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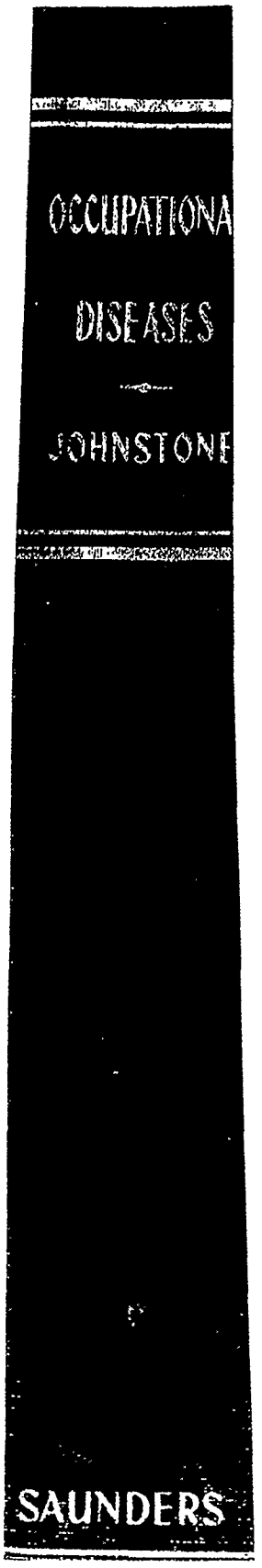

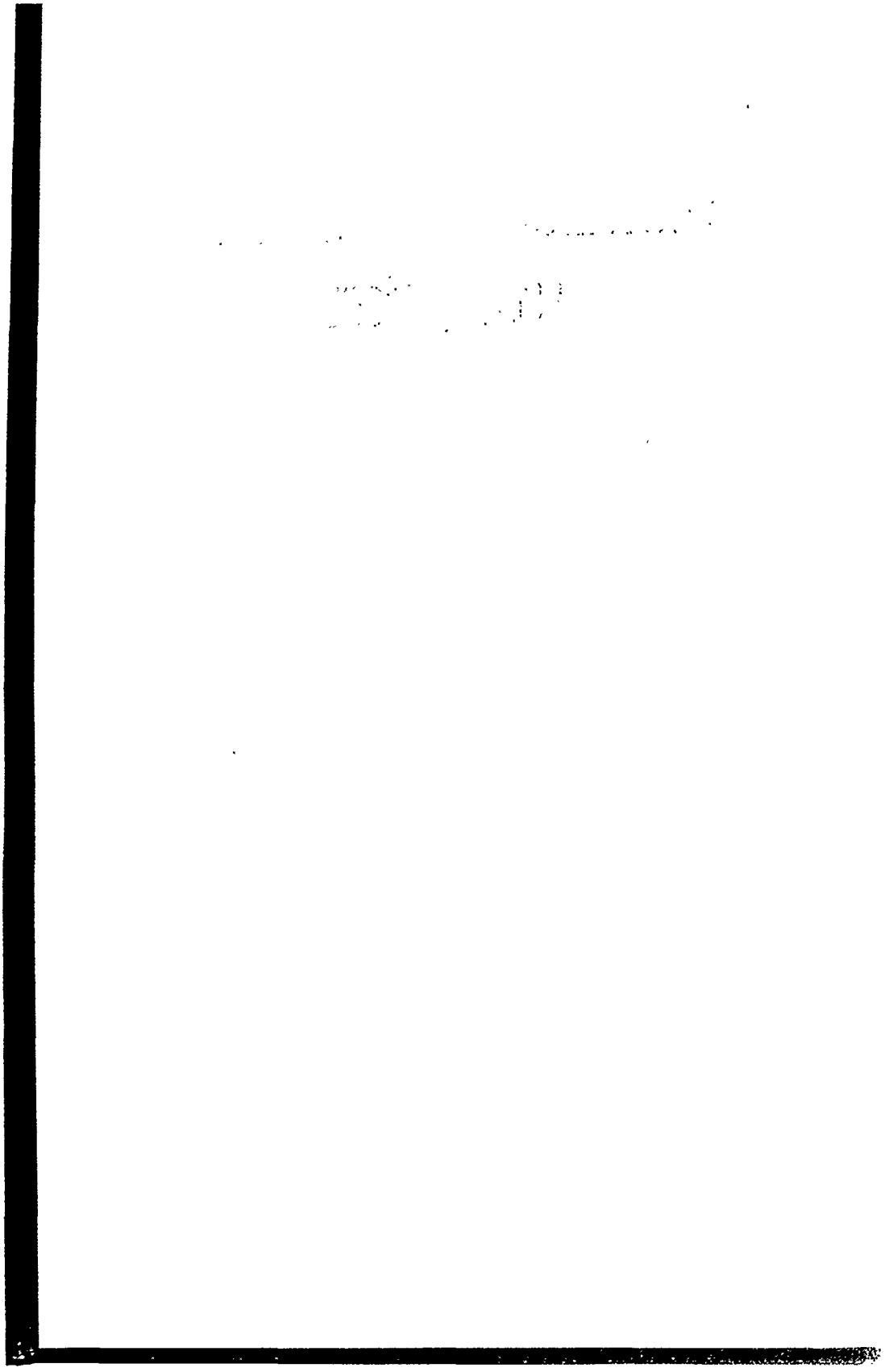

OCCUPATIONAL DISEASES

DIAGNOSIS, MEDICOLEGAL ASPECTS *and* TREATMENT

By

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PRESS OF

To Orpha

WHO WAS NOT JEALOUS OF THE TIME
AND LOVE DEVOTED TO THIS TASK

FOREWORD

THE recognition of diseases associated with certain occupations is almost as old as the beginnings of medicine. It was not, however, until comparatively recent times that any large-scale attempt has been made to prevent and control the conditions which give rise to such health hazards. Now, every industrial country has enacted laws and regulations for the protection of workers, with which employers are required to comply. This has given rise to a large corps of specialists in the fields of law, engineering, medicine, and related subjects. For the doctor who wishes to practice industrial medicine successfully, it is practically obligatory for him to have not only an intimate knowledge of the old, as well as the many new, diseases arising from the manufacture of products, but also extensive legal knowledge that will enable him to testify as an expert and to present evidence in accordance with court procedures.

The objective of medical service in industry is to give prompt emergency relief to the injured as well as to those who become ill while at work. But an even more important goal is to prevent both injury and disease arising from or aggravated by occupation. It is therefore imperative that the doctor should have a good working knowledge of occupational diseases and accident prevention, as well as familiarity with production processes, if he is to render that greater service toward which industry is always striving.

Through research and experience, industry has made important contributions to the promotion of health and in recent years has put into practice many hygienic measures that are far in advance of those required by law. These activities contribute to industrial and social advancement. The industrial physician and the medical departments of industry have come to be recognized as important factors for the attainment of industrial efficiency and social well being.

The changes in this special field are so rapid that books on industrial medicine seldom cover the entire subject. There has been a long-felt need for answers to the questions arising almost daily in the practice of industrial medicine, and this book meets that objective, in the fields of both relief and prevention, and from the standpoint of compensation law and other legal aspects. The author of this volume has collected and brought up to the minute an enormous amount of information that is presented clearly and comprehensively, based largely on his own practice and supported by the experience in this country and abroad.

VICTOR G. HEISER, M.D.

PREFACE

Until recently the physician was concerned primarily with diseases caused by infection, degenerative changes, or metabolic dysfunction. But almost overnight the industrial chemist created an occupational environment which offered a variety of hazards to normal health. To the diseases of commonalty have therefore been added those which find their origin in dusts, fumes, solvents, metals, and noxious gases.

The rapidity with which such occupational hazards have been evolved finds the physician uninformed and the undergraduate untaught. The occasional article in medical periodicals has called attention to some of the existing hazards, but false conceptions have frequently been provoked. Certain occupational hazards have escaped attention; some have been too greatly minimized, while others have been overemphasized. It is obligatory, therefore, that knowledge be disseminated to the profession as to what constitutes a harmful exposure, what exposures are invariably innocent, and what effects are to be expected from exposure to certain substances. In addition, the information to be found regarding some of the older industrial poisonings needs revision, especially as to diagnostic laboratory findings.

This changing aspect of medicine not only has challenged the profession to recognize these diseases of occupation, but has charged it with a medicolegal responsibility. Originally, the Workmen's Compensation Act was designed to compensate for injuries "by accident" and excluded disability not traumatic in origin. For this reason industrial service and treatment were largely surgical in nature, being administered by a comparatively few men who were employed by large plants or industries. But gradually the laws in many states have been amended to include all, or at least a specified number of, diseases of occupation. The indications are that all of the remaining states will soon make similar provision. Following such enactments, the medical responsibility shifts from a few surgeons to *all physicians in every community*. The workman with insidious disease of possible occupational origin will consult his family physician upon whom will be imposed the duty of determining its compensability. The foundation for the equitable fixation of liability, therefore, rests upon diagnosis.

The fundamental aim of this book is to outline a basis for the

diagnosis and *treatment* of the more common occupational diseases, to interpret the *medicolegal* phase, and to offer from experience the *expected disability*. The last two functions approach the first in importance, since the physician is expected to state whether or not the alleged exposure could cause the disability claimed and, if so, how long the disability period will be. The presentation of the problem and the arrangement of the material have been designed to meet the needs of the general practitioner, but the medical student has not been lost sight of in this design. In addition, the book should be of occasional value to the legal profession whenever the fixation of liability in the compensation case offers a problem.

The author offers no apology for the simplicity of this presentation. He is not a scientific investigator or toxicologist, but rather a practitioner in the field of occupational diseases. He has had to work out his own salvation in many instances unaided by books of reference. To him has come the realization that thousands like him need to know more about the common earmarks of these diseases and how to consider their medicolegal aspects. Invariably, the occasional discussion of these conditions is likely to appear in periodicals to which the ordinary physician does not subscribe. If he has access to such a source, the subject is usually discussed in a manner too scientific for practical application. In this connection, many of the illustrations utilized herein present no intricate problem to the experienced physician, but they have been purposely chosen because they represent the common run of cases wherein errors most frequently occur in estimating their status of compensability.

In order that the medicolegal phase of industrial medicine, as it is discussed in subsequent chapters, may be thoroughly understood, the author urges a thorough reading of the first three chapters. In them are discussed certain aspects of industrial medicine not always understood or appreciated by the beginner, or even by men who have long been engaged in this type of practice.

Of those substances which offer a hazard to health, the hydrocarbons, which comprise most of the solvents, have been roughly grouped as solvents rather than into the aliphatic or aromatic series. Those substances which are most prevalent or most injurious to health, such as benzene, lead, carbon monoxide, carbon tetrachloride, or silica, have received comprehensive consideration. *Treatment* is given in detail. It is a lamentable fault of most textbooks to refer to a certain drug or method of treatment without specifying the amount of the drug that should be used, or the procedure to be carried out. For instance, "treat the existing acidosis" is a statement made in most textbooks without detailing the manner of treating it. This text has

not assumed that every reader is acquainted with every phase of treatment or the dosage of every drug prescribed.

The material and experience which form the background for this book have been provided by a clinic which for twenty-five years has been devoted to a surgical and medical industrial service. It had its inception shortly after the adoption of the Workmen's Compensation Act by the state of California—which, it is important to add, was one of the first states not only to adopt this act but to include occupational diseases within the meaning of the act. Thus the tradition of this institution is linked closely with the beginning and progress of workmen's compensation.

