively large particle size, mechanical filters consisting of paper or cloth may be used. Where the material is colloidal in size, then it is necessary to use a finer type of mechanical filter which may consist of a cloth or paper impregnated with substances which render the pores of the filtering material small enough to retain colloids. In the case of gases, it is necessary to use material or chemicals in the gas mask absorbing chamber which is capable of absorbing the toxic gas and preventing its passage through the filter. Such materials as pumice or diathermatic earth or activated charcoal or silica jell may be used. It must be borne in mind that respiratory equipment is really a last resort in the protection of the worker.

All filters and respirators have a certain resistance to the passage of air. This resistance increases with the increased rate of air flow and therefore where a man is engaged in heavy laborious work, the resistance may become a serious impairment to his effort, increasing fatigue and decreasing working efficiency. Bear in mind that respiratory equipment may only be used if sufficient oxygen is present in the atmosphere or if sufficient oxygen is supplied from a self-contained oxygen bottle carried with the respiratory equipment. It should also be borne in mind that gas masks may be used only for low concentrations of toxic gases, in most cases in the neighborhood of 2 or 3 per cent, and that they only provide protection for that gas or group of gases for which they are designed. Their working life is relatively short, usually no longer than two or three hours, and finally that the use of a gas mask in a toxic atmosphere, particularly at moderate or hard labor, can only be carried out by health and trained workers.

LECTURE D-11

INDUSTRIAL SOLVENTS

May R. Mayers, M.D.

The World's Fair introduced many of us for the first time to an amazing array of new materials made possible by the ingenuity of our industrial research chemists. Since that time the war has greatly accelerated industrial chemical research; and in this phenomenal growth and development industrial solvents have played an indispensable role. Indeed, our material world would look entirely different without them.

We could never have developed our great synthetic rubber plants, so important to the war effort, without the use of a variety of special solvents. Solvents are revolutionizing the shoe industry where adhesives are largely taking place of machine or hand-sewing. This change is introducing benzol, one of our most toxic solvents, into an industry where solvents were little used before. As a preliminary step to electroplating and anodizing operations, or the spray-painting of metal parts, it is now the practice to remove the film of grease which tends to adhere to the surface of the metal in order to get a more perfect finish. For this, various solvents are used as degreasing agents - the most popular being tri and perchlorethylene. In the manufacture of waterproof materials - such as are used in making army raincoats - plastics have replaced rubber, and large quantities of solvents have been required.

These are only a few examples. New chemicals with special solvent properties and new combinations of solvents are constantly being developed to meet the production needs of special industries. In view of this, it is interesting to note that despite this competition, a few of our old time solvents continue to hold their own.

First among these, perhaps, are benzol and toluol, both aromatic hydrocarbons, which are now being produced in unprecedented amounts because of new chemical processes; which have been developed. Benzol, as you know, injures the hematopoietic system, causing the blood disease known as "Aplastic Anemia". It is very insidious in its action and its use in industry is very difficult to control. Since the war, toluol which has always been a good substitute for benzol in many industrial situations and is far less toxic, has been difficult to obtain for use as a solvent because of its priority for the manufacture of T.N.T. for explosives.

The chlorinated hydrocarbons constitute another very large group of substances of primary importance to industry. Many of these substances are highly toxic; not all of them, however, are solvents. Most familiar in this group are carbon tetrachloride and trichlorethylene.

Physicians are more familiar with carbon tetrachloride as a therapeutic agent for the treatment of intestinal parasites than as an industrial solvent. Sold as "Carbena" in drug stores, it is popularly regarded as an effective and harmless non-inflammable cleaning fluid. This is a serious error. Dermatologists have even prescribed it to young people suffering from acne - to remove excess grease from the skin. On such advice, one girl washed her face with it every night before going to bed for a year - and nothing happened to her. Also, nothing happened to the acne. Nevertheless, inhalation of carbon tetrachloride vapors, may result in toxic hepatitis, renal insufficiency or a hepatic-renal syndrome. There appears to be enormous difference in individual susceptibility to this substance. Fatalities from acute yellow atrophy of the liver have occurred in individuals who did not appear to have had any great exposure.

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A man working in a plant where refrigerators were being re-conditioned, made it a practice to cleanse the small metal parts by dipping them in a small pan of carbon tetrachloride which he kept on a bench beside him. He developed intense jaundice and almost died. A whole series of girls using small amounts of carbon tetrachloride to remove small stains from parachutes they were sewing, all developed serious illness, though the exposure did not look impressive. Exposure to really high concentrations may result in serious lung irritation with edema of the lungs, pneumonia, convulsions and death. We will discuss this substance later on in greater detail.

Trichlorethylene, as commonly used in industry, had been regarded as a relatively harmless solvent with simple narcotic properties. Under war conditions, however, it recently proved fatal to two workers who tried to clean out the sludge from the inside of degreasing tanks without using proper respiratory protection.

Similarly acetone and ethyl methyl ketone, are solvents which had been looked upon as relatively innocuous. Since the war, however, they too began to be used in unprecedented amounts and we began to discover toxic potentialities never before suspected. One girl subjected to an intense exposure, developed convulsions and another became unconscious. Both were promptly removed to a hospital where they fortunately recovered. In one plant, where army raincoats were being manufactured and these solvents were used to glue together the seams, total solvent vapors in the air were found to be so high as to reach explosive proportions. In this case, control of the toxic hazard automatically reduced the concentration below the explosive limit.

You probably remember the deaths which occurred through the country when ethylene glycol was tried as a vehicle for the sulpha drugs. In industry "methyl cellosolve" the mono-methyl ether of ethylene glycol was found to be extremely toxic both to the blood and the central nervous system when used in connection with the manufacture of so-called "fused" collars -- collars which come back stiff from the laundry without having to be starched.

Carbon disulphide is another of the solvents which is toxic to the central nervous system. It is used in the manufacture of rayon. Unless adequate control measures are instituted it is capable of producing very disturbing personality changes; psycho-neuroses and even true psychoses which may be indistinguishable, medically from similar conditions of non-occupational origin with which the psychiatrist is familiar.

The petroleum distillates; gasoline and petroleum naphtha, though highly inflammable, continue to be our safest solvents, toxicologically speaking.

Leaving systemic effects, for the moment, it is important to remember that since all of these substances are fat solvents they necessarily tend to defat the skin and dermatoses commonly result from skin contact.

What is far more interesting in this connection, however, is that dermatoses may develop in workers who never touch the solvents; dermatoses involving the face and neck, for example, of workers who merely stand around in the room where the solvent is used, or frequently pass through it, in the course of the day's work. This was something of a puzzle to us at first. Repeated air tests, however, in typical situations brought to our attention the fact that when sufficient solvent vapors are present in the air of a workroom, deposition of droplets from the air upon exposed parts of the skin is capable of causing a dermatitis.

In such cases the dermatitis can be prevented not by gloves or other protective clothing, but by the installation of a proper ventilating system to remove these air contaminants at their source and so prevent their dissemination into the atmosphere of the workroom. Simultaneously, such an approach will prevent systemic disease.

It will be readily seen from the above that the clinical entities which arise in connection with exposure to industrial solvents are very diverse -- illustrating the broad scope of industrial toxicology. This is a relatively new field to the medical practitioner who necessarily sees few of these cases in his practice, or even at the hospital. The sooner, however, that he comes to appreciate the problems involved -- and also the extent to which he will inevitably become personally involved -- the better prepared he will be to meet the new opportunities and responsibilities which are awaiting him. The medical practitioner is yet to experience the full impact of industrial chemical research upon the practice of medicine.

It is the purpose of what follows to discuss some of the basic medical problems with which the medical practitioner is confronted in handling a patient who has been exposed to an industrial solvent. This would seem to be more profitable than taking each solvent in turn and giving a resume of its toxicological properties, since these can be readily obtained from the literature.

Since exposure to benzol in toxic concentrations produces a blood picture with which most physicians are familiar -- the blood disease known as "aplastic anemia" -- this condition would seem to provide a particularly good medium for the present discussion. What is there about industrial blood dyscrasias which makes them different from those of non-occupational origin? What are the similarities? Is there anything special that a physician would be expected to do about them other than what he does in his regular practice? These are a few of the pertinent questions. The answers are interesting. Industrial blood dyscrasias do not differ in any fundamental way from corresponding blood diseases which are non-occupational in origin. The diagnostic procedures and laboratory techniques are essentially the same. Aplastic anemia is aplastic anemia whether it is due to benzol or it is of unknown etiology. But the important thing is that when it is due to benzol, the etiological factor is known; the cause can be removed, and the patient's life can often be saved. Furthermore, "aplastic anemia" of industrial origin can be prevented -- which is manifestly impossible where the etiology is obscure or unknown.

And so, it behooves the practicing physician not only to know how to recognize and make a correct diagnosis of blood dyscrasia which comes to his attention but to make careful inquiry into the possibility of an occupational etiology in that particular case. Furthermore, recognition of early evidences of injury to the hematopoietic system in cases of industrial origin provide the means for preventing the development of fatal blood disease. It provides the means for removal of the patient from injurious exposure while there is still an opportunity to save his life. Furthermore, such a person should never again be permitted to work with benzol or any other hematotoxic substance. Cases are reported where years after exposure to benzol had ceased and there had been an apparently complete recovery, a worker will for some unknown reason develop a recurrence of the aplastic anemia and die.

Benzol is a most insidious substance. Serious blood changes may be present in the absence of a suggestive clinical picture. Weakness, fatigue, loss of weight, epistaxis, dryness of throat, loss of appetite, nausea, dizziness, insomnia may all be absent even when blood studies can demonstrate injury to the hematopoietic system.

One occasionally finds a worker complaining only of the fact that "black- and blue spots" appear on his skin from time to time. He usually believes he has "bumped himself", though he does not remember exactly when. Or, a worker may have no complaints whatever. And yet an examination of his blood may show a critical blood dyscrasia; and the case may go rapidly down hill to a fatal conclusion, before adequate steps can be taken even to obtain hospitalization. Incidentally, the frequent lack of purpura and bleeding from mucous surfaces in industrial benzol poisoning has in some instances made it impossible to get the release of emergency hospital beds even for cases which terminated fatally, because the critical condition of the patient was not properly appreciated. Bleeding and coagulation time are not always increased even in cases with greatly reduced blood platelets.

As in all cases of "aplastic anemia", a marked leukopenia and reduction in the blood platelets are the characteristic blood findings. The relative lymphocytosis, much discussed is not usually present. An early reduction in red cells, which is extremely important has received insufficient attention. A characteristic increase in mean corpuscular volume, which also occurs early, will be overlooked unless the Wintrobe constants are studied. Contrary to popular belief, the bone marrow, on sternal puncture is not always aplastic. It may be hyperplastic.

Prevention is always more satisfying than treatment. And in industry, the plant physician has unique opportunities along these lines. They are: (1) cooperation with management in seeing to it that the chemical exposure is kept within safe limits, by ventilation or other safe practice procedures; and (2) periodic physical examinations of workers to detect early indications of blood abnormalities. Where such abnormalities begin to make their appearance in workers in a particular department, it is a sure sign that the exposure is excessive and something should be done to reduce it. Indeed, the extent of the exposure and actual absorption of benzol in specific departments may be further evaluated by examination of the urine for changes in the sulphate partition. There seems to be a correlation between benzol absorption and the ratio of organic to inorganic sulphates in the urine. Such changes provide interesting data as to absorption. They do not, however, give evidence of intoxication.

In plant medical surveys, what are the earliest blood changes to be looked for? Experience has shown that by and large, too great reliance should not be placed upon a leukopenia alone when one is attempting to spot early cases of benzol poisoning in such a survey. Any white cell count below 5,000 should be looked upon as having possible significance and the patient kept under careful medical observation. Marked leukopenia is more frequently found in severe than in early cases; and may occasionally be absent even in the former. Reduction in the red cell and platelet counts when associated with an increase in mean corpuscular volume appear to be the earliest signs of intoxication. Indeed, a macrocytosis in a benzol worker should, per se, be looked upon with suspicion even in the absence of all other blood abnormalities. The absence of purpura and bleeding from mucous surfaces should not lull the physician into a false sense of security.

Next to benzol, carbon tetrachloride is perhaps one of the greatest offenders of the industrial solvents. Its use during the war has increased enormously. This is partly due to the fact that its toxicity is not adequately appreciated; partly to the fact that it is sold under a great variety of trade names so that the user has not knowledge of its chemical composition; partly because it is peculiarly suitable, because it is non-inflammable, for use in cleaning large machinery, motors, dynamos and the like which cannot be dipped into the degreasing tanks used for small metal parts.