The annual admissions to this clinic exceed 11,000 patients, representing multiple and varied occupational diseases and conditions such as are found in the chemical, mechanical, and general manufacturing industries; in mining, agriculture, cattle-raising, and meatpacking; in citrus- and walnut-growing; in fishing and canning; in shipbuilding and airplane construction; and in many other occupations. Of particular value in the author's experience has been the fact that a goodly percentage of these admissions were referred cases from all over Southern California which had been previously diagnosed and their medicolegal status adjudged elsewhere before being studied at this clinic. This referred group has afforded an opportunity to observe a cross-section of the existing viewpoint among industrial physicians regarding the purpose of compensation; to note what phases of industrial medicine were not understood by the profession at large; and to be impressed with the fact that a scientific basis for diagnosis of the more frequent occupational diseases is not adhered to by the majority of physicians. By way of illustration, it is surprising how many practitioners look upon the basophilic stippling as the sole laboratory procedure to be resorted to in cases of alleged lead poisoning. Very few search for the lead in blood or urine; nor can many give the normal figures of such tests, either for lead or for other industrial intoxications. This book was therefore initiated because of the foregoing observation. Owing to this peculiar type of experience, which is not afforded to many, certain exhortations are reiterated many times in the following pages. They are not to be construed as unjust criticism, but as helpful guideposts to those who as yet may be groping their way in this, as yet too-little-understood, field of medicine.

It is impossible to give credit to all those who have played a part in this effort. With humble admiration the author salutes those pioneers in this field whose efforts have advanced industrial medicine and raised it to the level of other branches of medical practice. Unfortunately, a complete individual citation of this group of leaders in

industrial medicine cannot be made, lest in the enumeration a few deserving names be omitted. Whenever the writer's experience has, in his judgment, been inadequate in the presentation of this subject, he has been aided by personal correspondence or by reference to the publications of these investigators, which aid is duly acknowledged.

More intimately, the various members of the staff of this clinic have been most helpful. To Dr. C. E. Early, I feel a deep sense of gratitude difficult to express. As the Director and Chief of Staff of the clinic, he has permitted me a free rein in the management of my department and in the pursuit of this work. His fine sense of justice and his adherence to the highest ethical ideals in their application to industrial medicine have been a source of inspiration to all his associates.

Special credit must go to my associate in this department, Dr. Clifford B. Cherry, for the indispensable aid he gave in preparing the detailed treatment.

In a clinic devoted to industrial medicine, the incidence of skin lesions is exceedingly high. Whenever the diagnosis or occupational relationship is obscure, these cases are referred to our consultant, Dr. Nelson Paul Anderson, who has so kindly contributed the chapter on "The Occupational Dermatoses."

The author feels that the drawings which illustrate certain portions of the text and some of the *x*-ray films, especially those in the chapter on "The Industrial Back," are an important contribution to the book. For these he is sincerely grateful to Florence Sugden Pierce.

I am gratefully indebted also to Margaret Lee, who typed the first draft and checked the bibliography, and to Bertha Taylor Griffin for the many hours she spent in the correction, revision, and editing of this manuscript.

RUTHERFORD T. JOHNSTONE.

LOS ANGELES, CALIFORNIA,
October, 1941.

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OCCUPATIONAL DISEASES

PART I

CHAPTER I

THE PURPOSE OF WORKMEN'S COMPENSATION

THE practice of industrial medicine cannot be carried out with equity to all concerned without an understanding of the fundamental purpose of workmen's compensation. Thirty years have passed since the enactment of this law, which has done more to affect the trend of medicine than possibly any other legislative act. Yet a considerable number of practicing physicians have no knowledge of its meaning or of the manner in which it should function. Time was when such ignorance could be excused, since the administration of the act did not affect the majority of the profession. That day is past. The geographical extension of workmen's compensation, combined with the nationwide expansion of its wording to include the occupational diseases, renders it imperative that all physicians comprehend its purpose, intention, and philosophy. The physician may correctly diagnose a disease independent of his knowledge of the law, but he cannot correctly allocate responsibility if ignorant of this law.

Employer-Employee Relationship.—From the earliest times the relationship between master and servant was unsatisfactory. On farms or estates, or in small-scale industries, the personal relationship between the employer and employee was much closer, and care of an injured workman did occur voluntarily. But with the development of large industries this close interrelationship was lost. Accidents were on the increase. Mangled limbs, crushed skulls, or broken backs were the responsibilities only of the recipients. Recourse to the common law by the injured was usually futile because two defenses, namely, "assumption of risk" and "contributory negligence," were used to defeat the claim.

Employer's Liability Act.—The courts and legislatures sought to meet the condition by enactment of the Employer's Liability Act. This gave rise to the practice of transferring the assumption of risk

on the part of the employer to the insurance carrier, who in turn evaded the law through many loopholes. From the standpoint of the workman, the chief inadequacy of the Employer's Liability Act was the fact that it was necessary for the claimant to prove fault or negligence on the part of the employer. Even when fault was admitted or acknowledge by the court, the compensation return was not well standardized and was usually inadequate. For example, in Allegheny County, Pennsylvania, 235 married men were killed by industrial accidents in one year. In 53 per cent of these industrial fatalities, the widows and children bore the entire income loss; and in only 30 per cent of the cases did they receive over $500, even if one assumes that all unknown amounts were large and all suits pending would be decided in favor of the plaintiff. Of the fatal accidents which occurred in Cuyahoga County, Ohio, over a ten-year period, only 36 per cent were compensated under the existing laws, and the average amount received by the dependents was $838.61.

Abuses of the rights of the workman under the common law or the Employer's Liability Act incited action by the social and legislative forces of that day, resulting in certain recommendations by those who studied the problem.

First Real Compensation Acts.—From this study were evolved the first real workmen's compensation laws adopted by the state of New York in 1910, to be followed the next year by ten more states, until today all but two states have these laws. The cardinal principle of the new act was that liability for an industrial accident must rest upon the employer even without fault.

An exhaustive treatise on the law to explain its countless variations would be of no value to persons other than lawyers. It is not necessary that the physician be informed on all the technicalities of the law, but he should be familiar with that portion of the law which deals with the placement of the liability. Ignorance in this matter is primarily not the physician's fault. His legal course in medical school detailed only information regarding narcotics, illegal abortion, quarantine, and the like. Upon entering an industrial practice, he receives no instructions concerning the meaning of the Workmen's Compensation Act, nor has he ready access to any interpretation. For these reasons the author feels it is appropriate at the beginning of this book to "break down" the main portion of the act so that the reader may gain a working knowledge of its intent.

Purpose of Workmen's Compensation Act.—The Workmen's Compensation Act, or Law, proposes to compensate a workman to a limited extent for an injury, or in certain states a disease, caused by his employment. This benefit is not restricted to those who perform

manual labor, but includes any workman-artist, teacher, musician, actor, *nurse*, *physician*, and the like, who is employed to work for others. The law confers benefits of money and *medical care* for the period of disability, as well as benefits to dependents of the deceased workman when and if his death is due to an industrial exposure. This act is an exercise of the police power of the government designed to protect the workman and to insure a measure of rehabilitation in case of injury. It was not designed to confer damage benefits, or to give the injured *full compensation* for the harm done. Furthermore, and most important of all, the law cannot legally charge the employer *with* every ill to which men are heirs. Before workmen's compensation became effective, the workman *could rarely obtain* relief for a disabling industrial injury. Flagrant abuses by the employer and increasing industrial injuries paved the way for a reform. As a result of agitation by certain forces, this act was adopted as a compromise. To the employer it promised protection against damage suits which might mean financial ruin if in turn he would assume a prescribed limited liability. To the workman it guaranteed certain limited benefits in all cases of injury, even though he could not prove any negligence on the part of the employer.

Administration of the Act.—The administration of the act varies somewhat with the different states, but as a whole there is marked uniformity. The industrial *commissions*, or *boards*, or *bureaus*, as they may be called, have exclusive jurisdiction in all controversies arising under this law. The claimant must obtain his rights through this commission and cannot bring suit in other courts to enforce any right conferred by these statutes, except in certain instances by appeal. Any workman who feels that his case has not been fairly dealt with by the employer or insurance carrier, can file a formal application for hearing before the Industrial Accident Commission.

Liability under the Workmen's Compensation Act is created by a standardized phrase, taken bodily from the English act and incorporated in the majority of the state laws. Condensed it reads, "A case is compensable when it is a personal injury by accident arising out of and in the course of employment." Certain states have altered the wording "arising out of and in the course of"; for instance, Wyoming compensates for injury to employees "as a result of their employment," but the wording used intends that a causal relationship be shown between injury and employment. In order to understand this basic phrase, each part shall be considered.

"*Personal Injury.*"—"Personal injury" means physical harm only. The purpose of compensation is not to reimburse one for a financial loss or wrongs committed against the reputation, social position, or

other sensibilities of the employee. The term has been enlarged in scope in many states to mean that it is not necessary that a blow, fall, or actual violence occur to classify the case as compensable. Through the door of this revised interpretation have entered the occupational diseases in some states. Injury to the body can be sustained from noxious gases, dusts, metals, and the like. Many states having no schedule for occupational diseases have on occasion accepted a case as compensable upon this basis.

"*By Accident.*"—Here again is an attempt to differentiate between a diseased condition and trauma resulting from work. This term places emphasis upon the time interval between the alleged injury and the onset of disability. Obviously a laceration or fracture or severe contusion is apparent to all, and medical aid is immediately sought. In contrast exists the *occupational illness*, which usually develops insidiously and which is not immediately apparent. The effects of an accident may be *far-reaching*, and the ultimate result foreign to the expected one. For example, compensation was granted by the Supreme Court of Pennsylvania in a case in which a workman was thrown violently against an object, fracturing a rib. Pleurisy developed, and in the course of this complication the patient became delirious and committed suicide. It was held that each subsequent event was the result of the original injury. The eventual results from an accident may also be influenced by *unusual factors* requiring an opinion as to whether the end-result is compensable. In Wisconsin a claimant in the course of his employment cut his hand. He was given medical attention, and the wound was practically healed when he engaged in a boxing-match. As a result his injury became infected and ended in the loss of the hand. The Supreme Court sustained the decision of the Industrial Commission that the accident was not the cause of the ultimate disability.

"*Arising Out of and in the Course of Employment.*"—This refers to the *time*, *place*, and *circumstance* under which the accident, or disease, takes place. The first portion of this phrase connotes or implies the danger peculiar to the work, and under this portion of the phrase must be considered most of the occupational diseases. It rules out conditions common to the neighborhood or district, or conditions which may occur at home as well as at work. Employees spend the greater part of each day at their work, and illness of any type may have its onset while they are at their employment. But the fact that it arises while one is at work does not mean that it arises *out* of the work. This fact is too often lost sight of in regard to backache or hernia, especially. This term "arising out of" implies that it must be incidental to the work and not independent of the relation of master

to servant. It need not have been foreseen or expected, but after the event it must appear to have had its origin in the employment. Such an understanding enables one to render a just opinion regarding sunstroke or heat exhaustion or frostbite.

"In the Course of Employment."—An injury can occur in the course of employment only while the workman is engaged in rendering service to his employer under agreement of hire. If, after his day's work is done, an employee of his own volition remains to visit others on another shift and suffers an injury, it is not compensable. Should a truck-driver go out of his regular route for reasons personal to himself and not in the pursuit of his work, he would not be compensated for any injury he sustains while deviating from the work he was hired to do.

Importance of a Detailed History.—*It is apparent from the above* discussion that a detailed history is of fundamental importance in each case. Before treatment, the physician should go carefully into the allegation professed by the patient. Before the physician expends time and material on treatment he should ascertain whether or not the case is apt to be acceptable to the employer or insurance carrier. Horseplay, intoxication, awkwardness, arguments, or fights resulting in injury should all be reported to the carrier for action before proceeding with treatment upon a compensation basis. In rendering emergency treatment the doctor should inform his patient of the basis of his action.

Types of Cases.—Having broadly considered the gist of the law, it is evident that cases for consideration resolve themselves into three main groups:

1. Those obviously compensable
2. Those obviously not compensable
3. Those where compensability is obscure, doubtful, or not readily discernible

It is within this third group that most of the occupational diseases find classification.

Differentiation of Non-occupational and Occupational Diseases.—An analysis of the reports, diagnoses, and testimonies of a large cross-section of physicians relative to their allocation of a case into one of the above three classifications reveals the following facts:

1. Claims for an illness due to occupational environment run the gamut from Bell's palsy to athlete's foot. Any disease man is heir to is apt to be alleged as arising from occupation.
2. Since this is so, there is frequently a failure to discriminate between a non-occupational disease and an occupational one.

3. There exists frequently a failure to differentiate mere exposure from an environment constituting an actual hazard.
4. The more accurate diagnoses come from those physicians engaged entirely in industrial medicine. The inaccurate are more apt to come from physicians engaged part time, or only rarely, in this phase of medicine.
5. A large percentage of the errors in diagnosis can be traced to irresponsibility or disinterest on the part of the physician. He has failed to visualize the philosophy of compensation. Many feel it is easier to accept or deny a case automatically than to ferret out the actual cause of the illness.

Rejection of Claims.—Lest the reader think that the doctrine preached here and elsewhere in these pages indicates that the industrial physician or the insurance doctor has combined with the insurance carrier to defeat the employee in his claim for disability, let him consider the following statistics. The *percentage of rejections* by the various privately owned insurance companies and the self-insured employers is not available, but it is comparable to the figures issued by the State Compensation Insurance Fund of California. During the calendar year 1936, they accepted voluntarily, without any litigation, 96.6 per cent of all claims made upon them, thereby rejecting only 3.4 per cent. In 1937 the rejections were 3.2 per cent; in 1938, 2.8 per cent, and in the first ten months of 1939, 2.5 per cent. It has been pointed out by the Director of the State Department of Industrial Relations of California that certain of the cases that form the percentage of 2.5 per cent actually file for hearing and a certain number of them are awarded compensation, so that the figure for the number of rejections is nearer 1 per cent. Evidence that the Industrial Accident Commission is functioning justly is to be found in the fact that it was upheld in 89 per cent of the cases appealed after its decision.

Certainly these statistics offer proof that, from the standpoint of the workman, the philosophy of compensation as it was originally intended is rigidly adhered to in this state. Deviation from the original purpose should never be indulged in by either the lawmakers or the medical profession. The physician will best maintain this purpose by giving careful consideration to the analysis of the component parts of the basic phrase as they have been interpreted in this chapter.

CHAPTER II

THE ADMINISTRATION OF WORKMEN'S COMPENSATION AND METHODS OF EVALUATING DISABILITY

WORKMEN'S compensation laws were enacted to meet the economic needs of a large body of people. They are effective in each state only to the extent to which each state has endeavored to adopt the principles and enforce the intentions of the act. It is an acknowledged fact that many evils that crept into the administration of the act could have been avoided had the medical profession early entered into a full participation with the lawmakers. Today it is practically obligatory that the general practitioner and certain of the specialists become familiar with the essential features of the legal aspect of workmen's compensation. To this end the following brief discussion is directed. And also, although this book has dealt consistently with the occupational diseases themselves, and has not concerned itself with the traumata which occur in industry, the author appreciates the fact that many of his readers are engaged in a general industrial practice and it was, therefore, thought appropriate to discuss the method of estimating disability and to include tables illustrating the requirements and benefits of compensation as they are in effect throughout the United States. The tables herein quoted are authoritative, since they were obtained from the United States Department of Labor, Bureau of Labor Statistics, Bulletin No. 672, 1940.

Financing Payment of Benefits to Injured Workmen.—Financing the payment of benefits to injured workmen is accomplished through insuring the employer's liability. This may be effected through insurance with private companies, in a state fund, or by "self-insurance." A state fund or state insurance system exists in nineteen states, in eight of which only the state fund operates, while in the other eleven private companies enter into competition. In the majority of states where a competitive basis exists, an employer may become self insured if he satisfies the requirements specified by that state.

Workmen's Compensation Not Compulsory in All States.—It will be noticed from the table on page 8 that while all but one state (Mississippi) have some form of workmen's compensation, it is not compulsory in all of them. There are thirty-two states which have elective compensation, but in twenty-three of these election is presumed

TABLE I.—INSURANCE REQUIREMENTS OF WORKMEN'S COMPENSATION LAWS

State	Compensation Compulsory or Elective	Insurance Required in State Fund: Exclusive or Competitive	Insurance Required in Private Companies or by Self-insurance
Alabama	Elective		
Alaska	Elective		
Arizona	Compulsory	Competitive	Either
Arkansas	Compulsory		Either
California	Compulsory	Competitive	Either
Colorado	Elective	Competitive	Either
Connecticut	Elective		Either
Delaware	Elective		Either
District of Columbia	*Compulsory*		*Either*
Florida	Elective		Either
Georgia	Elective		Either
Hawaii	Compulsory		Either
Idaho	Compulsory	Competitive	Either
Illinois	Compulsory		Either
Indiana	Elective		Either
Iowa	Elective		Either
Kansas	Elective		Either
Kentucky	Elective		Either
Louisiana	Elective		Either
Maine	Elective		Either
Maryland	Compulsory	Competitive	Either
Massachusetts	Elective		Private companies
Michigan	Elective	Competitive	Either
Minnesota	Compulsory		Either
Missouri	Elective		Either
Montana	Compulsory	Competitive	Either
Nebraska	Elective[1]		Either
Nevada	Elective	Exclusive	
New Hampshire	Elective		Self-insurance
New Jersey	Elective		Either
New Mexico	Elective		Either
New York	Compulsory	Competitive	Either
North Carolina	Elective		Either
North Dakota	Compulsory	Exclusive	
Ohio	Compulsory	Exclusive	Either
Oklahoma	Compulsory	Competitive	Either
Oregon	Elective	Exclusive	
Pennsylvania	Elective	Competitive	Either
Philippine Islands	Compulsory		Either
Puerto Rico	Compulsory	Exclusive	
Rhode Island	Elective		Either
South Carolina	Elective		Either
South Dakota	Elective		Either
Tennessee	Elective		Either
Texas	Elective		Private companies
Utah	Compulsory	Competitive	Either
Vermont	Elective		Either
Virginia	Elective		Either
Washington	Compulsory	Exclusive	
West Virginia	Elective	Exclusive	Self-insurance
Wisconsin	Compulsory		Either
Wyoming	Compulsory	Exclusive	
United States Longshoremen's Act	Compulsory		Either
Civil employees	Compulsory		

[1] No security is required. Insurance is optional.

[2] No security is required, but in case beneficiary files notice of death employer may deposit $9000 with clerk of district court or give bond for that amount. In other cases

to obtain in the absence of positive rejection, this presumption affecting both the employer and employee. They are:

Alabama	Iowa	Oregon
Alaska	Kansas	Pennsylvania
Colorado	Louisiana	South Carolina
Connecticut	Missouri	South Dakota
Delaware	Nebraska	Tennessee
Florida	New Jersey	Vermont
Georgia	New Mexico	Virginia
Indiana	North Carolina	

In other elective states the employer must take positive action, and if he acts, the employee's acceptance is presumed, except in Kentucky, where he must sign an acceptance. In Arizona the law is compulsory for the employer, but the employee may elect not to be covered.

States Compensating for Occupational Disease.—While all but one of the states have some type of workmen's compensation, it is to be appreciated that not all of them have coverage for the occupational diseases. Originally in all states, compensation was paid for a disability resulting from injury, injury being construed to mean a single trauma to the body. Gradually the injustice of this was realized by a number of the states, and coverage for all or some of the occupational diseases was included in the meaning of the act. At the present time there exist three groups of states: those which cover all occupational diseases, those which exclude any occupational disease, and the last group which specifies the diseases covered by the act. I have attempted to list these accordingly, but this list cannot be considered accurate, since it was difficult to interpret the meaning in several of the acts. Injury in certain states is considered as resulting from a sudden, violent, or traumatic force and excludes "injury to the tissues" by insidious or slow accumulative effect. Other acts state directly, or infer, that injury is to be construed as any harmful effect upon the body structure, whether the cause is accidental trauma or exposure to harmful substances. The third group of states recognizes only certain specified hazards, and compensates for any disability arising from such. To us of the medical profession it is manifestly unfair that discrimination or total denial should exist in any state. Surely the man who is disabled from constant exposure to lead or

claimant may have writ of attachment issued unless employer files an undertaking in an amount double that sued for.

As to employers.

The state compensation court has ruled that all public employees are subject to the act, without right of election by either the employer or the employee.

By direct appropriation of Congress.

benzol is just as deserving of medical care and compensation as one who lacerates his finger or breaks his leg. There is no question in this writer's mind that this inconsistency in the law will rapidly be changed. Governmental bodies which compensate for *all occupational disease* are:

California	Indiana
District of Columbia	Wisconsin
Illinois	United States Government (for its own employees)

Those which *designate the occupations* or *make allowances* in the interpretation of the act are:

Arkansas	Massachusetts	New Jersey	Pennsylvania
Connecticut	Michigan	New York	Rhode Island
Delaware	Minnesota	North Carolina	Washington
Kentucky	Missouri (?)	North Dakota (?)	West Virginia (silicosis only)
Maryland	Nebraska	Ohio	

Those states which *specifically exclude occupational diseases* are:

Alabama	Kansas	New Mexico	Texas
Arizona	Louisiana	Oklahoma	Utah
Colorado	Maine	Oregon	Vermont
Florida (?)	Montana	South Carolina (?)	Virginia
Georgia	Nevada	South Dakota	Wyoming
Idaho	New Hampshire	Tennessee	

The following tables are self-explanatory and need no comment.

TABLE 2. STATES MAKING NUMERICAL EXEMPTIONS*

Employers Are Exempt Who Have Fewer Than								
2 Employees	4 Employees	5 Employees	6 Employees	10 Employees	11 Employees	15 Employees	16 Employees
Oklahoma	Arizona Florida† Kentucky Ohio Texas Utah Wisconsin	Colorado New Mexico‡ New York§ Puerto Rico Rhode Island	Alaska Arkansas Connecticut Delaware Kansas New Hampshire North Carolina Tennessee	Maine	Georgia	Missouri Vermont Virginia	South Carolina	Alabama

*The following states do not make numerical exemptions: California, District of Columbia, Illinois, Indiana, Iowa, Louisiana, Maryland, Massachusetts, Michigan, Minnesota, Montana, Nebraska, Nevada, New Jersey, North Dakota, Oregon, Pennsylvania, Philippines, South Dakota, Washington, West Virginia, Wyoming, and United States Civil Employees and Longshoremen's Acts. In some of these states coverage is limited to certain enumerated industries.

†Option of Workmen's Compensation Commission, numerical exemption does not apply to *hazardous employments*.

‡Tractor sawmills and other sawmills employing 10 persons are excluded.

§Numerical exemption does not apply to [illegible] at work upon any derrick, building, [illegible] structure 10 stories or more above ground.

¶Contract for building or repairing a railroad. Contractor employs 2 persons or more in the trade.