There appears to be an enormous difference in individual susceptibility to this substance. Alcohol seems to greatly dispose to acute poisoning. I like to tell the story of a physicist in our office who, on leaving college, got a research job in the physics department there. He was studying certain of the physical properties of benzol, carbon tetrachloride and carbon disulphide--all three apparently having a molecular structure of special interest to him. In order to do his work, which required the greatest degree of precision, it was necessary to keep all the windows and doors closed so as to prevent air movement. Occasionally, when he arrived in the morning, he found that despite all precautions, a fly had gotten into the room. In those cases he promptly killed the fly by spraying him with carbon tetrachloride from an atomizer he had ready for the purpose. He had no idea that he was dealing with poisonous substances. No one at the college warned him. He is perfectly well today. One day when he was telling us this story -- not realizing how hair-raising it sounded to industrial toxicologists -- I asked him whether he ever drank any alcohol in those days. He replied that he didn't because he couldn't afford it. I believe his lack of funds saved his life.

To the medical practitioner, carbon tetrachloride poisoning presents some very difficult problems in differential diagnosis. Excepting in extremely acute cases, one is dealing with a toxic hepatitis; a nephrosis, or a combination of both. There is nothing in the clinical picture which is pathognomonic of carbon tetrachloride. The patient is usually jaundiced when he first consults his physician.

Is it obstructive jaundice; catarrhal or epidermic jaundice; or is it a toxic hepatitis? Only a careful work-up of the case will produce a correct diagnosis; and in such a work-up, the occupational history is indispensable. That does not mean that a person who has been exposed to carbon tetrachloride may not also develop an obstruction of the biliary tract in no way related to his occupation. In a toxic hepatitis, however, one gets very suggestive changes in the blood chemistry: a phosphatase above 10; increase in serum cholesterol; a high serum bilirubin; low total proteins, with the serum albumin especially low, and the euglobulin somewhat elevated. The icteric index is elevated; one gets an indirect Van den Berg reaction and increased bilirubin in the urine. There may be an increase in blood guanidine to which some workers attribute the liver damage. The blood sugar and calcium are low. The blood nitrogen is high where there is an associated nephrosis, as there often is.

To the physician who sees the case early in the plant medical office, the patient may present a very different picture. He may come in complaining of gastro-intestinal disturbances only. There will be no jaundice at this stage and many of these cases are consequently unrecognized as potential liver cases. They are frequently given a pill, by the physician or the nurse and sent about their work. By the time jaundice develops, it may be impossible to save his life. This is not an uncommon experience. A plant physician should acquaint himself with the substances to which the workers, for whom he has assumed responsibility in the plant, are exposed. If they are working with a hepatotoxic substance such as carbon tetrachloride, he must regard any case coming in with gastro-intestinal complaints as a potential liver death and investigate the case thoroughly and without loss of time. He should see exactly where in the plant, the person has been working, and assure himself that the operation is conducted with proper safeguards so that he will not have a series of liver deaths on his hands in the immediate future. This has happened more than once in our experience.

The plant physician should make careful pre-placement as well as periodic examinations of workers who are to be exposed to toxic solvents of any kind. In the

case of carbon tetrachloride, persons with diseases of the liver, kidneys or heart particularly should not be exposed. Chronic alcoholics are definitely out. In periodic physical examinations suggestive changes in blood chemistry may give earlier indications of intoxication than will the liver function tests. If he looks for it, the plant physician may discover a concentric constriction of the visual fields in early carbon tetrachloride poisoning. Prevention is possible in the early stages. Once a true toxic hepatitis develops, it may be impossible to prevent a fatal termination.

Without going into detail as to therapy in industrial liver cases, it might be worth mentioning the fact that although a high carbohydrate diet, which is also high in calcium, is known to protect the liver, during acute illness, a high protein diet is indicated particularly in the presence of the low serum protein which is usually associated with this condition.

Recent animal experiments have demonstrated that certain of the sulphur-containing amino-acids appear to have the property of protecting the livers of animals exposed to known liver poisons, such as chloroform or carbon tetrachloride. Administration of these amino-acids before and sometimes even after giving a known fatal dose of these liver poisons have prevented the development of the toxic hepatitis and saved the animal's lives. Recently, one of these amino-acids, known as methionine, was reported to have saved the life of a man who accidentally drank a fatal dose of carbon tetrachloride. Experiments such as these are still in their infancy. But the results are very stimulating and full of promise. Perhaps some chemical substance will be available to us in the near future which would enable us to provide adequate protection to workers who, in the course of their work, are unavoidably exposed to substances potentially injurious to the liver.

There are some other animal experiments which, I believe, will be of interest to you. Animals exposed first to one and then to a second hepatotoxic substance, will develop acute yellow atrophy of the liver and die even when the doses in both instances were so low as to cause no demonstrable injury when given singly. Though analogies with animals do not always carry over to human beings, these experiments have made us feel that it is unwise to expose workers suffering from liver disease or those who are receiving treatment for syphilis with a hepatotoxic substance such as arephenamine, to a second hepatotoxic substance such as carbon tetrachloride, in the course of their work.

That a combination of toxic substances is apt to be more toxic than is the sum of its single components is a general principle which we hear reiterated from time to time. It apparently has a substantial basis in experience, both in the experimental laboratory and in industry. For this reason I would like to tell you briefly about an interesting case which came to our attention, where there was exposure to a combination of several of the chlorinated hydrocarbon solvents.

A man, 26 years of age, was engaged in applying a paint remover in reconditioning elevators in buildings. Because of the nature of the work, no ventilation was provided. The paint remover contained carbon tetrachloride, chloroform and a considerable number of the higher chlorinations of ethane, propane, butane and pentane; the exact chemical components being unknown even to the producers, as is often the case where chlorination processes are carried out on complex organic substances.

The man in question, worked with the paint remover for about a year and lost some 40 lbs. in weight during this period. We have insufficient knowledge of other elements in the clinical picture during this time because we learned

of the case only after his death. About five days before admission to the hospital, however, he complained of severe abdominal pain with vomiting, and presented a general picture of surgical abdomen.

Examinations of his blood in the hospital prior to operation showed that he had a white cell count somewhat below 3,000 and a red cell count slightly above 3 million. There was a marked increase in bleeding and clotting time and platelets were greatly reduced. A laparotomy revealed necrotic ulcerations of the colon. The bleeding was such that nothing could be done. At autopsy he was found to have an aplastic anemia with a granulocytopenia, generalized purpura, toxic nephrosis, hepatitis and necrotic ulcerations of the colon.

The combined injury to the hematopoietic system, the liver, and kidneys presented by this case is of the greatest interest in view of the nature of the exposure. Even more interesting, however, are the necrotic ulcerations because ulcers of the gastro-intestinal tract are reported from time to time in persons exposed to large amounts of carbon tetrachloride. Two such cases have recently come to our attention: one, a man with a perforated peptic ulcer which was operated upon; the other, a man who developed severe hematemesis and was hospitalized, where X-ray pictures taken later revealed definite evidences of a duodenal ulcer. Both men had had unusually heavy exposures to carbon tetrachloride immediately preceding the onset of these acute surgical conditions. Another subject for speculation in the case of the man using the paint remover was the relation of the ulcerated colon to his agranulocytosis. We all know that ulceration of the mouth and throat is common with this blood dyscrasia. However, that may be; and regardless of whether, in this particular case, the disease actually was caused by the paint remover, the clinical picture is so suggestive of what might be expected to occur under the circumstances, it is very impressive indeed.

We cannot leave the industrial solvents without a few words on the subject of narcosis. Lowgrade narcosis such as one sees in workers exposed to trichlorethylene, for example, tends to make them drowsy, irritable, restless and they are apt to complain of vague subjective gastro-intestinal disturbances which are not easy to verify on physical examination. Actually, we would like to know a lot more than we do about the long range effects of continued or frequently repeated narcosis of this general degree of intensity, over a long period of time.

A word of caution, however, is particularly necessary at this time when everyone is so psychiatrically-minded. There is an increasing tendency, now-a-days, to look upon these people as cases presenting personality changes and to refer them for study to a nearby psychiatric clinic, without stopping to consider a toxic etiology and investigate the occupational history. It has already been pointed out this evening that grave mental and neurological changes may result from exposure to certain industrial chemicals; including some of the solvents; and that these personality changes may be indistinguishable clinically from non-occupational psychoneuroses or even psychoses. I wish, therefore, to warn against a diagnosis of mental disease being made without first carefully investigating the occupational exposure of the patient.

Before leaving trichlorethylene, I would like to say a word about an explosion which recently occurred in the use of this supposedly non-explosive substance. This was believed to have been due to its use in a degreasing tank located in close proximity to some welding operations, where unusually high temperatures were being generated. Particularly now when welding operations are rapidly multiplying in industry, the possibility of such a contingency should at all times be borne in mind.

In conclusion, it might be well to stress once more the great variety of clinical disease entities which may be produced by exposure to industrial solvents in toxic concentration; and the fact that these syndromes do not necessarily bear any of the ear-marks of an occupational disease. Jaundice, aplastic anemia, nephrosis, psychoneurosis -- all of these conditions look essentially alike whether they are of occupational or non-occupational etiology. The same general diagnostic procedures and laboratory tests are required in either case in order to make a diagnosis.

Regardless, however, of how carefully and intelligently the case has been worked up; or how accurately the diagnosis, if the possibility of an occupational etiology has been overlooked the only means available for saving the patient's life may have been lost. For, if the condition is occupational in origin, no amount of treatment will be effective unless the patient is immediately removed from further exposure. Furthermore, recognition of an occupational etiology, where it exists, will provide invaluable information as to the type of work the patient must avoid in the future; it will provide a basis for prognosis; and it will make possible assistance to the patient and his family under our compensation laws - providing them with financial assistance during a period of disability. It even helps the doctor get his bills paid.

Recognition of the occupational etiology where it exists, contributes to proper reporting of occupational diseases, thereby making more accurate the occupational disease statistics available. Very few people realize that we have no reliable information at the present time as to the statistical incidence of occupational diseases, principally because physicians fail to recognize them. With the best intentions in the world, a physician will not report a case of aplastic anemia, if it does not occur to him that it is occupational in origin.

The plant physician has an unusual opportunity to protect the health of workers, exposed to industrial solvents in the plant under his charge. He should acquaint himself with the nature of the chemical substances involved and the ways in which they are processed. The Department of Labor whose legal responsibility it is to provide workers with safe and healthful working conditions is always ready to work with him and with management in ensuring safety in the use of these materials. The plant physician is thus in a strategic position not only to recognize cases early before irreparable damage has been done; but to prevent any damage whatever from being done in the first place.

Industrial solvents can, in general, be used with perfect safety if adequate control measures are instituted.

INDUSTRIAL DERMATOSES

Louis Schwartz, M.D.

HISTORY

Industrial diseases have occurred ever since the world was civilized enough to have industry. It is highly probable that the builders of the pyramids suffered from silicosis. Paracelsus; however, in the beginning of the Sixteenth Century was the first to write about occupational diseases. He refers in his book, "Morbis Metallius", to certain changes in the skin caused by salt compounds.

The first book which really gives us a compilation of occupational diseases of his day was written by Bernardino Ramazzini in about the year 1700. In this book he mentions painful and swollen hands noted in bakers and also that the sievers of corn developed an itch from the dust which he stated was due to an invisible parasite present in the cereal. This was a remarkable observation in those days before the invention of the microscope and remains true to this day, because grain itch, straw itch, linseed itch, etc., due to such parasites, occur even now among the handlers of these substances. Besides these, he also described fissures on the hands of wash women which he said were due to lye and the inflammation of the hands of midwives due to contact with the secretions of the genital tract during childbirth.

The first Englishman to write on occupational diseases was Percival Pott, who in 1775, called attention to chimney sweeps' cancer of the scrotum.

Among French writers, Alibert in the first quarter of the Nineteenth Century wrote extensively on occupational skin diseases. About the same time, C. Turner Thackeray, an Englishman, wrote a book called "The Effects of Arts, Trades, and Professions and of Fads and Habits of Living on Health and on Longevity". In this book he points out many occupational skin diseases and emphasizes the value of cleanliness in their prevention. Among French writers in the Nineteenth Century, the works of Chevalier and Bazin stand out.

In Germany, works on occupational dermatoses began to appear about the beginning of the Nineteenth Century and many German writers continued to work on the subject. Blaschko, Jadassohn, Bloch, Mayer, Oppenheim, Ullman, and Rilla are some of the names associated with this subject.

In England such writers as R. Prosser White, Percival, Sequeira, McCloud and Little have studied and written extensively on the subject.

In the United States, scattered contributions were made by various writers since the year 1900 on the subject. Occupational skin disease laws have been passed by a number of our States and many more of the States are about to pass such laws. In fact, industrial dermatoses have become of such importance in the United States that the U.S. Public Health Service in 1928 detailed an officer to make a study of this subject and opened a Section devoted solely to its study.

The prime importance in the control of industrial skin disease is the knowledge of the frequency of their occurrence. The gathering of statistics of the occurrence of occupational diseases, including dermatitis, is comparatively recent in the United States. The statistics in Great Britain in regard to dermatitis are more complete because in 1918, dermatitis due to dusts and liquids was legally made an industrial

disease. However, authorities even in Great Britain differ as to the prevalence of industrial dermatoses. E. Graham Little states that over 50% of occupational diseases in England are industrial dermatoses. R. Bartholomew states that among 8122 cases of occupational diseases reported in England from 1926 to 1931, 7011 cases, or 86%, were industrial dermatoses. S. Overton says that out of 1394 compensated cases of industrial diseases occurring in 1927 in English factories, 879 cases were dermatoses with a further 100 cases of chrome and epitheliomatous ulcerations.

The Workmen's Compensation statistics for 1928, 1929, and 1930 show that there was an increase in the number of cases of dermatitis during those years. In 1928 there were 1170 cases; in 1929 there were 1405 cases and in 1930 there were 1499 cases.

It is admitted by these English writers that the figures do not represent the actual occurrence of industrial dermatoses because only those cases are reported who are incapacitated from work for one week or more and R. Prosser White estimates that at least 16 to 19 thousand cases of industrial dermatitis occur yearly in England.

INDUSTRIAL DERMATITIS IN GERMANY

The number of cases of industrial dermatitis in Germany is not directly available, but according to official German statistics for 1930, the number of occupational diseases that year was 5920 in industry and 937 in public institutions and 7 in agriculture. These occupational diseases represent 1.08% of the total number of occupational accidents and illnesses. The figures do not include occupational dermatoses but judging from the proportion of occupational dermatoses to occupational diseases in other countries, it is safe to say, that in Germany there were at least 7000 cases of occupational dermatoses in 1930.

That there are many cases of occupational dermatoses in Austria, France, and the Scandinavian countries is evidenced by the many reports of dermatologists of these countries, but official statistics are not available.

INDUSTRIAL DERMATOSES IN THE UNITED STATES

With the advent of compensation legislation in the United States, more attention was given to occupational diseases and with them to occupational dermatoses. When the United States Public Health Service, in 1928, decided to make studies in occupational dermatoses, some of the States had already gathered statistics concerning their frequency of occurrence and had made compensation laws concerning them.

At the present time in the United States, 23 States still do not compensate for, nor record, the occurrence of industrial dermatoses. The following States now have laws which compensate to a more or less degree for occupational dermatoses and also gather statistics, more or less complete, concerning the occurrence of these diseases:

Washington	Pennsylvania	Ohio	Missouri
Oregon	Maryland	North Carolina	Tennessee
California	North Dakota	South Carolina	Wisconsin
Minnesota	New Jersey	West Virginia	Indiana
Connecticut	Rhode Island	New Mexico	Massachusetts
New York	Mississippi	Wyoming	Nebraska
	Illinois		

The laws in these States vary considerably. Fifteen of them take in all occupational dermatoses, or at least those occurring in the principal industries in those States. The others have laws which limit the dermatoses to those cases due to certain substances.

The gathering of statistics as to the occurrence of occupational skin diseases also varies a great deal in these States. Some of them make it compulsory that cases of occupational dermatitis losing a week or more from their work should be reported. Others, that dermatitis cases losing any time from their work should be reported, and some of the States require no reporting at all.

Ohio has excellent records. From 1933 to 1935 about 2500 cases of occupational dermatitis occurred in Ohio among a total of about 4000 occupational disease cases, or 60% of all the occupational disease cases in Ohio were occupational dermatitis. Between the years of 1926 to 1931, there were about 4600 cases of industrial dermatitis reported in Ohio. This was more than 70% of all occupational disease cases reported.

In New York State it is only within the last few years that anywhere near accurate statistics have been available of the occurrence of occupational dermatoses. The New York State Department of Labor cases from June, 1930 to February, 1932, show only 185 cases of occupational dermatitis, whereas the report in 1934 showed 626 cases of occupational dermatitis or 61% of 1012 cases of scheduled diseases, reported that year. The first six months of this year show over 1000 cases in New York. The figures do not include such traumatic skin affections as blisters due to friction, etc.

The records in Connecticut show that in 1931 there were 184 cases of occupational dermatitis reported. In 1932, 209 cases of dermatitis out of a total of 358 occupational diseases were reported; in 1933, 364 cases of dermatitis out of a total of 557 occupational diseases; in 1934, 414 cases of dermatitis out of a total of 485 occupational diseases. In these years, about 70% of all the cases of occupational diseases in Connecticut were dermatoses.

The last available records in New Jersey show that 73% of all occupational diseases were dermatoses.

In Massachusetts, about 65% were dermatoses.

In Oregon, a compensation is only paid for poison oak, spruce or hemlock dermatitis, yet there were 250 claims for compensation in the past year. In Oregon there are many cases of fruit poisoning contracted in canneries where employees working in fruit juices develop an infection around the finger nails, but the law does not cover these cases, nor are they reported.

In California, out of 1528 cases of occupational diseases reported, 1156 were occupational dermatitis, or about 75%. A great many of these cases were fruit poisonings.

In Missouri, 59% of all occupational diseases were dermatitis.

In Wisconsin, in ten years there were 1909 cases of dermatitis out of 3019 cases of occupational diseases, or 63%.

Altogether, it is estimated that industrial dermatitis constitutes more than 65% of all occupational disease cases reported in the United States and from available records it is estimated that there are about 20,000 cases of industrial dermatitis with sufficient severity to lose time from work occurring in the United States. Actually there are many times this number

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a compensation of about \$100.00, and that the average cost of medical care per case is also about \$100.00, so that an estimate of \$4,000,000.00 as the annual cost of occupational dermatoses in the United States is a conservative one.

The free clinics of hospitals see but few of these cases, because they are not charity patients. They are cared for by the plant physician, the insurance company physician, or the patient's own private doctor. In our investigations, we found that among over 25,000 new cases in the dermatological clinics of New York, only 58 were diagnosed as occupational, or about one-fourth of 1 per cent.

We found in the records of the clinics investigated that the histories of cases resembling contact dermatitis were not accurately taken and that patch tests were rarely performed. It seems that many of the cases of occupational dermatoses were lost in the large number of cases diagnosed as eczema and dermatophytoses and "tids". This may account for the relatively small percentage of occupational dermatoses reported in the New York dermatological clinics. However, it is not to be expected that, outside of housewives' adn wash women's hands caused by soap and cleaning fluids, there would be many cases of industrial dermatoses applying to the free clinics for treatment, because those cases are not charity cases. Their treatments are paid for either by the employers or by the insurance companies.

In our investigations as to the number of cases of industrial dermatoses treated in industrial clinics, we examined the records of the largest industrial clinic in New York City and found 103 cases of industrial dermatoses out of about thirty thousand industrial accidents treated. This clinic, however, did not draw from the basic industries where the majority of the cases of industrial dermatitis occur.

Dermatologists who are interested in occupational dermatitis and look for it in their clinics find a large percentage of them among their patients. For instance, Fordyce, in the Journal of American Medical Assoc., 1912, page 243 states that skin troubles from occupations form 2% of all skin diseases; Lane, in the Archives of Dermatology and Syphilology, November 1, 1922, pages 565-575, states that occupational dermatoses comprise four to five per cent of all cases of dermatitis; R. Prosser White states 6%; Gardner, in the British Journal of Dermatology and Syphilology, October, 1922, pages 297-320, states 7%. And Knowles, in the Journal of the Amer. Medical Assoc., 1917, p. 79, states 10%, and Oppenheim in the Transactions of the London Dermatological Society, 1924, pp. 51-74, states 20%.

CAUSES OF INDUSTRIAL DERMATITIS

There are many etiological factors in Great Britain and Germany which are not present in the United States. For instance, the scrotal cancers of chimney sweeps and mule spinners due to soot and oil; the skin cancers due to arsenic, which are reported in England; and the frequent occurrence of eczema among bakers both in England and Germany are not present in the United States.

G. H. Lancashire, in 1930, made an analysis of causes of dermatoses reported to Insurance Societies in Great Britain and found that 13% were due to aniline dyes; 12% occurred in painters and was due to turpentine or similar solvents; 18% occurred in rubber workers and was due to solvents and accelerators and 7% occurred among bakers. These figures are somewhat different from the available statistics in the United States.

The chief causes of industrial dermatitis differ in the various states

of the United States, according to the prevailing type of industry present. In 1934, in New York State, about 25% of all the cases of industrial dermatoses were due to alkalis, about 16% were due to petroleum derivations, 9% were due to plant products, 6% were due to dyes and 1% to acids.

In Ohio, the rubber industry formerly furnished nearly 25% of all the cases of industrial dermatoses and the oil and cutting compounds about 18%, but in the last few years, the number of cases of dermatitis caused by rubber compounds has taken second place to the number caused by oils. Alkalis are responsible for about 10% of the industrial dermatitis in Ohio; solvents for about 10% and poisonous plants for about 2%.

In Connecticut, the reports of industrial dermatitis in the last few years show that poisonous plants are responsible for about 50% of all the reported cases. Mercury is responsible for about 18% of industrial dermatitis cases in this State. This is because of the many hat making factories in Connecticut where mercury is used in converting rabbit hides into felt.

In Missouri, over 60% of the cases of industrial dermatitis reported were due to lime and cement, 15% were due to poisonous plants, 9% due to caustics and sulphuric acid and 6% were due to lye.

It can be stated in general that the majority of the occupational skin disease reported in the United States are caused by alkalis, oils, solvents, dyes and poisonous plants and acids.

All individuals are not susceptible to the same degree to skin irritants. The anatomical structure and the physiologic secretions of the skin are factors in susceptibility to external irritants. The defense mechanism of the skin against external irritants consists of the cornified cells of the outermost layer of the skin and the secretions of the glands of the skin. The cornified cells are tough and are insoluble in water and alcohol and will withstand the action of even fairly strong acids but are vulnerable to alkalis and sulphides. The perspiration acts as a diluent of irritants which are water soluble and the sebaceous secretions consisting of cholesterol and liquid waxes, form a protective coating against water soluble irritants. The pigment of the skin also acts as a protective factor against certain skin irritants, especially against light. The perspiration and the sebaceous matter may, however, under certain conditions act as aids to irritants on the skin. We will speak of this later.

The vulnerable portions of the skin are the openings of ducts and hair follicles through which certain chemical compounds, especially those which are fat soluble, may easily enter. Fanning or breaks in the cornified layer of the epithelium also act as portals of entry for external irritants.

RACE

In so far as the characteristic texture of the skin and the complexion affect the defense mechanism of the skin, it may be said that race plays a part in predisposition to sensitivity to external irritants. Blondes and thin-skinned individuals are more susceptible to certain irritants than those with darker, thicker and more oily skins. For this reason, many chemical factories will employ only negro labor for certain jobs in which irritants are handled- as for instance, workers on filter presses, driers and grinders in synthetic dye manufacture. Workers having greasy, oily, thick skins withstand the action of such solvents as scaps, turpentine, naphtha, etc., better than do those with dry skins. On the other hand, in occupations where oils, greases or waxes are apt

to soil the clothing, as for instance in oil refineries, machine shops and garages, those persons who have much hair on the arms and legs and who have excessive sebaceous secretions are more likely to develop acne-like lesions and folliculitis. Then again, in occupations where the worker comes in contact with water soluble irritant dusts, such as soda ash and lime, those who have an excessive perspiration are more likely to be irritated because these substances only irritate the skin when they are dissolved in water or perspiration.

In a study of skin hazards among workers in waxes, we found that the most severe cases of comedones and acne, due to these substances, occurred among those workers who have dark, swarthy, oily skins and who were in the acne age.

The various portions of the skin of the same individual differ in susceptibility to external irritants. We have found the inner surface of the forearm and the upper arm and the anterior portions of the body are more often affected by industrial dermatitis than are the other portions.

DIET

The diet of a worker influences his susceptibility to external irritants. A number of investigators have proven that when animals have been rendered acidotic or alkaline, they have been rendered more susceptible to the action of external skin irritants. We can readily see that in human beings the diet may influence the pH. of a perspiration and the pH. of a perspiration certainly influences its ability to act as a solvent on external irritants. The pH. of a perspiration also influences its action as a neutralizer of external irritants. For example, if a man is exposed to an alkali, such as soda ash, and he has a markedly acid perspiration, the acid of his perspiration is likely to neutralize the alkalinity of the soda ash, whereas if he has an alkaline perspiration, it may augment the action of the soda ash. We have partially verified this in industry.

AGE

Age also seems to exert an influence on sensitivity to external irritants. Most of the workers affected with acute industrial dermatitis are young and new workers. On the other hand, the chronic eczematoid types of industrial dermatitis usually occur in workers of middle age or beyond.

SEX

Women are very often irritated by substances in industry with which men usually work with immunity.

SEASON OF THE YEAR

Occupational dermatitis is more prevalent in warm weather. This may be due to the fact that little clothing is worn in warmer weather and contact with skin irritants is more likely to occur. Although in those industrial diseases in which lack of cleanliness plays a role, the fact that men are less inclined to bathe after work in the winter than in the summer, may equalize or even exceed in the winter the rate of the summer incidence.

Presence of Other Diseases

The presence of other skin diseases, especially of the itching type where scratching tends to rub in any irritant which may be deposited on the skin, also predisposes to industrial dermatitis. It has also been noted that those

workers suffering with seborrhea and with mycotic infections are more prone to develop industrial dermatitis than others.