**Numerical exemption applies only in case of non-hazardous employments. However, in 11 specified industries no exemption exists. Extra-hazardous. Exemption seldom applies.

††Numerical exemption does not apply to employments in mines and in building, repairing or [illegible]

‡‡Numerical exemption applies only to [illegible] non-hazardous employments.

§§Casualty and [illegible] 5 persons. Other [illegible] 25 employees are excluded.

TABLE 3.—WAITING TIME REQUIRED BY EACH STATE

No Waiting Time	1 to 6 Days	7 Days	10 or 14 Days	Compensation Paid for Waiting Period if Disability Lasts Specified Time
Oregon	Alaska[1]	Arizona	Alabama[1,14]	Alabama[4] 4 weeks
	Florida[4]	Arkansas	Colorado[10]	Arizona[2] 2 weeks
	Illinois[6]	California	Iowa[14]	Arkansas[4] 4 weeks
	Maryland[3]	Connecticut		Connecticut[4] 4 weeks
	Missouri[3]	Delaware		Delaware[4] 4 weeks[5]
	Oklahoma[5]	District of Columbia		District of Columbia[7] 7 weeks
	Rhode Island[3,5]	Georgia		Hawaii[7] 7 weeks
	South Carolina[3,5]	Hawaii		Idaho[4] 4 weeks
	Utah[3]	Idaho		Illinois[30] 30 days[8]
	Washington[3]	Indiana		Iowa
	Wisconsin[3]	Kansas		Kentucky[4] 4 weeks
	United States[5] Civil Employees[3]	Kentucky		Louisiana[6] 6 weeks
		Louisiana		Massachusetts[2] 2 wks
		Maine[2]		Michigan[6] 6 weeks
		Massachusetts		Minnesota[4] 4 weeks
		Michigan[9]		Missouri[4] 4 weeks[5]
		Minnesota		Montana[3] 3 weeks
		Montana		Nebraska[6] 6 weeks
		Nebraska		Nevada[1] 1 week
		Nevada		New Hampshire[1] 1 week[5]
		New Hampshire		New Jersey[7] 7 weeks
		New Jersey		New York[5] 5 weeks
		New Mexico		North Carolina[4] 4 weeks[5]
		New York		North Dakota[1] 1 week
		North Carolina		Rhode Island[2] 2 weeks
		North Dakota		South Carolina[2] 2 weeks[5]
		Ohio[11]		South Dakota[6] 6 weeks
		Pennsylvania[12]		Tennessee[6] 6 weeks
		Philippines[5]		Texas[4] 4 weeks
		Puerto Rico[2]		Virginia[6] 6 weeks[5]
		South Dakota		West Virginia[3] 3 weeks
		Tennessee		Wisconsin[10] 10 days
		Texas		Wyoming[15] 15 days
		Vermont		United States[5] Longshoremen[7] 7 weeks
		Virginia		
		West Virginia		
		Wyoming[13]		
		United States Longshoremen		

[1] Applies only to temporary disability.

[2] In case of death, compensation is payable from date of death.

[3] Compensation begins on date of injury in case of permanent partial disability.

[4] If disability period exceeds 4 weeks, waiting period is to be reduced by 4 days, and by 1 additional day for each week the total disability exceeds 4 weeks.

Total disability, but compensation payable from first day of disability in case of partial disability.

[5] Applies only to temporary total incapacity.

If compensation extends beyond such number of weeks after injury, compensation for fifth, sixth, and seventh week is increased by two-thirds.

[6] In case employee has no beneficiary or dependent residing in United States. If there is no such beneficiary or dependent, waiting period is 2 weeks, but if disability continues 6 weeks, compensation payable from date of injury.

[7] Also, no compensation allowed for the first week of total disability, whenever it may occur.

No compensation to injured "work-relief employees" during first 26 weeks of disability, except in cases of permanent injuries in specific schedule or death.

[8] Applies to temporary disability only. No compensation is allowed for first 7 days following date on which employee presents himself to physician for treatment.

TABLE 4.—MINIMUM AND MAXIMUM BENEFITS UNDER WORKMEN'S COMPENSATION LAWS, BY EXTENT OF DISABILITY AND BY STATE, JULY 1, 1940

Death

State	Percentage of Wages	Maximum Period	Limit of Payments Per Week: Minimum	Per Week: Maximum	Total maximum
Alabama	25–65	300 weeks	$5 or actual wage if less	$18.00	
Alaska					$9,000
Arizona	15–66⅔	Widowhood, or specified minority age of children			
Arkansas	15–65	450 weeks	$7	20.00	7,000
California	65		$6.50	25.00	7,575
Colorado	50	312 weeks	$5	14.00	4,375
Connecticut	50	312 weeks	$7	25.00	7,800
Delaware	15–60	285 weeks; thereafter to children till specified age	Weekly wage deemed not under $10	18.00	5,130
District of Columbia	15–66⅔	Widowhood or specified minority age of children	Weekly wage deemed not under $12	25.00	7,500
Florida	35–60	350 weeks	$6 or actual wage, if less	18.00	5,000
Georgia	42½	300 weeks			
Hawaii	25–60	312 weeks; thereafter to children to age 16 (for 104 weeks more, if unmarried and incapable)	Weekly wage deemed not under $5	30.00	5,000
Idaho	10–55	400 weeks (in case of incapacitated, unmarried child, 400 weeks more after reaching 18 years)	$6 or actual wage, if less	12.00	
Illinois		417 weeks	$7.50	20.00	5,500
Indiana	55	300 weeks	$8.80 or actual wage, if less)	16.50	5,000
Iowa	60	300 weeks	$6	15.00	4,500
Kansas			Total minimum not less than $2000	18.00	4,000
Kentucky	65	400 weeks	$5	12.00	4,800
Louisiana	32½–65	300 weeks	$3 or actual wage, if less	20.00	6,000
Maine	66⅔	300 weeks	$6	18.00	5,400
Maryland	66⅔	416 weeks	$8 or actual wage, if less	18.00	7,000
Massachusetts		400 weeks; thereafter to children till specified age; other dependents, 66⅔ percent—maximum period, 500 weeks	$4	40.00	
Michigan	66⅔	300 weeks	$7	18.00	7,400
Minnesota	30–66⅔	300 weeks; thereafter, if dependent wife and children to total of $7500	$8 or actual wage, if less)	20.00	7,500
Missouri	66⅔	692⅓ weeks	$6	20.00	13,845
Montana	30–66⅔	400 weeks	$8	31.00	8,400
Nebraska	66⅔	325 weeks	$6 or actual wage, if less	15.00	4,875
Nevada	10–66⅔	Widowhood, or specified minority age of children		18.46	
New Hampshire					5,400
New Jersey	35–60	300 weeks; thereafter to children till specified age	$10 or actual wage, if less	30.00	
New Mexico	30–60	400 weeks	$5	18.00	7,400
New York	15–66⅔	Widowhood, or specified minority age of children unless blind or crippled		24.98	
North Carolina	60	450 weeks	$7	18.00	6,000
North Dakota	40–66⅔	Widowhood, or specified minority age of children	Weekly wage deemed not under $18	30.00	15,600

Total maximum payments computed by Bureau of Labor Statistics, where not stipulated by law.

In case of uncomplicated silicosis or asbestosis, maximum is $500 if death or disablement occurs in first month of becomes effective and maximum increases $50 every month until limit provided for respondent is reached.

For injuries occurring after July 1, 1939, [illegible] disablement rate, but not the aggregate amount, [illegible] increased 10 per cent.

For widow, plus $2.50 for each child under 18 or over 18 and incapacitated.

150 times average weekly earnings.

TABLE 4 MINIMUM AND MAXIMUM BENEFITS UNDER WORKMEN'S COMPENSATION LAWS, BY EXTENT OF DISABILITY AND BY STATE, JULY 1, 1940 *Continued*

Death Continued

State	Percentage of Wages	Maximum Period	Limit of Payments		
			Per Week		Total maximum
			Minimum	Maximum	
Ohio	66⅔	Period between death and 8 years after date of injury		$18.75	$6,500
Oklahoma					
Oregon		Widowhood, or specified minority age of children		30.00	
Pennsylvania	15 66⅔	300 weeks, thereafter, reduced payments to children to age 16	Weekly wage deemed not under $15	18.00	
Philippine Islands	45 60	208 weeks	Weekly wage deemed not under 4 nor over 30 pesos	18.00	3,000
Puerto Rico					3,000
Rhode Island	50	500 weeks	$8	16.00	8,000
South Carolina	60	450 weeks	$5	25.00	6,000
South Dakota					3,000
Tennessee	50	400 weeks	$5 or actual wage, if less	16.00[a]	5,000
Texas	60	360 weeks	$7	20.00	7,200
Utah	60	Period between death and 6 years after date of injury	Total minimum not under $2000	16.00	7,500
Vermont	15 50	260 weeks	Weekly wage deemed not under $5	13.46	3,500
Virginia	55	330 weeks	$6	16.00	5,000
Washington		Widowhood, or specified minority age of children		55.00	
West Virginia		Widowhood, or specified minority age of children		30.00	
Wisconsin[b]			$10	15.00	
Wyoming			$180 per year to 1 child	50.00	8,500
United States:					
Civil employees	10-66⅔	Widowhood, or specified minority age of children	Monthly pay deemed not under $87.50	26.92	
Longshoremen	15 66⅔	Widowhood, or specified minority age of children	Weekly wage deemed not under $12	25.00	7,500

Permanent Total Disability

California	55 65	550 weeks	$5 or actual wage, if less	$18.50	80 *mm*
Alaska					
Arizona	65	Life			
Arkansas	65	450 weeks	$7	20.00	7,000
California	65	241 weeks; thereafter 40 per cent of wages for life	$6.50	25.00	
Colorado	50	Life	$3	14.00	
Connecticut	50	520 weeks	$7	[c]25.00	13,000

In case of uncomplicated silicosis or tuberculosis, maximum is $500 if death or disablement occurs in first month act becomes effective and maximum increases $50 each month until limits provided for residents are reached.

Per month, for widow, plus $8 for each dependent child.

Pesos.

Plus 10 per cent for each dependent child not to exceed 5 *under 18 years.* Benefits may be extended beyond 400 weeks in meritorious cases.

Per month, for widow with 2 children, plus $5 for each additional child under 16 years.

Amounts are increased or decreased up to 15 per cent for employers or employees violating safety regulations, triple compensation for minors illegally employed.

Compensation equals 4 times average annual wage, payable in installments of 50 per cent of average weekly rate, but total for death, plus disability compensation, may not exceed 70 per cent of weekly wage for from 280 to 1000 weeks, depending on age. Additional compensation allowed to dependent spouse with dependent children, according to age.

Per month, to widow, plus $5 for each child under 16 years.

Per month, for widow or invalid widower (each, $3500), plus $180 per year per child, total $5000 or $5 or actual wage, if less, after 400 weeks.

Plus additional allowance for constant attendance if necessary.

Additional compensation at same rate for maximum of 104 weeks for persons not permanently helpless or *at least* needy or wards for confinement in prison not to exceed 520 weeks.