Cleanliness

The most important predisposing cause of industrial dermatitis is, to my mind, the lack of cleanliness. Cleanliness of the environment—that is to say, clean rooms, clean floors, clean walls, clean machines and the air kept free from irritating fumes, vapors, and dusts, will diminish the occurrence of industrial dermatitis. Personal cleanliness is of equal importance. The daily changing to clean underclothes and work clothes, cleansing shower baths immediately after work is finished, so as to remove potential irritants from the skin, are all important factors in preventing industrial dermatitis.

ALLERGY

By this we mean a hypersensitivity in which an antibody is produced in the system in response to an external irritant which acts as an antigen. This antibody cannot be demonstrated in all cases. Most cases of industrial dermatitis are not due to allergy. They can be explained by changes in the defense mechanism of the skin of the worker or by increase in the vulnerability of the skin, or by an excessive amount or concentration of the external irritant. However, the fact that there is a specific hypersensitivity of certain individuals to the action of many substances in industry cannot be denied. I have seen cases where the mere presence in the same room with certain nitro compounds has produced pruritus in the hypersensitive individual.

Allergy can be acquired by exposure to certain substances. This exposure may be through the skin or through the respiratory or gastrointestinal tracts. Allergy to mercury, chrome, and formaldehyde has been experimentally produced by investigators.

Desensitization has also been experimentally produced to these substances. In factories, we have noted workers who first developed a dermatitis after a few days' exposure to a substance and later on, after they continued the work for a period of time, the dermatitis disappeared and immunity developed and they became "hardened", as they themselves call it.

Among the users of substances processed with chemicals, allergy is the principal cause of dermatitis because substances distributed to the public are harmless to by far the larger percentage of people. As for instance, furs, dyes, cosmetics, etc., while used by millions cause dermatitis in comparatively few.

The chief active causes of industrial dermatitis differ in various localities according to the prevailing type of industry. In rural communities, plant poisonings are prevalent, whereas in large manufacturing communities the chemical irritants are the chief causes.

A simple classification of the actual causes of industrial dermatoses is as follows:

1. Mechanical and Physical: Under this heading we have such conditions as cuts, abrasions and pressure. The latter two may cause painful callouses as occur on the heads of the glass blowers from handling heavy blowing iron. Heat, which

may cause scalds in the wet form and burns in the dry form. Cold which causes such conditions as chilblains and frost bites in workers exposed to the weather.

Radiation, both artificial and natural, as from the X-Rays and Radium which causes severe burns and cancers among technicians and radiation from the sun which causes characteristic changes in the skins of such workers as sailors and farmers.

2. Chemical Irritants: These may be divided into general irritants and specific irritants. The general irritants are those which will irritate the skins of all individuals. The specific irritants are those which will irritate the skins of certain hypersensitive individuals. The general irritants may be divided into inorganic and organic. The inorganic irritants can be subdivided into acids and their salts, alkalis, and the salts of irritant metals. The principal inorganic irritant acids and their salts are sulphuric, nitric, hydrochloric, hydrofluoric, chromic and arsenious. The principal irritant alkalis are sodium and potassium hydrate and carbonate and calcium oxide and calcium hydrate. The principal metals which have irritant salts are mercury, chromium, nickel silver, zinc, arsenic, antimony and phosphorous.

The principal organic general irritants can be divided into organic acids and their salts and organic solvents. The principal organic acids which may cause dermatitis are oxalic, carbonic, cresylic, formic, lactic, acetic, valeric, hydrocyanic, phthalic, and abietic. The principal organic solvents which may cause dermatitis are the various alcohols, turpentine, benzol, carbon bisulphide, carbon tetrachloride, trichloroethylene, amyl acetate, butyl acetate, toluol, gasoline and solvent naphtha.

3. Specific Irritants: These cause dermatitis only among certain hypersensitive individuals. Many dyes and their intermediates are skin irritants. Aniline oil, nitro compounds, compounds of naphthalene, benzidine benzanthrone and many of the sulphonic acids. Certain of the dyes cause dermatitis-fur dyes among which are paraphenylenediamine, paramido phenol, aniline black. Leather dyes among which are amido azo toluene, amido azo benzene, Bismarck Brown and Negroine.

Photo developers often cause dermatitis. The principal one is metal. Others are paraphenylenediamine and pyrogallol. Soaps often cause dermatitis because they contain free alkali, especially when mixed with water and also because some of them contain certain substances, such as phenol or cresol, in order to make them disinfectants, and they also sometimes contain perfumes which may irritate hypersensitive individuals.

Many fabric dyes have been reported to have caused dermatitis. I cannot give a full list of them, but some of the principal ones are Crystal Violet, Malachite Green, Methyl Violet, Victoria Blue, Victoria Green, Metanil Yellow, Chrysoidine, Auramine and a great many of the dyes which require chrome mordants.

The rubber compounds and their impurities often cause derma-

atitis. Wild rubber, that is, rubber obtained from wild rubber trees in the forest and gathered by native labor and smoked or cured over fires using certain nuts. Rubber so cured contains many of the products of combustion, such as phenols, acetic acid and the tarry matters all of which are potential skin irritants. Plantation rubber is also cured by smoking but only the surface of it contains the products of combustion and this is easily washed off before the rubber is used for manufacturing purposes. Many of the compounds used in rubber are skin irritants. The accelerators, substances which are used to hasten the vulcanization of rubber, are often the causes of skin irritation among rubber workers. The principal accelerators which have caused dermatitis in the rubber industry are hexamethylenetetramine, commonly called "Hexa", the guanadines, tetramethylthiuram disulphide, commonly called "cuzads", mercapto benzo thiazole, commonly called "Captax", and thiethyl trimethyl triamine, commonly called "Trimene". Para and Ortho toluidine have also caused dermatitis. Anti-oxidants are also used as compounds of rubber in order to prevent the rotting or oxidation of rubber. Phenyl Beta naphthylamine is one of the common rubber anti-oxidants which sometimes cause dermatitis, especially if it should "bloom" out of the rubber.

Many of the insecticides and fungicides are skin irritants. The compounds of mercury, arsenic, fluorine, nicotine, and pyrethrum are the principal irritants in this class of chemicals.

Explosives are also irritants to a considerable number of workers. During the World War, when explosive manufacturing plants had to be rapidly built and safety precautions were at a minimum, dermatitis among munitions workers was very prevalent. Some of the principal offenders among the explosives are trinitro toluene, tetra nitro methyl aniline, trinitro resorcin, fulminate of mercury, picric acid and its compounds and styphnic acid and its compounds.

Dermatitis may also arise in the manufacture and use of cosmetics. Many cosmetics contain irritant dyes and the essential oils used in cosmetics and perfume are nearly all irritants if used in sufficient strength.

The vegetable and the mineral oils are also frequent causes of dermatitis. In machine shops and among mechanics the cutting oils and the cutting compounds are the chief offenders. Petroleum distillates, such as gasoline and lubricating oils also cause dermatitis among garage men, automobile mechanics and other people using them.

Plants: Many plants are irritating to the skins of certain individuals and gardeners and florists may suffer from dermatitis from them. Among the most common plant irritants are Poison Ivy, Sumac, Poison Oak, Pyrethrum, Cocobola, Brazilian Walnut, and Primrose.

Biologic Agents: These are often the cause of industrial dermatitis and may be subdivided into three classes:

1. Parasitic Insects, such as will cause grain itch, straw itch and linseed itch.
2. Bacterial Infections, such as erysipeloid caused by the bacillus of swine erysipelas and occurring among butchers and carcass handlers; anthrax occurring among hide and wool

handlers, and pyogenic infections.

3. Fungus Infections, such as monilia infections, occurring on the hands of fruit packers; Ringworm occurring among barbers, wool sorters, fur handlers, animal handlers and bath attendants.

The actions on the skin of these various causative factors may be classified as follows: Some act as

1. Keratin and Fat Solvents, such as mild alkalis and soaps.
2. Dessicators, or Hygroscopic Agents, which take the water out of the skin, such as sulphuric acid and powerful alkalis.
3. Some are Protein Precipitants, such as heavy metal salts, which form albuminates in combining with the skin.
4. Some are Oxidizers, and by their strong affinity for the hydrogen in water, will liberate oxygen. Such oxidizers are chlorine gas, peroxide of hydrogen, chromic acid and its salts.
5. Then, there are substances which tend to hydrolyze when coming in contact with the moisture of the skin and form irritating compounds. An example of this class is Hexamethylenetetramine, which on hydrolyzing first forms formaldehyder, and then later goes on to formic acid.
6. There are certain substances which act as stimulants to the keratin-forming cells of the skin and are apt to cause new growth. arsenic, petroleum products and aniline compounds are examples of this class.
7. There is still another group which is known as Sensitizers. exposure to them for varying lengths of time, the skin becomes sensitized to their action. These substances may be called Anaphylactoid in their action. The nitro and the nitroso compounds and certain vegetables and fungi belong to this class.

Hypersensitive workers may not immediately upon coming in contact with an irritant, develop a dermatitis. A period of time usually elapses. This period may vary from a few hours to a few days. Some workers develop only a mild dermatitis and are able to continue working and finally they become immune. Others develop so severe a dermatitis that it necessitates their discontinuing work. These usually do not develop an immunity. The immunity, if developed, in most instances lasts only a short time--from a week to a month after discontinuing work. There is one class of worker and fortunately this type is rare, who after working without any trouble for many years, suddenly becomes sensitized to the materials which he formerly handled with safety and develops a severe dermatitis. These workers never become immune. They may even develop a polysensitivity to many of the ordinary materials of daily life and as a result suffer from chronic, incurable eczemas.

DIAGNOSIS

Workers may have inflammations of the skin due to causes other than the hazards of their occupation. Therefore, it is important to determine whether a dermatitis from which a worker is suffering is of industrial origin--first, because it has a direct bearing on the treatment of the case and on the prevention of its recurrence. Second, because of the compen-

sation involved, and third, in order to determine who pays the physician's fee.

If it can be determined that the dermatitis is due to exposure to certain occupational skin hazards, then the major portion of the treatment consists in preventing further contact with these hazards. That is, removing the patient from his working environment, or providing him with suitable protective clothing. In these cases, the prevention of the recurrence of the disease consists in preventing contact with the offending material either by methods stated above or by installing proper safety measures, such as totally enclosed processes, adequate ventilation, clean work rooms, clean clothing, etc.

The compensation laws in many of the States are so worded that if a physician undertakes to treat a worker and he makes a diagnosis that the disease is of industrial origin, then his fee is practically taken care of either by the Compensation Commission or by the Insurance carrier. But, if after treating the patient the physician should determine that the dermatitis is not of industrial origin, the employer or insurance carrier will not pay for the treatment and the physician must look to the patient for his compensation. You can readily see that this has a tendency to make the physician lean towards a diagnosis of industrial dermatitis because very often his chances of being paid a fee by the poor worker is uncertain. It is to the advantage of insurance carriers and Compensation Boards to devise means to remedy this condition.

There is no one factor upon which a diagnosis of industrial dermatitis can be made. In most instances the appearance of the lesion gives no clue to the irritant. Especially is this so in the acute and chronic eczematoid types of occupational dermatoses. All of the following factors must be considered and each one forms only a link in the chain of evidence on which a diagnosis of industrial dermatitis should be made.

1. History: The history of the dermatitis is most important. In order for the dermatitis to be considered as of possible occupational origin, it must be brought out that such a dermatitis was not present before the patient entered on the occupation. It must also be shown that the dermatitis developed during the period of industrial exposure, or after a lapse of a reasonable incubation period since the cessation of exposure. This incubation period should not be over a week. If the history shows that the other workers similarly employed are similarly affected, or that new workers at the process are usually similarly affected, then the possibility of a diagnosis of industrial dermatitis is strengthened. If the history should disclose the fact that the patient has had similar attacks of dermatitis previous to the present exposure, then the possibility of the present attack being due to his occupation is weakened, but not necessarily done away with, because it may be possible that in his previous employment he may have met with the same irritant or conditions which are now causing his dermatitis. Knowledge by the physician of the working processes in which the patient is engaged and the substances with which he comes in contact is important, because this enables him to know whether the worker is exposed to known irritants or to conditions which tend to cause dermatitis. For instance, if a worker appears with a dermatitis of the hands and forearms and states that he works on a rubber mixing mill, then we know that he is exposed to all dermatitis than is a rubber worker who handles only cold vulcanized rubber objects. If the history shows that the dermatitis develops whenever the worker is at work, gets well or improves when he is away from work and again recurs when he returns to work, then the history itself establishes a definite cause and relation factor between the occupation and the dermatitis.

2. Site of Eruption: The site of the eruption is also important. In examining patients, they should be completely divested of clothing. This may in many cases reveal areas of dermatitis on portions of the body not complained of by the patient and may give the clue to a proper diagnosis. Occupational dermatitis usually begins on the exposed parts—the hands, the fingers, and the forearms, if the offending material is a solid or a liquid and also on the face and neck, if it is a vapor. The covered parts of the body may also be affected, if fumes or vapors penetrate the clothing, or if the clothing is not frequently washed and becomes saturated with irritant chemicals. Thus, dermatitis may occur on the body of the worker handling irritant dusts which penetrate the clothing, such as finely powdered rubber compounds. Or dermatitis may occur on the covered parts of the body when the clothing becomes saturated with petroleum oils and waxes, especially if the worker does not take daily cleansing baths and if he does not change his work clothes daily.

Occupational bacillary infections such as erysipeloid of butchers and veru-
ruca necrogenica of cadaver handlers also usually occur on the hands. The malignant pustule of anthrax among hide and leather handlers usually occurs on the head, face, and arms.

Occupational dermatitis is also found at points of friction on the body. The wrist, where the ends of the gloves or sleeves rub; the belt line, where the belt or the top of the trousers cause friction; the ankle at the shoe tops; and the neck at the collar line, are all sites where friction aids the action of industrial irritants. Sometimes a dermatitis of undoubted occupational origin may become generalized. This occurs when the irritant is one to which the worker has developed a high degree of sensitivity. Many substances are known to be sensitizers. Nitro, nitroso and the chloro compounds are notorious sensitizers. In such instances a primary localized dermatitis or burn may also sensitize the patient and a few days later a generalized dermatitis may develop.

3. Characteristic Appearance of Lesions: An industrial dermatitis of the acute eczematoid type begins as an erythema followed by papules and vesicles and when the vesicles break, by an oozing and crusting, no matter what irritant is the cause.

Occupational mycotic infections, such as ringworm among bath attendants, barbers, beauty parlor operators and yeast infections among cannery workers, usually occur on the hands, but the appearance is no different than that of ringworm or yeast infections of non-industrial origin.

There are, however, a few classes of industrial irritants which produce more or less characteristic lesions on certain portions of the body. Paronychia and onycholysis are common lesions among fruit and vegetable canners. The chlorinated naphthalenes and diphenyls produce acne-like lesions on the face and on the parts of the body which come in contact with the work clothes if the work clothes are not frequently washed and changed. Certain tar compounds also cause acne-like lesions on the exposed parts. Oils cause folliculitis and boils, especially on the hairy portions of the body. Petroleum and unrefined paraffin, grease and tar cause keratosis to develop on the hands and forearms, face and scrotum and these keratoses occasionally become malignant. However, the scrotal cancers reported in England as occurring among mule spinners and chimney sweeps have not been noted in this country. Certain hygroscopic chemicals, such as sugar, salt and lime which remove the water from the skin, and solvents, such as the petroleum distillates which remove the fat from the skin, may over a long period of time cause dry fissured eczemas. Exposure to certain aniline derivatives, such as alpha-naphthylamine and benzidine, are known to have caused malignant growths.

Gehrman has reported papillomata of the bladder among the workers exposed over long periods of time to such aniline derivatives. Arsenic, especially if taken internally, has also caused new growths in the form of keratoses and epithelioma, particularly on the palms of the hands and soles of the feet. Keratotic lesions excessive pigmentation around the face and the neck may be occupational among workers exposed to the sun, such as farmers and sailors.

Occupational dermatitis must be differentiated from such diseases as seborrheic dermatitis, fungus, infections, lichen planus, impetigo contagiosa, pityriasis rosea, erythema multiforme, drug eruptions, neurodermatitis and dermatitis due to contact with irritants met with outside of the place of occupation. The industrial physician or the general practitioner may at times be in doubt about the diagnosis of these conditions in a worker exposed to an occupational skin hazard, especially if he does not strip the patient and examine the entire surface of the skin, but to the dermatologist the characteristic location and appearance of seborrheic dermatitis, lichen planus, impetigo contagiosa and neurodermatitis and the generalized eruptions of pityriasis rosea, erythema multiforme and drugs offer no great problem in differential diagnosis from occupational dermatitis. It is true that the presence of these conditions does not bar out the fact that an industrial dermatitis may also be present. In fact, the presence of certain of these skin diseases often predisposes a worker to an industrial dermatitis. The greatest difficulty in differential diagnosis is presented by dermatitis due to contact with substances met outside of the place of occupation. In these cases the lesions are similar in appearance and location, and only the most careful consideration of all the facts can lead to a correct etiology. It is here that the patch test is of greatest value.

The patch test is based on the theory that if a dermatitis is caused by hypersensitivity to a certain substance, then if that substance is applied to an area of unaffected skin of the susceptible individual and left on for a period of time, it will cause an inflammation at the spot where it touches the skin. In doing patch tests, it is important to know what concentrations of certain chemicals can come in contact with the normal skin for a stated period of time without causing an inflammation or reaction. It is also important that no general irritants, such as strong acids or alkalis are used in the patch test, because obviously they will burn any skin. The portion of the body on which a patch test is to be performed is also of importance because it has been found that the different portions may vary in sensitivity to certain chemicals. For instance, the tough horny portions of the skin, such as the palm of the hand, are less susceptible to irritants than is the more tender skin on the inner surface of the forearm. Then again, it has been found that the portion of the skin which is inflamed is more sensitive than other portions of the skin. For this reason, patch tests performed on uninflamed skin adjacent to the eruption are more likely to give positive reactions than when performed on more distant areas. It may even be necessary to wait until the eruption heals, so that patch tests may be performed on the portion of the skin which was actually affected.

If the worker is handling known irritants and his fellow workers are also affected, the cause is obvious and the patch test is unnecessary, but if he is the only one of the group who is affected, then he should be patch tested with the materials with which he comes in contact in the course of his occupation. If he is patched with only one substance then a control patch should be placed on him. If he is patched with more than one substance, then any negative reaction from one of these substances serves as a control. It is also desirable to use as a control

one of the workers who has no dermatitis.

In patching with solids, best results are obtained by moistening them, preferably with perspiration from the patient which can be obtained from the axilla. Sometimes, it may be necessary in order to obtain a reaction from a patch test, to use perspirations of different hydrogen ion concentrations. (Ref. Schwartz, Louis: "Sensitivity to External Irritants in Industry", New York State Journal of Medicine, December 15, 1936, Vol. 36, No. 24).

The results of patch tests must be correlated with the worker's particular occupation, the history of the dermatitis, the site and morphology of the lesion in order to arrive at a correct etiology. Patch tests are only a link in the chain of evidence on which a diagnosis of industrial dermatitis is made. A positive reaction only shows that the portion of the skin on which the patch was applied was at that time sensitive to the substance with which patched. In order to state that this substance was the cause of the occupational dermatitis, we must be sure that the patient was exposed to the substance in the course of his work and presuppose that the patient's skin was also sensitive at the time of industrial exposure.

When negative results are obtained from patch tests with the materials met with in the course of the patient's occupation, we must not hastily conclude that the dermatitis is not of industrial origin, because the skin area over which the patch was placed may not be hypersensitive. Or, if the eruption has disappeared, the patient may no longer be sensitive when the patch test is performed but may have been sensitive at the time he had the eruption and when he was industrially exposed. Or, a negative patch test reaction may be due to the fact that the patch test never accurately reproduces actual working conditions, such as friction, maceration, heat, cold and sunlight, which may be additional factors adding to the irritating effect of the substance to which the patient is exposed. Or, it may be that the concentration of the chemicals applied as a patch test may not be as great as they actually were during industrial exposure. Or, finally, the actual industrial irritant may not have been discovered and applied as a patch test. When negative reactions are obtained from patch tests with substances encountered in the work room and the dermatitis which the worker has resembles a contact dermatitis, an effort must be made to perform patch tests with materials met with in the patient's home which may be the causes of dermatitis. For instance, certain plants, or perhaps paints or even new furniture. Tests of this kind will in some cases show that the patient is sensitive to materials met with outside of industry and not sensitive to the materials which he meets in his place of employment.

The technic of performing patch tests is important in obtaining and evaluating results. When patch testing for hypersensitivity to general irritants such dilutions must be used in the tests as are known not to irritate the normal skin. (Ref. Schwartz, Louis: "Sensitivity to External Irritants in Industry", New York State Journal of Medicine, December 15, 1936, Vol. 36, No. 24). The insulating materials inserted between the chemical and the adhesive plaster should be a non-irritant substance, such as unvarnished cellulose, or better still, a thin sheet of mica may be used. The resin on waterproof cellophane itself may be an irritant, as may be some of the compounds in dental rubber. The adhesive plaster used to hold the patch in place often itself causes an erythema of the skin. (Ref. Schwartz, Louis and Peck, Samuel H.: "The Irritants in Adhesive Plaster", Public Health Reports, June 14, 1935, Vol. 50, No. 24).

At the time the patches are removed there may be no reaction present, but some time later (a few hours to a few days) a delayed reaction may develop

at the site of the patch. There is some dispute as to the significance of delayed reactions. Some hold that the skin was sensitized by the patch test, but I think that in our present state of knowledge we should regard delayed reactions as denoting hypersensitivity just as undelayed reactions do. Patch tests properly performed and evaluated can be of great help in the diagnosis of industrial dermatitis, but if improperly performed and evaluated, they may lead to confusing and unjust conclusions.

Fungus infections also offer a problem in differential diagnosis from industrial dermatitis. A large percentage of workers are affected with mycotic infections in some form or another. Epidermophytosis, trichophytosis, tinea cruris and tinea versicolor are common skin diseases. Allergic reactions in the form of dermatosis on distant parts of the body resulting from these fungus infections are recognized by allergists and dermatologists. These allergic reactions or phytids may be confused with industrial dermatitis. If the phytids or the mycotic infections appear on portions of the body not exposed to industrial irritants, they are not so apt to cause doubts in diagnosis, but they often appear on the hands and here they are apt to cause trouble in diagnosis. The various tests with fungus extracts, such as trichophytin, are of little value in making a differential diagnosis because nearly every one has or has had a fungus infection and positive reactions are the rule. They are of more value when the tests are negative, because then they tend to show that the present eruption is not of fungus origin, although even here it is not absolute proof, because among other reasons the causative fungus may not be the one from which the testing extract is made. Then again, the fact that a worker has a fungus infection does not preclude the fact that he may also have an industrial dermatitis. In fact, it is held by some dermatologists that the presence of a fungus infection predisposes to hypersensitivity to other external irritants. Patch testing may also offer some help but here again the industrial exposure, the history of the eruption, the site of the lesions and their morphology must all be carefully considered before a diagnosis is made.

Chronic eczemas, more or less generalized and of long standing, offer very difficult problems in etiology, especially when they are complicated by secondary infections. Patch tests are of little value in most of these cases, because polysensitivity is usually present. It is impossible to determine whether the dermatitis and polysensitivity was caused by industrial exposure or whether the dermatitis and sensitivity was caused by exposure to substances encountered outside of the work room.

Cases sometimes appear before Compensation Boards claiming compensation for disability due to a dermatitis which the worker claims to have suffered as a result of his occupation, but which at the time the case is being heard, has disappeared. In these cases it is also difficult to determine the causative factor. If patch tests done at this time are positive, they are of great help, but if negative, they are not, because the patient may have developed an immunity by his recovery.

From these facts it can be seen that there is no one characteristic symptom on which a diagnosis of an industrial dermatitis can be made. The worker's occupation, the history of his skin eruption, its site and morphology and evaluation of the patch tests must all be taken into consideration by a dermatologist familiar with the substances and the

processes of the worker's occupation before we can hope to make a true diagnosis as to the etiology of a dermatitis in a worker exposed to an occupational skin hazard. However, these two cardinal rules are usually true. Read Chart 13.

CANCER IN INDUSTRY

Cancerous changes in the skin may occur in wounds or scars caused by industrial accidents, such as burns and wounds. Cancerous degeneration also occurs in so-called sailors' and farmers' skins, supposedly due to overexposure to the ultra violet rays of the sun. Radium and X-Ray cancers among technicians and physicians we are familiar with. Certain chemicals are also said to cause cancers of the skin. The soot cancer of chimney sweeps described in England has not been described in America. Coal tar, certain oils, and petroleum products predispose to the development of cancer.

In our studies in oil refineries we have verified this fact. Workers in arsenic also tend to develop keratotic lesions on the exposed parts. Such lesions have been reported by European observers, but in our studies in factories where arsenical insecticides are manufactured, we have failed to note their occurrence. We have, however, seen such lesions occurring as a result of the internal administration of arsenic over long periods of time.

An unusual number of papillomata and carcinomata of the bladder has been found to occur among synthetic dye workers both in Europe and in America.

Workers exposed to such dye bases as Anthracene, Benzidine and Alpha-Naphtylamine, are said to have cancers of the bladder occurring amongst them thirty times more frequently than the average population.

Aniline and its derivatives are also said to cause the occurrence of cancer. Cancers of the liver have been experimentally produced in rats by feeding them certain aniline dyes, amido azotoluene and also by rubbing certain of these aniline dyes into wounds of the skin. Other substances said to cause cancer are Toluidin, Xylol, Xyloidin, Cumidin, Anisidin, Dyphenylamin, Naphthol, Kong-Red, Safranin, Benzopurpurin and Blue-Rosanilin.