TABLE 4.—MINIMUM AND MAXIMUM BENEFITS UNDER WORKMEN'S COMPENSATION LAWS, BY EXTENT OF DISABILITY AND BY STATE, JULY 1, 1940.—Continued

Permanent Total Disability—Continued

State	Percentage of Wages	Maximum Period	Limit of Payments: Per Week: Maximum	Limit of Payments: Per Week: Maximum Weekly Benefit	Limit of Payments: Total Maximum Amount
Delaware	50	425 weeks	$15 or actual wage, if less	$15.00	$4,000
District of Columbia	66⅔	Whole period of disability	$18 or actual wage, if less	25.00	7,500
Florida	50–60	550 weeks	$16 or actual wage, if less	18.00	5,000
Georgia	50	450 weeks	$14 or actual wage, if less	20.00	7,000
Hawaii	60	312 weeks	$15	25.00	7,000
Idaho	55–60	400 weeks; thereafter 55 weekly benefit, if dependent children	$16	16.00	
Illinois	50–66⅔	427 weeks	$7.50, $11 if a child under 16, $12 if 2, $13 if 3, and $14 if 4 or more	30.00	
Indiana	55	500 weeks	$9.80 or actual wage, if less	9.50	7,000
Iowa	60	400 weeks	$16 or actual wage, if less	15.00	6,000
Kansas	60	416 weeks	$16	18.00	7,488
Kentucky	65	520 weeks	$15	15.00	7,500
Louisiana	65	400 weeks	$13 or actual wage, if less	20.00	8,000
Maine	66⅔	500 weeks	$16	18.00	6,000
Maryland	66⅔	Whole period of disability	$18 or actual wage, if less	20.00	6,000
Massachusetts	66⅔	500 weeks	$9 or actual wage, if less, but not under $7 for normal weekly hours of 45 or over	18.00	4,500
Michigan	66⅔	500 weeks	$17	8.00	9,000
Minnesota	66⅔	Whole period of disability	$18 or actual wage, if less	20.00	10,000
Missouri	66⅔	300 weeks; thereafter 25 per cent of wages for life	$16	20.00	
Montana	50–66⅔	500 weeks	$15	21.00	10,500
Nebraska	66⅔	300 weeks; thereafter, for life 45 per cent of wages (but not under $4.50 or actual wage, if less and not over $10)	$16 or actual wage, if less	15.00	
Nevada	60	Life	$5.92	4.85	
New Hampshire	50	500 weeks	$8	18.00	5,000
New Jersey	66⅔	400 weeks; thereafter at reduced rate in proportion to earning capacity if submitting to rehabilitation	$10 or actual wage, if less	30.00	
New Mexico	60	550 weeks	$10 or actual wage, if less	18.00	9,900
New York	66⅔	Life	$15 or actual wage, if less	25.00	
North Carolina	60	400 weeks	$7	18.00	6,000
North Dakota	66⅔	Whole period of disability	$6 or actual wage, if less	20.00	15,000
Ohio	66⅔	Life	$8 or actual wage, if less	28.75	
Oklahoma	66⅔	500 weeks	$8 or actual wage, if less	18.00	9,000
Oregon		Whole period of disability	$6.92	8.08	
Pennsylvania	66⅔	500 weeks	$3 or actual wage, if less, but not under $5	18.00	7,500
Philippine Islands	60	208 weeks	4 pesos	18.00	3,000
Puerto Rico	50	340 weeks	$3	9.00	3,000
Rhode Island	50	1,000 weeks	$7	24.00	12,000
South Carolina	60	500 weeks	$5	15.00	6,000
South Dakota	55		$7.50	17.00	7,000

Pesos.

Plus additional allowance for constant attendant if necessary.

In addition to compensation, employee undergoing vocational rehabilitation is paid compensation, maximum $10 weekly.

Award increased 15 per cent for each dependent child.

In case of injuries occurring after July 1, 1939, compensation is increased 50 per cent.

Benefits are paid until amount equals death benefit. Thereafter spouse, if dependent, is paid 25 per cent of total previous payments.

Decrease 50 per cent of wages during first week.

Plus $8 and 5% for each dependent child.

TABLE 4 MINIMUM AND MAXIMUM BENEFITS UNDER WORKMEN'S COMPENSATION LAWS IN EFFECT — DISABILITY CASES — STATE, JULY 1, 1940 — Continued

Permanent Total Disability — Continued

State	Percentage of Wages	Maximum Period	Limit of Payments: Per Week — Maximum	Limit of Payments: Per Week — Minimum	Limit of Payments: Total Maximum
Tennessee	50	550 weeks	$15 or actual wage, if less	$16.00	$5,000
Texas	60	401 weeks	$7	5.00	5,020
Utah	60	Whole period of disability	$7	16.00	
Vermont	50	260 weeks	$7 or actual wage, if less	15.00	4,000
Virginia	55	500 weeks	$6	16.00	6,000
Washington		Whole period of disability	$14.62	60.00	
West Virginia	66⅔	Life	$8	16.00	
Wisconsin	50	Life	$14	21.00	
Wyoming			$50 per month, additional benefit for disfigurement	20.00	6,000
U. S.					
Civil employees	66⅔	Whole period of disability	$14.46 or actual wage, if less	26.92	
Longshoremen	66⅔	Whole period of disability	$8 or actual wage, if less	25.01	7,500

Permanent Partial Disability

Maximum period for specific injuries is given, followed by maximum period of greater for disabilities not listed in schedule of specific injuries there referred to as "non-listed disabilities"[a]

Alabama	55-65	400 weeks	$15 or actual wage, if less[b]	$18.90		
Alaska					$7,500	
Arizona	55	260 weeks, during disability, for non-listed disability				
Arkansas	65	200 weeks, 450 weeks for non-listed disability	$7	20.00	$7,000	
California	65	240 weeks	$6.50	15.00	[c]	
Colorado	50	208 weeks	$7	14.00	4,944	
Connecticut	50	225 weeks; 520 weeks for non-listed disability	$7	[d] 25.00	15,000	
Delaware	50	194 weeks; 485 weeks for non-listed disability	$15 or actual wage, if less	15.00	4,475	
District of Columbia	66⅔	288 weeks; during disability for non-listed disability	$18 or actual wage, if less	25.00	$7,500	
Florida	50-60	200 weeks, 450 weeks for non-listed disability	$16 or actual wage, if less	18.00	[e] 5,000	
Georgia	50	200 weeks, 300 weeks for non-listed disability	$14 or actual wage, if less	20.00	[e] 5,000	
Hawaii	60	312 weeks		12.00	[e] 5,000	
Idaho	55	238 weeks		16.00	[e] 5,808	

[a] Amounts are increased or decreased up to 15 per cent for employers or employees violating safety regulations. Triple compensation for minors illegally employed.

[b] $15 or actual wage, if less, after 400 weeks.

Plus additional allowance for constant attendant if necessary.

Additional compensation for total disability, and 104 weeks for serious and permanent disfigurement of face, neck, head, or hands, but combined compensation not to exceed 520 weeks.

In addition to compensation, employee undergoing vocational rehabilitation is paid cost of maintenance, maximum $10 weekly.

Compensation for 260 weeks, then 35 per cent of average weekly wages for life. In addition, each child increased 5 per cent for each dependent could not exceed 5 under 18 years.

[c] Per month if wife and 2 children, plus $5 for each additional child, plus $25 for constant attendant if necessary.

In addition employee receiving rehabilitation instruction is paid cost of maintenance, maximum $20 weekly, for maximum of 20 weeks.

[d] Per month, plus $150 per year for each child.

Additional benefit for disfigurement.

[e] In addition to compensation for temporary total disability, for non-listed permanent injuries, employee receives $640. Receives an additional amount for disfigurement.

[f] 45 cents per $100 payroll, but for maximum of $6,000 yearly.

TABLE 4.—MINIMUM AND MAXIMUM BENEFITS UNDER WORKMEN'S COMPENSATION LAWS, BY LAW OF DISABILITY AND BY STATE, JULY 1, 1940.—Continued

Permanent Partial Disability—Continued

State	Percentage of Wages	Maximum Period	Limit of Payments Per Week		Total Maximum
			Minimum	Maximum	
Illinois	50–65	125 weeks, 417 weeks for non-listed disability	$7.50, $11 if 1 child under 16, $12 if 2, $13 if 3, and $14 if 4 or more	[1] $20.00	[2] $ 5,500
Indiana	55	250 weeks, 500 weeks for non-listed disability	$8.80 or actual wage, if less	$16.50	$ 5,000
Iowa	60	225 weeks	$6 or actual wage if less	$15.00	$ 4,455
Kansas	60	240 weeks, 417 weeks for non-listed disability		$18.00	[3] $ 4,470
Kentucky	65	200 weeks, 335 weeks for non-listed disability	$5	$12.00	$ 4,000
Louisiana	65	400 weeks	$3 or actual wage, if less	$20.00	$ 8,000
Maine	66⅔	450 weeks, 800 weeks for non-listed disability	$6	$18.00	[4] $ 5,400
Maryland	66⅔	200 weeks	$8 or actual wage if less	$18.00	[5] $ 4,750
Massachusetts		175 weeks		[6] $30.00	$ 4,750
Michigan	66⅔	200 weeks; 500 weeks for non-listed disability	$7	$18.00	[7] $ 9,000
Minnesota	66⅔	450 weeks, including 25 weeks each for healing and rehabilitation.	$8 or actual wage, if less	$20.00	[8] $ 9,000
Missouri	66⅔	232 weeks; 400 weeks for non-listed disability	$6	$20.00	$ 8,000
Montana	50–66⅔	200 weeks, 500 weeks for non-listed disability	$8	$21.00	$ 10,500
Nebraska	66⅔	225 weeks; 300 weeks for non-listed disability	$6 or actual wage, if less	$15.00	[9] $ 4,500
Nevada	50	260 weeks	$6.92	$13.85	[10] $ 3,600
New Hampshire	50	300 weeks	$8	$18.00	$ 5,400
New Jersey	66⅔	230 weeks, cumulative for 2 or more specified injuries, to maximum of 500 weeks	$10 or actual wage, if less	$20.00	[11] $ 10,000
New Mexico	60	180 weeks	$10 or actual wage, if less	$18.00	$ 3,240
New York	66⅔	312 weeks; during disability, for non-listed disability	$8 or actual wage, if less	$25.00	
North Carolina	60	200 weeks; 300 weeks for non-listed disability	$7	$18.00	$ 6,000
North Dakota	66⅔	234 weeks, 450 weeks for non-listed disability	$6	$20.00	$ 9,000
Ohio	66⅔	215 weeks		$18.75	$ 4,000
Oklahoma	66⅔	250 weeks; 300 weeks for non-listed disability	$8 or actual wage, if less	$18.00	$ 4,500
Oregon		208 weeks for married workman	$8.08	$11.54	$ 2,400
Pennsylvania	66⅔	215 weeks; 300 weeks for non-listed disability, weekly maximum $15	$9, or full wage, but not under $5	$18.00	[12] $ 4,500
Philippine Islands	50	208 weeks in addition to compensation for temporary total disability, benefit for continuing treatment			[13] $ 2,000
Puerto Rico	50	300 weeks	$1.50	$4.00	$ 1,200
Rhode Island	50	150 weeks, 500 weeks for non-listed disability, weekly maximum $17	$8	$20.00	[14] $ 7,000

Penalties.

[1] In addition to compensation, employee undergoing vocational rehabilitation is paid cost of such service, maximum $10 weekly.

[2] In case of injuries *occurring after* July 1, 1939, compensation is increased 10 to 50%.

[3] Additional benefit for disfigurement.

[4] In addition to compensation for permanent total disability. Payments are discontinued if employee engages in any gainful occupation.

[5] In addition to compensation for temporary total disability.

[6] In addition to payments for total disability not to exceed 52 weeks. Employee is also entitled to reasonable medical service, not exceeding $150. General disfigurement cases are excepted.

[7] In addition to all other compensation. In cases of specific loss of sight of one eye, or hearing of one ear, or loss of one finger, or toe, or joint thereof, or loss of use of one finger, or toe, or joint thereof, or loss of 1 testicle, $18 weekly, not to exceed $1,500.

TABLE 4 MINIMUM AND MAXIMUM BENEFITS UNDER WORKMEN'S COMPENSATION LAWS, BY EXTENT OF DISABILITY AND BY STATE, JULY 1, 1940 Continued

Permanent Partial Disability Continued

State	Percentage of Wages	Maximum Period	Limit of Payments Per Week		Total Maximum
			Minimum	Maximum	
South Carolina	60	200 weeks, 300 weeks for non-listed disability	$7	$15.00	$6,000
South Dakota	55	200 weeks; 312 weeks for non-listed disability	$7.50 or actual wage, if less	15.00	$4,680
Tennessee	50	400 weeks	$5 or actual wage, if less	10.00	6,400
Texas	60	240 weeks; 300 weeks for non-listed disability	$7	21.00	6,000
Utah	60	240 weeks; 312 weeks for non-listed disability		20.00	6,250
Vermont	50	170 weeks; 260 weeks for non-listed disability	$7 or actual wage, if less	7.00	1,820
Virginia	55	300 weeks; 400 weeks for non-listed disability	$5	20.00	6,000
Washington					[4] 4,000
West Virginia	66⅔[4]	240 weeks; 340 weeks for non-listed disability	$8	16.00	5,440
Wisconsin[20]	70	500 weeks, plus 70 per cent of earnings for healing period. Maximum for non-listed disability, 1000 weeks	$14	21.00	[21] 21,000
Wyoming			$50 per month	21.00	[22] 2,500
U. S.:					
Civil employees	66⅔	Whole period of disability		26.92	
Longshoremen	66⅔	288 weeks; during disability for non-listed disability	$8 or actual wage, if less	25.00	$7,500

Temporary Total Disability

Alabama	55	65	300 weeks	$5 for actual wage, if less	$18.00	
Alaska		65	Whole period of disability			
Arizona		65	433 weeks			
Arkansas		65	450 weeks	$7	20.00	$7,000
California		65	239 weeks	$6.50	27.00	7,975
Colorado		50	Whole period of disability	$5	14.00	
Connecticut		50	520 weeks	$7	25.00	13,000
Delaware		50	475 weeks	$5 or actual wage, if less	15.00	4,000
District of Columbia		66⅔	Whole period of disability	$8 for actual wage, if less	25.00	7,500
Florida	50	60	350 weeks	$6 for actual wage, if less	18.00	5,000
Georgia		50	350 weeks	$4 for actual wage, if less	20.00	5,000
Hawaii		60	312 weeks	$5 for actual wage, if less	25.00	7,000
Idaho	55	60	400 weeks; thereafter $6 weekly $8, if dependent children	$5	16.00	

[1] In case of uncomplicated silicosis or asbestosis maximum is $500 if death or disablement occurs within month act becomes effective and maximum increases $50 each month until limits provided for accidents are reached.

[2] Amounts are increased or decreased up to 15 per cent for employers or employees violating safety regulations. Triple compensation for minors illegally employed.

[3] In addition to compensation, employee undergoing medical rehabilitation is paid cost of treatment to maximum $10 weekly.

[4] Plus 5 per cent for each dependent child.

[5] In addition employee receiving rehabilitation instruction is paid cost of maintenance, maximum $10 weekly, for maximum of 20 weeks.

[6] Additional benefit for disfigurement.

[7] In addition to compensation for temporary total disability. There is an additional amount for loss of hand.

[8] In addition to compensation for amputation of arm or leg.

[9] For non-listed disability 50 per cent of wages.

[10] Plus 5 per cent for each dependent child not to exceed 5 under 18 years.

[11] Where there are 2 or more injuries, amounts received under workmen's compensation in excess of [illegible]

[12] Per month.

Plus $10 for more than one dependent [illegible] United States.

TABLE 4.—MINIMUM AND MAXIMUM BENEFITS UNDER WORKMEN'S COMPENSATION LAWS, AS TO EXTENT OF DISABILITY AND BY STATE, JULY 1, 1940.—Continued

Temporary Total Disability.—Continued

State	Percentage of Wages	Maximum Period	Limit of Payments: Per Week: Minimum	Per Week: Maximum	Total Maximum
Illinois	50	65 Whole period of disability	$7.50 ($11 if 1 child under 16, $12 if 2, $13 if 3, and $14 if 4 or more)	[1] $20.00	$5,500
Indiana		75 500 weeks	$8.80 or actual wage, if less	16.50	5,000
Iowa		60 300 weeks	$6 (or actual wage, if less)	15.00	4,500
Kansas		60 415 weeks	$6	18.00	7,488
Kentucky		65 520 weeks	$5	17.00	7,500
Louisiana		65 300 weeks	$3 or actual wage, if less	20.00	6,000
Maine		66⅔ 500 weeks	$6	18.00	6,000
Maryland		66⅔ 312 weeks	$8 or actual wage, if less	20.00	5,750
Massachusetts		66⅔ 500 weeks	$9 or actual wage if less, but not under $7 for normal weekly hours of 45 or over	18.00	4,500
Michigan		66⅔ 500 weeks	$7	18.00	9,000
Minnesota		66⅔ 300 weeks	$8 (or actual wage, if less)	20.00	6,000
Missouri		66⅔ 400 weeks	$6 (or actual wage, if less)	20.00	8,000
Montana	50	66⅔ 300 weeks	$8	21.00	6,300
Nebraska		66⅔ 300 weeks	$3 (or actual wage, if less)	15.00	4,500
Nevada		60 433 weeks	$6.92	16.62	[2] 7,200
New Hampshire		50 300 weeks	$8	18.00	5,400
New Jersey		66⅔ 300 weeks	$10 (or actual wage, if less)	20.00	6,000
New Mexico		60 550 weeks	$10 (or actual wage, if less)	18.00	9,900
New York		66⅔ Whole period of disability	$8 (or actual wage, if less)	25.00	5,000
North Carolina		60 400 weeks	$7	18.00	6,000
North Dakota		66⅔ Whole period of disability	$6 (or actual wage, if less)	20.00	
Ohio		66⅔ 312 weeks	$8 (or actual wage, if less)	18.75	3,750
Oklahoma		66⅔ 300 weeks	$8 (or actual wage, if less)	18.00	5,400
Oregon	40	66⅔ Whole period of disability	$6.92 (or actual wage, if less)	22.38	
Pennsylvania		66⅔ 500 weeks	$9 (or actual wage, but not under $5)	18.00	7,500
Philippine Islands		60 208 weeks	4 pesos (or actual wage, if less)	[3] 18.00	[4] 3,000
Puerto Rico		50 104 weeks	$1.50	10.00	1,040
Rhode Island		50 1000 weeks	$7	20.00	10,000
South Carolina		60 500 weeks	$5	25.00	6,000
South Dakota		55	$7.50 or actual wage, if less	15.00	3,000
Tennessee		50 300 weeks	$5 (or actual wage, if less	16.00	4,800
Texas		60 401 weeks	$7	20.00	8,020
Utah		60 312 weeks	$7 (or actual wage, if less)	16.00	6,250
Vermont		50 260 weeks	$7 (or actual wage, if less)	15.00	3,900
Virginia		55 500 weeks	$6	18.00	6,000
Washington		Whole period of disability		[5]	
West Virginia		66⅔ 78 weeks	$8	16.00	1,248
Wisconsin		70 Whole period of disability	$7.35	21.00	
Wyoming		Whole period of disability	$50 per month	[6] 90.00	
U. S.:					
Civil employees		66⅔ Whole period of disability	$13.46 (or actual wage, if less)	26.92	
Longshoremen		66⅔ Whole period of disability	$8 (or actual wage, if less)	[7] 25.00	7,500

Pesos.

[1] Amounts are increased or decreased up to 15 per cent for employers or employees violating safety regulations; triple compensation for minors illegally employed.

In addition to compensation, employee undergoing vocational rehabilitation is paid cost of maintenance, maximum $10 weekly.

[2] In case of injuries occurring after July 1, 1939, compensation is increased 10 per cent.

Plus 5 per cent for each dependent child not to exceed 5 under 18 years.

Plus $10 per month for total dependents residing in United States.

[3] Same as for permanent total disability, special schedule of monthly payments for first 6 months of disability, for married or widowed employee.

[4] Per month, additional benefit for disfigurement.

TABLE 3.—NUMBER OF WEEKS FOR WHICH COMPENSATION IS PAYABLE FOR SPECIFIED INJURIES, 50 STATES

State	Arm at shoulder	Hand	Thumb	Index Finger	Middle Finger	Ring Finger	Little Finger	Leg at hip	Foot	Great Toe	Other Toe	Sight of an Eye	Hearing, 1 Ear	Hearing, Both Ears
Alabama[a]	200	150	60	45	30	20	15	175	125	30	10	100		150
Arizona[2]	[b]260 [c]217	[b]217 [c]173	65	39	30	22	17	217	173	30	11	[d]130	87	260
Arkansas[3]	200	150	60	35	30	20	15	175	125	30	10	100	40	150
California[4]	[b]240 [c]220	[b]180 [c]160	[b]40 [c]36	[b]32 [c]28	[b]24 [c]20	[b]24 [c]20	[b]16 [c]12	240	160	32	8	80	40	160
Colorado[5]	208	104	30	26	18	11	13	208	104	26	11	[d]139	35	139
Connecticut[2]	225	175	60	38	30	25	20	208	156	38	13	208	52	156
Delaware[1]	194	148	60	35	30	20	13	194	145	30	10	111		
District of Columbia[2]	280	212	51	28	18	17	7	248	173	26	8	140	52	200
Florida[4]	200	150	60	35	30	20	15	175	125	30	10	100	40	150
Georgia[2]	200	150	30	35	30	20	15	175	125	30	10	100		150
Hawaii[2]	312	244	60	46	30	25	15	288	205	38	16	128	60	312
Idaho[2]	240	200	70	40	40	30	20	180	125	30	12	[d]140	45	150
Illinois[2]	225	170	70	40	35	25	20	190	135	35	12	120	50	125
Indiana[1]	250	200	60	40	35	30	20	200	150	60	[e]	150		200
Iowa[1]	225	150	40	30	25	20	15	200	125	25	15	100	50	150
Kansas[4]	210	150	60	37	30	20	15	200	125	30	10	110	25	100
Kentucky[1]	200	150	[d]60	[d]45	[d]30	[d]20	[d]15	200	125	30	10	100		
Louisiana[1]	200	150	50	30	20	20	20	175	125	20	10	100		
Maine[3]	150	125	50	30	25	18	15	150	125	25	10	100		
Maryland[2]	200	150	50	30	25	20	15	175	150	25	10	100	50	100
Massachusetts[6]	[f]75 [g]50	[f]75 [g]50	[d]40	[d]20	12	12	12	50	50	12	12	50		
Michigan[1]	200	150	60	35	30	20	15	175	125	30	10	100		
Minnesota[2]	200	175	60	45	30	20	15	200	150	30	10	100	52	156
Missouri[1]	[b]232 [c]212	[b]175 [c]160	[d]60 [d]55	[d]45 [d]40	[d]35 [d]30	[d]35 [d]30	[d]22 [d]16	[h]207	150	40	14	[d]118	44	168
Montana[1]	200	150	60	30	30	20	12	200	125	30	12	[d]120	20	120
Nebraska[2]	225	175	60	35	30	20	15	215	150	30	10	125	50	100
Nevada[2]	[b]260 [c]217	[b]217 [c]173	65	39	30	22	17	217	173	30	11	[d]130	87	260
New Hampshire[6]	170	140	40	25	20	15	10	170	120	20	8	100	42	170
New Jersey[2]	230	175	65	40	30	20	15	175	125	30	10	100	40	160
New Mexico[2]	180	110	50	25	20	15	12	180	100	30	12	[d]125	35	137
New York[6]	312	244	75	46	30	25	15	288	205	38	16	160	60	150
North Carolina[1]	200	150	60	35	30	20	15	175	125	30	10	100	70	150
North Dakota[1]	234	195	45	29½	21[i]	15½	13½	234	136½	19½	7½	100	29½	156
Ohio[2]	215	165	[d]60	[d]45	[d]30	[d]20	[d]15	190	140	30	10	125		
Oklahoma[1]	250	200	60	35	30	20	15	175	150	30	10	100		
Oregon[2]	209	165	52	35	20	17	13	191	139	22	9	87	78	208
Pennsylvania[1]	215	175	60	35	30	20	15	215	150			125		150

Payments under this schedule are exclusive of or in lieu of all other payments.