TREATMENT OF INDUSTRIAL DERMATOSES

A person who is so sensitive to the material with which he works that he cannot develop an immunity, should seek some other occupation. Most of the cases of industrial dermatitis develop on new employees and are usually mild in character. Such workers should be given a protective ointment to put over the exposed parts, proper protective clothing, such as rubber gloves, aprons, etc., and kept on the job. Most of them will develop immunity. Those who do not, should be given other work to do where they will not come in contact with the irritant. This usually effects a cure.

In applying medication to the lesions, only the mildest form of ointments or lotions should be used, such as boric acid or calamine. Strong ointments are apt to irritate the skin and cause more dermatitis.

Desensitization has not met with any encouraging results.

PREVENTION

The ideal prevention is to so safeguard the operations that injurious chemicals do not come in contact with the skin. This can be done by installing totally enclosed processes. Modern factories are being built so that the worker need not come in contact with any of the chemicals used from the beginning of the

manufacturing process to the very end. New chemicals are brought to the factory in enclosed railroad cars, emptied by suction hose into enclosed storage containers from which they are sent by turning valves to the various retorts and kettles through a closed system. From these kettles the finished product is processed in totally enclosed machinery, such as filters, grinders, etc., and placed into closed shipping containers by means of hose or pipes. In some industrial processes, where this is not possible, or where there is old equipment, the workers must be protected by being compelled to wear suitable protective clothing, such as rubber gloves, aprons, masks, and goggles. This protective clothing should be frequently cleaned and laundered. Over processes from which are given off irritating dusts or fumes, a special suction ventilating apparatus should be installed so that these irritant can not come in contact with the worker. The work rooms themselves should have proper and adequate ventilating equipment so that dusts and fumes have no chance to accumulate in any concentration. Rules for the use of protective clothing should be enforced. When respirators or gas masks are furnished, the men should be compelled to wear them.

Cleanliness is of prime importance. The floors and walls of the work rooms should be kept scrupulously clean as should all machinery. Convenient shower baths should be installed and the worker should be compelled to take baths after work. In factories where there are special skin hazards, a double set of locker rooms should be provided, that is, one for street clothes and one for depositing the soiled work clothes. In this way, the worker coming to work will enter the first locker where he will strip and leave his clothes in the locker. From there he proceeds through a hall to the second locker where clean work clothes are waiting for him. From this locker room he proceeds to his work. At the end of the work shift, he enters the locker room in which he had put on his clean work clothes. Here he deposits his soiled work clothes and goes to the shower baths. From the shower baths he proceeds to the locker room where he had left his street clothes and then goes home.

There are many protective ointments on the market designed for the prevention of industrial dermatoses. These ointments are supposed to act by forming a protective film over the skin and thus to prevent the irritant from coming in contact with the skin. Some of them are useful, others only imperfectly perform their functions. They may be easily rubbed off during work or may be washed off by the perspiration. Some of them may offer protection against a particular chemical. There are very few of them, however, which can protect against strong alkalis. There are instances where a protective ointment may be of value, especially if used during the time pending installation of the more permanent protective measure which I have described. Protective ointments are usually washed off by the worker before leaving the factory. This act also removes the irritant chemicals from the skin and is no small part of the protection supposed to be given by the ointment. Ointments should never be relied upon as permanent preventive measures, but only as temporary expedients pending the installation of proper safety appliances.

New applicants for work should be carefully examined and those having such predisposing skin conditions as previously stated should not be employed in occupations where there is a skin hazard. In occupations which present serious skin hazards, all applicants for jobs should be stripped during the medical examination and should be patch tested with the materials with which they are to work and if found to be hypersensitive, they should be rejected. The workers should be encouraged to report to the medical department all irritations of the skin, no matter how trivial. There should be frequent medical examinations of the workers to discover the presence of skin diseases which have not been

reported. The physician in charge should have a working knowledge of dermatology and give proper treatment for these cases, or if he does not have such knowledge, dermatological cases should be sent to a consulting dermatologist.

It is not always necessary to remove the worker from his job in order that he may get well. Indeed, it is sometimes better to allow him to work, if he has a mild dermatitis, and treat him while working in the hope that he will develop an immunity or become "hardened", as the workers themselves call it. A great many workers will become hardened and the immunity thus acquired usually lasts while working and sometimes will continue for a considerable period after contact with the chemical has ceased so that the worker may stay away from his job for two or three weeks and come back and still be immune. If, however, he stays away for a few months, he will in most cases lose his immunity and will again have to go through the process of becoming "hardened". Those workers who develop such a severe dermatitis that they can not get well while working, should be removed from the job and when they have become well, they should be placed in some other part of the plant where they will not come in contact with that irritant.

Municipal and State laws should be formulated to compel factories to report all cases of occupational dermatitis to either the Department of Labor or Health and these Departments in turn should report their cases to the United States Public Health Service. It is only by knowing where industrial dermatitis occurs that we can hope to be successfully cope with it.

Municipal and State Departments of Labor or health should have divisions of industrial hygiene within their departments, the inspectors of which should be required to make periodic inspections of factories in order that they may note the occurrence of occupational dermatitis and the safety precautions adopted for its prevention. Special studies should be made by the Bureau of Industrial Hygiene of unusual outbreaks of dermatitis in order to determine the cause and to institute preventive measures. Bureaus of Industrial Hygiene should strive for the passage of laws compelling factories to install proper safety devices against occupational dermatitis.

Insurance carriers should also have trained inspectors to inspect the factories of proposed risks before accepting them. If preventable hazards are found, the risk should not be accepted until proper safety devices are installed. Insurance carriers should also have trained sanitary and safety engineers could be used for the safety of the worker and the financial benefit of the insurance carrier. The inspectors of insurance carriers can acquaint the superintendents and foremen of factories concerning the hazards present in the factory and how to guard against them and the foremen can in turn acquaint the workers.

The manufacturers of chemicals should have laboratories to study the health hazards of their products and how to guard against them and they should impart this knowledge to their customers.

Plant physicians should take every opportunity to teach safety and cleanliness to the workers. They should also carefully investigate the causes of any unusual occurrence of dermatitis and, if necessary, call in consulting dermatologists and safety engineers to help discover the cause and to institute proper measures to prevent recurrences. Plant physicians should also advocate pre-employment examinations and the regular periodic examinations of workers.

[illegible][illegible]

1. The first step in the process of the investigation is the identification of the problem. This is done by the investigator who is assigned to the case. The investigator must first determine the nature of the problem and the scope of the investigation. This is done by reviewing the available information and by conducting interviews with the relevant parties. The investigator must also determine the objectives of the investigation and the methods to be used.

2. Once the problem is identified, the next step is to define the objectives and goals of the project. This helps to clarify what needs to be achieved and provides a clear direction for the team.

3. The third step is to develop a plan or strategy to address the problem. This involves breaking down the problem into smaller, manageable tasks and determining the resources needed to complete each task.

4. The fourth step is to implement the plan. This involves putting the strategy into action and monitoring progress to ensure that the project is on track.

5. The final step is to evaluate the results of the project. This involves assessing the outcomes against the objectives and goals and identifying any areas for improvement.

LECTURE D-VI

DUSTS IN RELATION TO DISEASES OF THE LUNGS Leroy H. Gardner, M.D.

(Outline of lecture)

I. Pneumoconiosis - a condition of the lungs induced by prolonged inhalation of dust. The tissue reaction varies from simple phagocytosis to fibrosis depending upon the composition of the inhaled particles.

II. FOUR MAJOR TYPES OF PNEUMOCONIOSIS:

1. Simple benign-reaction to most kinds of inorganic dust.
2. Silicosis-reaction to free silica.
3. Modified silicosis-reaction to free silica mixed with large amounts of other minerals.
4. Asbestosis-reaction to any one of a group of silicates having fibrous structure.
5. Other forms not yet differentiated by basic anatomical study to supplement impressions obtained from roentgenograms alone. Some of these suggested forms may actually be modified silicosis, others may be largely the result of infection; some may actually be specific types of dust reaction. It is also possible that accumulations of radio-opaque particles may themselves cast shadows on an X-ray film.

Essential features of the four types:

1. Simple benign.

A. Anatomic characteristics - minor chronic inflammatory changes around the major lymphatic trunks with no significant effect upon functional tissues. Distribution largely perivascular and in extreme cases involving interlobular septa.

B. X-ray pattern - generalized exaggeration of the normal vascular tree shadows. Superimposed reticulation when septa are heavily involved.

C. Specificity - linear exaggeration in X-ray may result from any cause which widens blood vessels, more blood in lumen, thickening of wall, deposits of dust about vessel. Dust of any kind, including free silica, may have latter effect.

Linear exaggeration may be a precursor of specific silicotic nodulation but this outcome cannot be inferred even with exposure to free silica.

D. Clinical significance - usually none. With certain types of dust, particularly where there is associated reticulation, there may be complaints of dyspnea.

E. Susceptibility to pulmonary infection - generally unaltered.

2. Silicosis.

A. Anatomic characteristics

- a. In a lung free of infection - discrete, 2 to 4 mm nodules of hyaline fibrous tissue, uniformly distributed throughout all parts of both lungs.
- b. In association with healed or latent chronic infection of lungs - tendency for excess of nodules to form about infectious lesion, developing large areas of fibrosis. Compensatory emphysema and chronic adhesive pleurisy.

B. X-ray pattern

- a. Without infection - generalized nodulation involving all parts of both lungs. In advanced cases the costophrenic regions may be clear due to subsequently developing emphysema.
- b. With associated infection - massive conglomerate shadows on a background of generalized nodulation. In advanced cases contraction of massive fibrosis and emphysema may disturb the pattern of generalized nodulation.

C. Specificity - produced only by dust high in free silica, typified by quartz. Silica combined as silicates has no such effect.

D. Clinical significance.

- a. Without infection most cases have no symptoms or at most dyspnoea on exertion only. Disability none. The pulmonary reserve is sufficient to meet all ordinary demands.
- b. With infection, dyspnoea is always more marked. If infection is healed but fibrosis massive and emphysema marked there may be dyspnoea at rest and real disability.

If infection is still active symptoms of intoxication may not be manifest for years and marked disability may be deferred till a short time before death.

E. Susceptibility to pulmonary infection. Specific increase in susceptibility to tubercle bacillus but not to pneumococcus or other organisms.

- a. If latent tuberculosis antedates silicosis the infection may be reactivated producing the very chronic combined lesion known as tuberculosilicosis. This is a specific kind of disease differing from either silicosis or tuberculosis in pure form with a bad ultimate prognosis.
- b. If tuberculosis is superimposed upon a pre-existing active silicosis the infection is apt to run an acute course and terminate fatally in a few months.

3. Modified Silicosis

A. Anatomic characteristics. The silicotic nodules are surrounded by zones of cellular connective tissue containing large amounts of non-siliceous dust.

Similar depositis occur about major lymphatic trunks and often connect adjacent silicotic nodules. Reaction to non-siliceous dusts may result in small conglomerations of nodules. Infection probably essential for the larger ones.

B. X-ray pattern - usually very similar to that of silicosis. The perilymphatic linear reactions may cause some reticulation and occasionally the shadows of the nodules are less clear cut.

C. Specificity. The basic irritant is free silica; the extent and severity of the modification is dependent upon the concentration and character of other minerals.

D. Clinical significance - as in less complicated silicosis.

E. Susceptibility to infection - ditto. Certain associated minerals like iron or coal may favor healing of the infection but massive fibrosis nevertheless ensues.

4. Asbestosis

A. Anatomic characteristics a diffuse, non-nodular fibrosis of the parenchyma of the lung starting as sleeves about the terminal bronchioles. Groups of patent air spaces persist throughout the scar tissue. Peripheral bronchioles are dilated producing thin-walled blebs. Chronic adhesive fibrous pleurisy. "Asbestos bodies" - iron-staining protein deposits about inhaled fibres. Diagnostic when mineral associated with characteristic fibrosis.

B. X-ray pattern. Light diffuse haze to an obliterating "ground appearance" most marked in the lower thirds of both lungs. In advanced cases pleuropericardial adhesion may produce the "porcupine heart" shadow.

C. Specificity. Produced only by fibrous silicates known as asbestos. Occasional asbestos bodies found in routine histologic examination of lungs due to accidentally inhaled asbestos.

D. Clinical significance. Dyspnoea more marked in proportion to apparent severity of reaction seen in roentgenogram. Disability may be total.

E. Susceptibility to infection. Many die of broncho-pneumonia; several reported autopsies of death due to tuberculosis; surveys of employed asbestos workers show no excess TB. Experimentally no alteration of native susceptibility to tubercle bacillus.

III. ETIOLOGY. Prolonged exposure to excessive concentrations of very fine dust. These conditions obtain only in industry. Defense mechanisms of body adequate to protect any dusts in state of nature. Hence pneumoconiosis is strictly an industrial condition.

1. Duration of exposure bears reciprocal relationship to concentration and

particle size. Measured in years and not months or weeks. Except under very unusual conditions exposures of 10 to 12 years essential to produce silicosis; for asbestosis 7 to 8 years. Silicosis never less than $1\frac{1}{2}$ to 2 years.

2. Concentration. Usually expressed in millions of particles per cu. ft. of air. May be in mgs/cu. ft. Determined by microscopic counts of samples collected in impingers or other apparatus.

3. Particle Size. Particles must be small enough to stay in atmospheric suspension and still smaller to be inhaled into lung. Not many larger than 10 microns found within terminal air spaces. Fibres of asbestos an exception - some inside lungs measure 100 to 200 microns.

Experiment shows that quartz particles larger than 3 microns have no specific irritating action.

4. Mineralogical Composition - best determined by X-ray diffraction analysis. Nature of particle determines its capacity to irritate.

A. Free silica is a chemical poison whose action is proportional to amount of surface in contact with living cells. Cover the surface chemically with alumina or mechanically by grinding with soft iron and quartz becomes inert. Therefore, its action is not mechanical.

B. Asbestos - may be a mechanical irritant although experimental proof is not complete.

C. Other minerals exert minor degrees of injury and resultant reaction usually short of true fibrosis.

IV. DIAGNOSIS. - Based upon three essentials:

1. History of adequate exposure to industrial dust.
2. Demonstration of x-ray pattern of the particular pneumoconiosis.
3. Physical examination.

(1.) History of exposures.

A. Occupational history must cover entire industrial lifetime. Last occupation may be misleading because partially disabled men often employed in jobs like watchman and elevator operators.

B. Should include both industry and particular job with approximate dates of each employment.

e.g. All foundrymen not exposed to quartz dust but chippers and sand-blasters are. Soft coal miners frequently exposed to no quartz but most metal miners are.

C. Determine nature of materials used. Grinders may have used sandstone wheels (90% plus free silica) in the past; today they use emery, carborundum or aluminum oxide wheels that cause no silicosis. But grinders in foundries may grind siliceous sand from castings and so be exposed to a hazard.

(2) Characteristic X-ray patterns.

- A. Exaggerated linear marking and perhaps reticulation of simple benign pneumoconioses.
- B. Generalized discrete nodulation of simple silicosis.
- C. Nodulation with localized conglomerate shadows of silicosis plus infection, usually tuberculosis in healed or latent stages. Tuberculo-silicosis.
- D. Nodulation plus superimposed patterns of typical tuberculosis. The less commonly associated conditions running an acute course.
- E. Diffuse haziness of both lower lung fields of asbestosis.

(3) Physical Examination.

- A. In the uncomplicated case characterized by paucity of signs in comparison with extent of x-ray findings. Signs present those of emphysema and perhaps chronic bronchitis.
- B. In cases complicated by infection, physical examination may be more helpful.
- C. Determination of disability if present.
- D. Sputum examination.
 - a. Mineral particles of little significance. X-ray diffraction necessary for identification of small sizes.
 - b. Tubercle bacilli. In tuberculo-silicosis absent for years until shortly anti-mortem. In silicosis with typical tuberculo-silicosis occur early.

V. COMPLICATIONS OF SILICOSIS.

1. Lungs.

Emphysema - in advanced cases.
Chronic pleurisy in conglomerate types.
Tuberculosis - the most common and the major cause of disability.
Other infections - not unduly common.
Primary carcinoma of lung - probably no specific relationship. Silicosis most common in cancer age and repeated x-ray examinations of silicotics reveal more early lung cancer.

2. Heart.

Uncomplicated discrete nodulation, no effect upon heart.
Conglomerate fibrosis of tuberculo-silicosis has had effect upon pre-existing heart disease common in the age period (over 45) when silicosis most prevalent.
Debatable whether tuberculo-silicosis actually initiates disease in a normal heart.

Other Organs.

No proof of any effect upon other viscera. Large silicotic abdominal lymph nodes sometimes cause symptoms from pressure.

The Nature of Industrial Exposure to the Metals

The most important route for the absorption of metals under the conditions of industrial exposure is by way of inhalation of vapor, fume, dust and spray, and subsequent penetration of the pulmonary membrane. However, except in the case of true vapors and of particles so small as to behave like vapors, there is always ample opportunity for alimentary absorption of inhaled metallic compounds. Within various ranges of particle size, twenty-five to eighty per cent of the particulate matter entering the upper respiratory tract is removed without reaching the pulmonary alveolae, some being exhaled, the remainder being deposited en route and eventually being swallowed. (Some, of course, is absorbed in the upper respiratory tract.) By reason of the greater ease of absorption of most metallic compounds in the lung as contrasted with the alimentary tract, the smaller quantity of material in the alveolae of the lung represents the greater toxic hazard. Nevertheless the present tendency among industrial hygienists to disregard the alimentary absorption of the metals is an oversight which is of greatest practical importance when it results in disregard of the opportunities for the ingestion of these compounds by industrial workers.

This brings us to another type of exposure - that which results from taking into the mouth and swallowing certain quantities of potentially toxic agents. It is quite unnecessary that the food and drink of workmen be contaminated with the substances with which they work, but it is not unusual to find that they are. Facilities for adequate personal hygiene are not uniformly available in industrial plants, and spotless and dustless lockers and lunch rooms are not always found. All too often the men have lunch boxes and bottles of milk in work rooms or in contaminated adjacent rooms, and all too frequently they handle tobacco, and eat or drink snacks or even regular meals without properly washing. They even put tools and other contaminated articles into their mouths. The example of brushes of the workers with luminous paints is well known. Other examples could also be cited. These are matters of ordinary sanitation and housekeeping which, perhaps were emphasized beyond their relative importance in an earlier period, but which sometimes are neglected in the midst of the modern emphasis on air contamination alone.

In general the percutaneous absorption of metallic compounds is of little importance among industrial workmen. There are notable exceptions, however; the alkyl lead and mercury compounds are readily absorbable through the skin, as are also the carbonyls, including iron pentacarbonyl. The generalization is justified that when a metallic or organo-metallic or organo-metallic compound is a liquid at ordinary temperature or at body temperature, one may suspect that it will penetrate the unbroken skin with dangerous speed. Solutions of metallic compounds in water were not appreciably absorbed through the intact skin as a general thing, and there are few if any demonstrated instances of percutaneous absorption of inorganic metallic compounds out of organic or oil solvents, but it should be remembered that new organic vehicles for metallic compounds need to be investigated in this regard if they are to be employed under conditions in which cutaneous contact will occur.

General and Specific Clinical Characteristics of Industrial

Intoxication Induced by the Absorption of Metals

I. Lead Poisoning

1. General - Involvement of muscle both smooth and striate, voluntary and involuntary neuro-muscular apparatus, central nervous system and hematopoietic tissues.

INTOXICATION BY METALS

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Human absorption of metallic compounds in the course of industrial employment, and as the consequence of industrial processes, is a matter of frequent, almost regular, occurrence. Moreover as new technological procedures develop and gain widespread application a greater number of previously unused metals and metallic compounds come under consideration from the viewpoint of their physiological effects upon man. It is a temptation, therefore, to examine these materials as a group and to consider what are their common characteristics, and what general physiological or clinical principles are involved in the prevention, recognition and medical treatment of intoxications that may arise from their absorption. We shall make some attempt in that direction from time to time in the discussion that ensues, but any comprehensive consideration of the subject in this vein would take us far from the practical matters that must be dealt with. Moreover the conclusions that would result from such a discussion would be far from satisfying except as an intellectual exercise, since we should find that most of the facts needed to demonstrate both the similarities and differences in the physiological behavior of the metals and their compounds are yet to be obtained by the patient and elaborate research. Accordingly we shall try briefly to summarize the more useful facts on several of the metals that give rise to industrial poisoning at present, while illustrating the type of information that should be obtained generally, through examples of data that are now available on only a few.

The metals that are responsible for most of the cases of systemic intoxication that occur in connection with present day industrial operations, (as differentiated from contact dermatoses), may be listed in the approximately decreasing order of their frequency of occurrence as follows; lead, zinc, mercury, cadmium, radium, nickel, iron and thallium. The exact position of several of these metals in the list is somewhat uncertain in view of the present status of statistics on morbidity, but that of lead is in no doubt since it heads the list by a very large margin. Zinc must be included because of its importance, in common with certain other metals such as beryllium, magnesium and perhaps copper, in the production of metal fume fever. However, the latter is hardly to be considered a manifestation of metal poisoning, and no further reference will be made to it or to zinc. Copper is not listed for the reason that industrial copper poisoning does not appear to have occurred. This is a strange and interesting fact in view of the high toxicity of copper salts for simple organisms and the serious character of copper poisoning seen in sheep as the consequence of the ingestion of copper sulfate introduced into the rock salt supplied on the range. (Bull. No. 499, Texas, Agric. Exper. Station, Dec. 1934). It seems probable that both the dosage and the mode of absorption are important factors of safety under the conditions of industrial exposure to copper, but we shall not be in position to understand this peculiarity of copper nor the apparent vagaries in the behavior of other metals in the complex human organism, until we have learned how they are transported and distributed in the tissues and how and in what form they are eliminated. Our knowledge of these matters is quite fragmentary for most metals.

For present purposes one might be led to include in this discussion certain elements the poisonous effects of which resemble those induced by toxic doses of the metals, as arsenic, antimony, selenium, tellurium, chromium, manganese and certain less common elements. However, in addition to the fact that time will not permit more than this passing reference, none of these elements is regarded as a metal in the strict sense of the word. Therefore, only one of them, manganese, will be added to the list given above, because of its practical importance as the cause of a systemic type of industrial intoxication.

of skin and appendages (also conjunctival and upper respiratory irritation)

4. References

- P. A. Neal et al; U.S. Pub. Health Bull., 234: (1937) and 263 (1941).
D. A. Hunter et al; Quart. J. Med; 9:193 (1940)

III. Cadmium Poisoning

1. General - Involvement of respiratory tract in acute toxic and inflammatory process, with similar involvement of alimentary tract under suitable conditions and especially following repeated low-grade exposures.

2. Clinical types

- (1) Acute (mortality high at severe levels of exposure, with no permanent sequelae in survivors)

Respiratory manifestations - naso-pharyngitis, tracheitis, constricting pain, cough, pulmonary irritation, dyspnoea, pulmonary edema, cyanosis and asphyxial syndrome. Gastro-enteric manifestations - metallic taste, pharyngitis (swelling and pain on swallowing), nausea, vomiting, diarrhoea.

Other manifestations - headache, dizziness, weakness, chills, increasing sometimes to great severity, fever (terminally).

- (2) Chronic - headache, nausea, vomiting and diarrhoea, with respiratory manifestations of minor importance.

3. References

- L. Prodan, J. Ind. Hyg. and Toxicol. 14:132 (1932)
Division of Hygiene, National Institute of Health, U. S. Pub. Health Reports, 57:601 (1942)
E. T. Johnstone, Occupational Disease, Saunders, Philadelphia, 1941

IV. Radium and Mesothorium Poisoning

1. General - A chronic disease involving long-continued effects upon osseous tissue in which the metal is retained and upon the bone marrow contiguous thereto.

2. Clinical characteristics - (low morbidity with high incidence of progressively increasing disability and eventual fatality)

Skeletal manifestations - radiation osteitis characterized in stage

- (1) by bone marrow changes (irritative hyperplasia with basophilic stippling followed by destructive action causing aplastic anemia); in stage (2) by replacement of bone marrow with cellular fibrous tissue; in stage (3) by appearance of non-cellular fibrous tissue with decalcification - (may be associated with spontaneous fractures).

Osteitis of jaw associated with secondary infection results in necrosis of mandible. Single or multiple osteosarcomata

Hematological manifestations - changes in peripheral blood represented by basophilic stippling and varying forms of anemia, sometimes regenerative and sometimes aplastic apparently determined by stage and or speed of development of process

Upper respiratory manifestations

Carcinoma involving paranasal sinuses

3. References

- H. S. Martland, J. Am. Med. Assoc., 92:466, 522, (1929)
R. D. Evans et al - Series of papers, latest of which, with references to others found in J. Ind. Hyg. and Toxicol. 24:116 (1942)

2. Clinical types

- (1) Gastro-enteric - (mild form, with highest morbidity, insignificant mortality and negligible permanent disability)
Anorexia, foul taste in mouth, constipation (occasionally diarrhoea), abdominal distress, colic, general weakness and loss of weight.

- (2) Neuro-muscular (voluntary) - (severe form with low incidence, low mortality, and high relative incidence of permanent sequelae).

Extensor pareses and paralyses, especially wrist-drop.

- (3) Cerebral - (Most severe form, with very low incidence, high mortality, and moderate incidence of permanent disability)

Encephalopathy

- (4) Mixed types

Combining various characteristics of above types.

- (5) Generally associated evidences of intoxication in all types

Vascular (cutaneous pallor, lowered vascular tone, bradycardia.

Hematopoietic (hyperplasia, toxic changes, aplasia (?), i.e. basophilic stippling, anisocytosis, anemia) Weakness, inanition, loss of weight, arthralgia, low body temperature.