Payments under this schedule are in addition to payments for temporary total disability during the healing period.

[a] For major member.

[b] For minor member.

[c] By enucleation.

[d] Payments under this schedule are exclusive of or in lieu of all other payments. Amounts depend on age and occupation of employee. Figures given are for unskilled workman 39 years old.

[e] Payments under this schedule are in addition to payments for temporary total disability during healing period; 90 per cent of specific schedule to be paid employee. Employer must pay 2 per cent additional to special indemnity fund.

For loss of second toe, 30 weeks; third toe, 20 weeks; fourth toe, 15 weeks; and fifth toe, 10 weeks.

[f] For loss of a metacarpal bone (or corresponding thumb, finger, or fingers), 10 weeks are added.

[g] Payments cover total disability for period specified. Partial disability based upon wage loss may be compensated to end of period given for not over 300 weeks in all.

[h] Payments under this schedule are in addition to all other compensation.

[i] Plus compensation for actual hearing period in an excess of specified number of weeks, except in case of loss of hearing.

[j] In lieu of other payments, unless period of temporary total disability exceeds fixed periods for each class of injury.

[k] Computation based on compensation payable to a married man or a man with dependent child. Payments are in addition to payments for temporary total disability during healing period.

TABLE 5.—NUMBER OF WEEKS FOR WHICH COMPENSATION IS PAYABLE FOR SPECIFIED INJURIES, BY STATES—Continued

State	Loss or Loss of Use of													
	Arm at shoulder joint	Hand	Thumb	Index finger	Middle finger	Ring finger	Little finger	Leg at hip	Foot	Great toe	Other toe	Sight of an eye	Hearing in 1 ear	Hearing, both ears
Philippine Islands	208	160	40	30	25	20	10	190	130	25	10	100	40	208
Puerto Rico	300	[a]200 [a]180	50	45	25	25	15	250	160	25	15	[a]160	25	200
Rhode Island	190	80	50	26	20	20	18	190	80	70	50	80		
South Carolina	200	150	60	35	30	20	15	175	125	40	30	100	50	150
South Dakota	200	150	50	35	30	20	15	160	125	50	[a]40	100		
Tennessee	200	150	60	35	30	20	35	175	125	40	30	100		150
Texas	200	150	60	[a]45	30	21	[a]15	200	125	30	30	100		150
Utah	200	150	60	30	30	20	12	180	125	30	32	[a]120		
Vermont	170	140	40	25	20	15	10	170	120	30	8	100	42	170
Virginia	200	150	60	35	30	20	15	175	125	30	30	100	50	
West Virginia	240	200	80	40	28	20	20	240	140	40	16	112		
Wisconsin[b]	500	185[c]	125	50	40	30	30	500	250	85[c]	[a]25	[a]275	50	225
United States: Longshoremen[d]	280	212	51	28	18	17	7	248	173	26	8	110	52	200

[a] For additional loss of 1 or more toes other than the great toe, an additional period of 10 weeks.
[b] In lieu of all other benefits except medical and hospital.
[c] For loss of second toe, 30 weeks.

TABLE 6—STATES LIMITING THE PERIOD AND AMOUNT OF MEDICAL BENEFITS

Neither Time nor Amount Limited	No Limitation on Amount	No Limitation on Time	Both Amount and Time Limited
Arizona	Alaska	Florida	Alabama
California	Arkansas	Iowa	Colorado
Connecticut	Indiana	Louisiana	Delaware[a]
District of Columbia	Massachusetts	Maryland	Georgia[a]
Hawaii	Michigan	New Jersey	Kansas[a]
Idaho	Nevada	New Mexico	Kentucky[a]
Illinois	New Hampshire	Ohio	Maine[a]
Minnesota	North Carolina[a]	Oregon	Missouri[a]
Nebraska	Oklahoma	Rhode Island	Montana
New York[b]	South Carolina	Utah	Pennsylvania
North Dakota	Texas	West Virginia	South Dakota
Philippines	Virginia	Wyoming	Tennessee
Puerto Rico			Vermont
Washington[c]			
Wisconsin[a]			
United States:			
Civil employees			
Longshoremen			

[a] Additional services in special cases or in discretion of commission.
[b] Except in case of treatment for silicosis or asbestosis.
[c] In case of disability resulting from inhalation of harmful dust, period of treatment is limited.
[d] Virtually unlimited under administrative practice.

TABLE 7—MAXIMUM PERIODS AND AMOUNTS OF MEDICAL SERVICE, BY STATES

State	Maximum Period	Maximum Amount	State	Maximum Period	Maximum Amount
Alabama	90 days[1]	$200.	New Hampshire	30 days	Unlimited.
Alaska	1 year	Unlimited.[2]	New Jersey	Unlimited	$100.[1]
Arizona	Unlimited	Unlimited.[2]	New Mexico	Unlimited	$400.
Arkansas[3]	60 days[4]	Unlimited.	New York[7]	Unlimited	Unlimited.
California	Unlimited	Unlimited.	North Carolina[8]	10 weeks[9]	Unlimited.
Colorado	4 months[4]	$500.	North Dakota	Unlimited	Unlimited.
Connecticut	Unlimited	Unlimited.	Ohio	Unlimited	$200.[1]
Delaware	30 days[1]	$150.[1]	Oklahoma	60 days[1]	Unlimited.
District of Columbia	Unlimited	Unlimited.	Oregon	Unlimited	$250.[1]
Florida	Unlimited	$250.[5]	Pennsylvania	60 days	$150.[1]
Georgia	10 weeks[1]	$500.	Philippines	Unlimited	Unlimited.
Hawaii	Unlimited	Unlimited.	Puerto Rico	Unlimited	Unlimited.
Idaho[6]	Unlimited	Unlimited.	Rhode Island	Unlimited	$200.[10]
Illinois[7]	Unlimited	Unlimited.	South Carolina	10 weeks[1]	Unlimited.
Indiana[8]	90 days[1]	Unlimited.	South Dakota	20 weeks	$300.
Iowa	Unlimited	$600.	Tennessee	30 days[1]	$200.
Kansas	60 days	$100.[1]	Texas	4 weeks[11]	Unlimited.
Kentucky	90 days[1]	$200.[1]	Utah	Unlimited	$500.[1]
Louisiana	Unlimited	$250.	Vermont	2 weeks	$50.[11]
Maine	30 days[1]	$100.[1]	Virginia	60 days[12]	Unlimited.
Maryland	Unlimited	$500.	Washington	[13]	Unlimited.
Massachusetts	2 weeks[1]	Unlimited.	West Virginia[8]	Unlimited	$800.
Michigan	90 days	Unlimited.	Wisconsin	Unlimited	Unlimited.
Minnesota	Unlimited	Unlimited.	Wyoming	Unlimited	$150.[14]
Missouri	90 days[3]	$750.	United States:		
Montana	6 months	$500.[6]	Civil employees	Unlimited	Unlimited.
Nebraska	Unlimited	Unlimited.	Longshoremen	Unlimited	Unlimited.
Nevada	6 months[3]	Unlimited.[2]			

[1] Additional service may be given at option of employer.
[2] Employees contribute.
[3] Additional service in special cases or at discretion of commission.
[4] Additional service may be authorized, not to exceed 91 days.
[5] In case of hernia, if employee requires operation he is entitled to service without limitation of time.
[6] In surgical cases maximum may be increased by order of commission to $500.
[7] A special operating fee of $100 allowed in case of hernia.
[8] In case of disability resulting from inhalation of harmful dust, period of treatment is limited to 90 days, but may be extended for an additional 360 days by the Industrial Board.
[9] Special limitations upon medical benefits in case of certain occupational diseases.
[10] Exclusive of hospital treatment.
[11] In case of employee receiving hospital treatment for more than 14 days, the maximum is $250.
[12] Also hospital first 30 days, maximum $150.
[13] Extended in unusual cases. Not to exceed 180 days.
[14] In case of temporary disability, continues not longer than period of compensation, and in case of permanent disability not beyond the date of award. Employees contribute.
[15] Additional expenditure of not more than $200 for medical service and $300 for hospital treatment may be authorized by court.

DIGEST OF WORKMEN'S COMPENSATION ACT (CALIFORNIA)

The following is a digest of the Workmen's Compensation Act as it is administered in California. Naturally, the act in certain of the other states will vary from the California law, but this digest will

serve to present a survey of the basic phases of workmen's compensation in general :

Excluded Employments.—The law does not give compensation to employees engaged in household domestic service (household domestic servants who work for one employer for fifty-two or more hours per week are not excluded from the provisions of the workmen's compensation laws and the employer of such household domestic servants must carry workmen's compensation insurance).

The law does not give compensation to employees engaged in labor that is both casual and not in the course of the trade, business, profession, or occupation of the employer unless the employer carries compensation insurance or has filed an acceptance of the law with the commission, nor to employees engaged in farm or agricultural labor of any description, if the employer has rejected the compensation act and posted notices of rejection on his premises; or if the employer's payroll does not exceed $500 in the year preceding the injury. Employees working on board vessels in navigable waters and railroad employees engaged in interstate commerce, at the time of the injury, are also excluded as there are federal laws governing such employments and the state law cannot be made applicable.

Questions as to whether employment is excluded should be submitted to the commission for advice.

Employer's Security.—Every employer, except the state and all of its political subdivisions or institutions, who has one or more persons employed, even part time, in occupations not specifically excluded, is required to secure the payment of compensation in either of the following ways:

1. By insuring and keeping insured for full coverage with an authorized compensation insurance carrier
2. By obtaining a certificate of consent to self-insure from the Industrial Accident Commission

To obtain the commission's certificate of consent to self-insure, the employer will be required to furnish security in the form of a surety company bond or approved collaterals that are legal investments for savings banks in this state. The minimum amount of security required in this respect is now $30,000 but is always subject to regulation by the members of the commission.

If the employer complies with this provision, all of his liability to his injured employee or the dependents of the deceased employee is fixed by and limited to the provisions of this law.

If the employer does not secure the payment of compensation in either of the two ways mentioned, he is not only liable for compen-

sation but subject to a suit for damages in the superior courts. In case of the wilful failure by an employer to secure the payment of compensation, the amount of compensation otherwise recoverable shall be increased 10 per cent, such increase in no event to exceed $1000.

The law makes it a misdemeanor to fail to secure the payment of compensation, and the penalty for this misdemeanor is a fine of not more than $500, or imprisonment for not more than six months, or by both fine and imprisonment.