- (6) Special type - (low incidence, high mortality, negligible incidence of permanent sequelae)

Tetraethyl lead poisoning always acute cerebral excitation without hematological manifestations, and with little evidence of associated visceral intoxication.

3. Reference

Report of Committee on Lead Poisoning, Am. Public Health Assn., New York 1943.

II. Mercury Poisoning

1. General - An essentially chronic disease involving the central system, and to lesser extent mucous membranes and excretory tissues.

2. Clinical characteristics - (low morbidity at present and low mortality, with slow recovery and high incidence of permanent sequelae in well-established cases)

CNS manifestations - fine tremors of fingers, lips and tongue, mental aberration and deterioration

Gastro-enteric manifestations - nausea, vomiting, diarrhoea, gingival irritation, bleeding and inflammation, stomatitis, salivation, foul taste and breath, anorexia with weakness and loss of weight.

Renal manifestations - edema and irritation (albuminuria and hematuria, excretory dysfunction (retention of mercury, etc.).

3. Special types

- (1) Intoxication from alkyl mercury compounds - focal cerebral lesions characterized by ataxia, disarthria, lateral column and ocular lesions.

- (2) Local effects (mercury fulminate) - irritation, erosion and ulceration

Diagnosis of Metallic Poisoning in Industrial Workmen

The diagnosis of poisoning resulting from industrial exposure to toxic metals is made by methods corresponding to those employed in other types of illness. One aspect of the problem is somewhat different in that such diagnoses often require substantial proof of their correctness, not only for the purpose of instituting adequate measures for the elimination of further hazardous exposure, but also to substantiate the validity of a claim for compensation. It is necessary, therefore, to do a thorough clinical job, to obtain the evidence necessary to establish the facts, and to follow up the case or cases so as to determine the extent and the duration of disability.

The steps in the diagnostic procedure may be defined briefly.

1. Establishment of the Character, Severity, and Duration of the Occupational Exposure

It may be possible to accomplish this purpose by interviewing the patient. Often and in fact usually, this is inadequate. Generally it is necessary to investigate plant processes and conditions in order to evaluate the potential hazard of the exposure to any metallic fume, dust, or vapor. The value of the "history of occupational exposure," so frequently referred to as one essential in the triad of diagnostic procedures, is commonly overemphasized. It is valuable or misleading to the extent that it is factual on the one hand or supposititious on the other. It is rarely more than a working diagnostic lead. Physicians who deal with industrial intoxications are not justified in drawing inferences and in making snap judgments, but unfortunately they do so commonly.

2. Elicitation of Symptomatic Information and Physical Signs of Illness or Disease

Little need be said to the careful clinician concerning the importance of obtaining a detailed history of the onset and course of an illness. There are certain points, however, that may be emphasized. The relationship of occupation to illness must be examined in terms of the entire occupational history of an individual, since the occupation at the onset of the illness may be quite different from that responsible for the illness. Another feature of importance is the history of previous illnesses and their relation to a present or previous occupation. Inquiry concerning time lost from work is often fruitful of information. Of still greater importance is the establishment of the date of the last workday - i.e., the last day of exposure - in relation to the onset of illness, the severity of present symptoms, and the interpretation of laboratory data.

The value of a comprehensive physical examination is also referred to generally in speech, but the actual performance and recording of the observations commonly leave much to be desired. Detailed clinical observations are necessary to arrive at a sound diagnosis and even the best of skill may be inadequate. Clearly, therefore, nothing but confusion can result from a cursory and incomplete examination. The neurological findings are often of the greatest importance and therefore a thorough neurological examination must be made. It is not always easy or even possible for the general clinician to make the nicely balanced interpretation of neurological data required for differential diagnosis, but there is little excuse for his neglect of systematic tests of the function of cranial and segmental nerves and end organs.

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V. Manganese Poisoning

1. General - a chronic disease characterized by nervous manifestations and focal degenerative lesions of the brain and spinal cord.
2. Clinical characteristics (relatively high incidence of permanent disability, with negligible mortality)
 - Cerebral manifestations - extra pyramidal and basal ganglion involvement, with stuttering, monotonous and economical speech, masked facies (inconstant), and lingual tremor, without mental impairment.
 - Cord manifestations - coarse tremor of hands increased by volition, apastic gait on broad base, incoordination, tendency to fall, motor ataxia, muscular spasms, and impairment of motor power.
 - Hematological manifestations - apparently inconstant leucopenia, and anemia.
3. References
 - P. H. Flinn, et al, U. S. Public Health Bull. 247 (1940)
 - L. H. Fairhall and P. A. Heal, National Institute of Health Bull. 182 (1943)

VI. Nickel Carbonyl and Iron Carbonyl Poisoning

1. General - Acute intoxications, dealt with together because of their clinical similarity, characterized essentially by effects of finely divided and diffuse metallic deposits in pulmonary tissues.
2. Clinical characteristics (duration 8 to 14 days in survivors, mortality varies with severity of exposure, with no permanent disability in survivors)
 - Stage 1 - frontal headache, dizziness, reduced body temperature and exhaustion, after variable latent period.
 - Stage 2 - Cough, dyspnoea, chest constriction, pneumonitis, and pulmonary edema with cyanosis and asphyxial syndrome.
 - Associated inconstant manifestations - delirium, convulsions, cerebral hemorrhage, hepatic involvement
3. References
 - H. J. Armit, J. Hyg., 7:1907; 8:1908
 - Flury and Zernik: Harmful Cases, Berlin, 1931
 - K. Kotzing, Arch. P. Gewerbepath., 4:500, 1933

VII. Thallium Poisoning

1. General - Involves nervous tissues in general, including the autonomic apparatus, with both irritative and degenerative effects. General toxic changes in viscera are relatively prominent following large dosage in short time, the nervous effects being predominant in more prolonged exposure to small doses.
2. Clinical characteristics -
 - Gastro-enteric manifestations - anorexia, nausea, vomiting, gingivitis, stomatitis, and fetid breath
 - Nervous manifestations - irritability, somnolence, exaggerated deep reflexes, generalized tremor, convulsions, pareses and paralysis of muscles of extremities, trunk, eyes, face, throat, and larynx, iritis, retrobulbar neuritis
 - Cutaneous manifestations - keratitis, (corneal) vasomotor changes and alopecia
 - Renal manifestations - albuminuria
3. References
 - James C. Munch et al; J.A.M.A. 101:1315 (1933) and 102:1929 (1934) (numerous other references given in these articles)

Table 2

Mean Concentrations of Certain Metals in Normal Urine

National Groups	Number of Samples	Milligrams of Metal per Liter of Urine				
		Mn	Pb	Sn	Al	Cu
Frenchmen	34		0.030	0.001	0.114	0.036
Mexicans	30	0.012	0.022	0.009	0.054	0.039
N. Americans	30	0.010	0.029	0.018	0.052	0.028
Germans	13		0.027			

The occurrence of these metals in the urine presupposes their carriage in the blood, and for various reasons their concentration in the blood is of considerable practical interest, as is also their distribution in the blood components. Tables 3 and 4 give some of the pertinent facts in this regard.

Table 3

Mean Concentration of Metals in Normal Blood

National Groups	Number of Samples	Milligrams of metal per 100 grams whole blood				
		Mn	Pb	Sn	Al	Cu
Mexicans	30	0.018	0.023	0.010	0.012	0.126
N. Americans	30	0.012	0.027	0.014	0.014	0.103

Table 4

Distribution of Metals in the Components of
the Blood of North American Adults

	Milligrams of Metal in Plasma and Cells of 100 Grams of Blood				
	Mn	Pb	Sn	Al	Cu
Whole Blood	0.012	0.027	0.014	0.014	0.103
Plasma	0.004	0.0015	0.002	0.024	0.043
Cells	0.008	0.024	0.011	0.003	0.059

The limits of safe absorption of the metals

There is no valid reason for the assumption that the absorption of small quantities of the metals to which man has been unavoidably exposed throughout his existence is in any way harmful. On the contrary many of these metals have been found to be necessary in carrying out certain complex physiological processes in various forms of animal life. Therefore, three obvious questions arise in relation to the concentration of these elements in the tissues or excreta of animals and man. (1) What limits of concentration, if any, are required for physiological reasons? (2) What are the upper limits of the concentrations that are compatible with normal healthy existence? (3) What are the ranges of concentration associated with injurious effects? As corollaries of these questions, what are the relationships between rates of absorption, rates of excretion, and the resultant rates of retention in those instances in which excretion fails to keep pace with absorption? Also, in what tissues, if any, do these metals accumulate? In terms of such questions the industrial toxicology of these metals becomes merely a series of problems in mineral metabolism, essentially indistinguishable in principle

3. Collection and Interpretation of Laboratory Data

The usual laboratory tests on the urine, and the common hematological observations require neither elaborate knowledge or training to perform or interpret but these should be attended to with care and precision. Additional laboratory procedures should be selected for performance on the basis of their relevancy. It is here that a background of comprehensive information is required, since the choice of methods must be based on a sound understanding of the genesis and character of the toxic phenomena that are to be identified or measured. Only complete unfamiliarity with the problem at issue will permit the physician to trust the choice and performance of such procedures to ordinary laboratory technicians or even to otherwise well trained technical personnel who lack physiological and medical background. Both the technique and the interpretation of specific procedures require special training and experience, and the interpretation can only be sound if based on comprehensive physiological knowledge. The need for such knowledge will appear as our discussion enters its next phase.

The Significance of Specific Laboratory Procedures in the Recognition of Hazardous Exposure to Metals and in the Diagnosis of Metallic Poisoning

The Behavior of Metallic Compounds in the Human Organism

Normal absorption of metals

The conditions of life on this planet are such that normal individuals, without reference to occupational exposure, absorb into their tissues measurable quantities of metals. Table 1 below gives a few representative data on the distribution and concentration of certain metals in the tissues of persons with no occupational or otherwise unusual exposure to these metals.

Table 1

Concentration Levels of Certain Metals in Normal Human Tissues

<u>Tissues</u>	<u>Milligrams of Metal per 100 Grams of Fresh Tissue</u>			
	<u>Mn</u>	<u>Pb</u>	<u>Cu</u>	<u>Ag</u>
Kidney	0.060	0.027	0.166	0.000
Heart	0.032	0.038	0.190	0.000
Brain	0.030	0.013	0.400	0.003
Liver	0.205	0.130	0.710	0.005
Spleen	0.022	0.030	0.085	0.000
Lung	0.022	0.028	0.110	0.001
Muscle	0.050	0.010	0.125	0.000
Long Bone	0.300	1.880	1.190	0.000
Flat bone	0.170	0.470	0.410	0.010

These metals are taken in food and drink, and to a lesser degree are inhaled in the form of dust that occurs in low concentration in the air. Generally speaking the great proportion of ingested metallic compounds traverse the alimentary tract unabsorbed and appear, little diminished, in the feces. Some of that ingested or inhaled is absorbed, however, this appearing in the tissues and also the urine. The order of magnitude of the urinary excretion is shown by the mean values in Table 2.

from those arising in connection with the more common mineral elements.

Available information does not establish the limits of safe concentration in the tissues, blood, or urine of exposed human beings for the metals under consideration, except in the case of lead. In this case only has the basic information on human metabolism been worked out to such an extent as to justify practical industrial standards based on physiological considerations. It has been shown that when the mean concentration of lead in the urine of representative groups of workmen exposed to lead compounds, does not exceed 8.10 mg. per liter, cases of lead intoxication do not occur. The range of values and their frequencies, that are compatible with safety, are shown in Table 5. Similar data on blood concentrations are available but are not so reliable, for reasons that will appear in later discussion.

Table 5

Lead Concentration in Urine of Persons with Occupational Lead
Exposure Maintained for Years Near Threshold of Toxicity,
but without Occurrence of Illness

<u>Lead in Mg./liter</u>	<u>Frequencies of Occurrence Results Obtained</u> <u>on Single Samples of Urine of Exposed Men</u>
0.000 - 0.019	3
0.020	14
0.040	17
0.060	10
0.080	9
0.100	6
0.120	9
0.140	4
0.160	1
0.200	1
0.220	1
Total	74
Mean and P. E. of Mean Standard Deviation	0.097 - 0.004 0.045

Other pragmatic standards based on the concentrations of fume, dust, vapor or spray in the air breathed by workmen have been set up, however, in lieu of physiological standards. These are given in Table 6 below.

Table 6

Generally Accepted Upper Limit of Safe Concentration of Certain
Metallic Dusts and Fumes in Atmosphere of Workrooms for 8 Hours
per Day over Prolonged Periods

<u>Metal</u>	<u>Concentration in mg. per 10 Cu. M.</u>
Cadmium	1.00
Mercury	1.00
Lead	1.50
Zinc	15.00
Manganese	30.00

(The remainder of the discussion will be presented verbally and can be taken down in notes. It will concern itself with the physiology of lead absorption, retention, and excretion, in illustration of diagnostic, hygienic, therapeutic and physiologic concepts that should be extended by experimental means to include other metals.)

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