If an employer is continuously uninsured for thirty days or more, the tribunal before whom his case comes must at least fine such employer $300. The commission also has the power through court proceedings to abate or close up any business in which the employer fails or refuses to carry workmen's-compensation insurance on his employees.

Every employer subject to compensation laws and not self-insured must keep posted at his headquarters, or at one or more of his places of employment, a notice that shall state the name of the employer's current compensation-insurance carrier. It is a misdemeanor not to keep such notice posted.

Information concerning the failure of any employer of labor, other than those excluded, to secure the payment of compensation in either of the two ways mentioned above or information concerning the failure to post such notices of current insurance carriers should immediately be forwarded to the Industrial Accident Commission.

Liability of Employer.—When an employee sustains an injury "arising out of and in the course of the employment" (and the employment is not one of the excluded classes), the law requires the employer to furnish the injured with certain measures of relief.

The term "injury," as used in this law, includes any injury or disease arising out of the employment, including injuries to artificial members. The obligation to furnish the measures of relief becomes the liability of the employer immediately after knowledge or notice, and must be furnished or tendered without demand.

Disability indemnity is paid by the employer or his insurance carrier. The Industrial Accident Commission does not pay such indemnity. Such insurance carrier may be the State Compensation Insurance Fund or any private insurance carrier authorized to write workmen's compensation insurance.

Insured Employers.—If the employer carries compensation insurance, the insurance carrier assumes the obligations of the employer, but the employer must understand that his contract (policy) of insurance requires him to notify the insurance carrier promptly

of any injury to his employee. The necessity for such notice cannot be too strongly urged. Serious results sometimes grow out of apparently slight injuries, and the employer may become involved for failure to comply with his contract. Insurance carriers furnish forms for report and will inform where injured are to be sent for medical and surgical treatment.

An employer in addition to posting and keeping posted the notice of his current compensation-insurance carrier should immediately notify any injured employee of the name and address of such insurance carrier.

Notice to Employer.—An injured employee should promptly notify the employer, his employer's superintendent, foreman, or other person in authority, of the fact of injury if the employer or those mentioned do not have actual knowledge of such injury, in order that the employer may have opportunity to furnish the measures of relief. Failure to give such notice may prejudice and defeat a claim for compensation.

Compensation.—The measure of relief which the law provides is termed "compensation." This term, therefore, includes both treatment and indemnity payment, and is to be furnished to the following extent:

1. All the medical, surgical, and hospital treatment, including medicines, medical and surgical supplies, crutches and apparatus, including artificial members, that may be reasonably required to cure and relieve from the effects of the injury.

If the employer or insurance carrier does not furnish such treatment after due notice or knowledge of injury, and within a reasonable time, the injured employee has a right to obtain it elsewhere and the employer or insurance carrier will be held liable for the reasonable cost.

If treatment is obtained elsewhere, the injured must select a practitioner who is licensed to practice in medicine or surgery in order to recover the reasonable cost of treatment.

If the injured prefers to select his own doctor and treatment he may do so, but it will then be at his own cost unless the employer or insurance carrier consents and agrees to pay for such service.

Where treatment is not furnished by the employer or insurance carrier, the injured must nevertheless submit to examination by a physician or surgeon selected by the employer or insurance carrier when so requested. Failure or refusal to submit to such examination will prejudice the right of the injured to disability indemnity.

2. If the injury causes disability of more than seven days, exclusive of the day of injury, then the employer or insurance carrier shall thereafter pay the injured 65 per cent of his loss of *average* weekly wages during the remaining period of disability.

No indemnity is payable for the first seven days of disability, and disability commences the day after the injured quits work as a result of the injury. This indemnity is payable on the employer's regular payday, but not less frequently than twice in each calendar month.

The disability indemnity is not 65 per cent of the *actual* wage. The law provides that the disability indemnity shall be 65 per cent of the *average* wage, and that the *average is* 95 per cent of the *actual*.

Wages in excess of $40.48 per week, or $175.43 per month, are not taken into account, as the law provides that the *average* weekly wage shall not be taken at more than $38.46, nor less than $10. For these reasons the weekly indemnity for temporary total disability is never more than $25, nor less than $6.50 a week. In arriving at the actual earnings, overtime is included, and also the market value of board, lodging, or other advantages if they are furnished as a part of the remuneration. To figure the disability indemnity correctly, where the earnings are between the minimum and maximum, multiply the daily wage by the number of days employed per week; add the overtime and value of other considerations, if any; deduct 5 per cent from this sum, and multiply the remainder by 65 per cent.

3. If the injury causes the loss of any member, or impairs the normal use of any member of the body, such as amputation of bone, permanent stiffness of joints, loss of sight, hearing, etc., the result is what is termed a "permanent partial disability."

Where such injury occurs, or results, the commission should be applied to for a rating when the medical and surgical treatment has been completed or the condition no longer yields to treatment. For each 1 per cent of loss that the rating tables adopted by the commission give for such injury, the injured will be entitled to four weeks of disability indemnity. The indemnity for permanent partial disability is not in addition to what may have been paid during the period of treatment and recovery. The rating will show the total number of weekly payments to be made, beginning the eighth day after the injury, and any payments made during the period of treatment and recovery are a credit on the total to be paid under the

rating. The remaining payments, if any, are to be paid as they become due and without regard to when the injured returns to work or what his earnings may be after he does return. If the disability is rated at 70 per cent or over, the injured will be entitled to the weekly indemnity of 65 per cent of his average weekly wages for 240 weeks, and thereafter a weekly life pension of 1 per cent of those wages for every 1 per cent of disability in excess of 60 per cent.

4. If the injury causes death, the employer or insurance carrier is obligated to pay burial expense to the extent of $150, and a death benefit to those who were dependent upon the deceased for support at the time of the injury.

A person or persons totally dependent will be entitled to a death benefit of three and one-half times the average annual earnings of the deceased but not to exceed $6000, and not to be less than $2000.

A person partially dependent will be entitled to a death benefit of three and one-half times the annual amount devoted by the deceased to the support of such person, this amount not to be more than three and one-half times the average annual earnings of the deceased nor to exceed $6000.

Death benefits are payable in installments of 65 per cent of the average weekly wage of the deceased until the total death benefit has been thus paid.

The commission will determine who are dependents, and the extent of their dependency, upon the request of any party in interest.

Penalties and Forfeitures.—The law provides that if the injury is caused by the serious and wilful misconduct of the employer, indemnity is increased one-half.

The employer cannot carry insurance against this penalty, which is chargeable to, and payable by, the employer only.

The law further provides that if the injury is caused by the serious and wilful misconduct of the employee, indemnity is reduced one-half.

The last-mentioned provision does not apply when the injury causes death, or results in a permanent disability rated at 70 per cent or over, or where the employee is under sixteen years of age.

The fact of wilful misconduct must be established and so held by the commission in formal proceedings before indemnity can be increased or reduced.

No compensation is recoverable where injury is caused by the intoxication of the employee or where the injury is intentionally self inflicted.

Limit of Time to Enforce Compensation.—Proceedings to compel the payment of any of the benefits provided by this law are brought before this commission, but the legal right to maintain such proceedings is barred after a certain length of time.

Where no medical treatment has been furnished or disability payment made, proceedings must be commenced within six months from the date of injury.

Any payment of compensation or agreement therefor, or furnishing of medical treatment, will extend the time six months from the date of the agreement or the last payment or last medical treatment.

Proceedings for the collection of the death benefit must be commenced within one year from the date of death, subject to certain restrictions.

These limits of time do not begin to run against an employee or dependent under the age of twenty-one until he has reached that age, or an incompetent person, unless a legal guardian has been appointed for such person.

The commission has 245 weeks' continuing jurisdiction over its formal orders and awards, and any amendment must be made within that time.

Application for Adjustment of Claim.—Proceedings for the adjustment of any claim are instituted when an application for such adjustment is filed with the Industrial Accident Commission.

Blank forms of application will be furnished on request or will be prepared for signature at either office of the commission.

It should be distinctly understood that no report of injury, correspondence, or application for permanent disability rating will be considered the commencement of proceedings. A formal application is headed in bold type *Application for Adjustment of Claim.*

When an application for adjustment of claim is filed, due notice will be given interested parties of the time and place of hearing. A referee will conduct the hearing, and opportunity will be afforded parties to present competent evidence in support and defense of claims, and all parties should be prepared to present their evidence at such time.

Agents or Attorneys.—If claimants desire to be represented by an agent or attorney, the commission will fix the fee to be paid the agent or attorney out of any award, and no agreement to pay a greater sum is valid or binding upon claimants. Indemnity cannot be assigned before payment, and it is only subject to liens fixed by the commission.

Miscellaneous Provisions.—The law provides that the term *casual*, as applied to excluded employments, shall be taken to refer

only to employment where the work contemplated is to be completed in not exceeding ten working days, without regard to the number of persons employed, and where the total labor cost is less than one hundred dollars.

An injured employee is entitled to one change of physicians. When so requested, the employer or insurance carrier shall nominate three practicing physicians competent to treat the particular case, from whom the employee may select.

Medical and surgical treatment includes nursing, medicines, medical and surgical supplies, crutches and apparatus, and artificial members.

A permanent disability rating is not the commission's award. The issuance of a rating does not protect the injured against the limit of time to enforce payment.

ESTIMATION OF DISABILITY

Trial by Jury versus Expert Opinion.—Estimation of the extent of disability and the amount of the award is made in most states by the industrial commission, which is a fact-finding body. In a few states, however, *trial by jury* is the method used to determine the extent of injury, and appeal is permitted as in other civil contentions. This latter system is obviously unfair. Juries are too often influenced by sentiment, emotional suggestions, and a desire to see the claimant secure personal retaliation. These conditions have no place in the just administration of compensation. Much better is the system which entails *two separate opinions;* one is from a licensed medical doctor, who indicates the extent of physical disability or functional loss; and the other is from the lawmaking body, which bases its award upon a rating schedule as adopted by the state. Most states have fashioned their schedules upon a plan of flat rating of disability benefits. In California the benefits paid for permanent disability vary with age and occupation. The philosophy of this variation will be explained subsequently.

Estimating Temporary Disability.—The term "temporary disability" implies that the condition is one not apt to be permanent. In the legal sense it is the healing period, but from the medical viewpoint it is the prognosis. On first thought, estimating temporary disability should not be difficult. In the ordinary practice of medicine it is not, for recovery is estimated purely from the scientific factors entailed in the illness or injury. But in industrial cases recovery is often contingent upon other factors. The workman may have been fired; or the job may have been about finished at the time of injury; or he may be aware of a strike's being imminent; or he may be overly cautious about returning to the same hazard. Phy-

sicians often prolong temporary disability or create a claim for permanent disability by tactless statements or erroneous diagnoses. A salesman, following an automobile accident, was hospitalized in a nearby town, an upper body cast was applied, and he was informed that he had a fracture of a thoracic vertebra. He developed marked tremor of the head and right arm, which persisted until a compromise and release was effected after the payment of considerable money. Actually, he had a failure of fusion of a thoracic vertebra, but no pathological injury (Fig. 91).

Estimating Permanent Disability.—Estimating permanent disability is a complex procedure, and as yet the method is devoid of uniformity. It most frequently involves the loss or use of a member such as a finger, hand, arm, or leg. Variations in the amount of disability as expressed by several examining physicians will often exceed from 25 per cent to 40 per cent. No two physicians employ the same type of implement for measuring motion, or include identical factors in their estimation of the patient's chance of rehabilitation.

McBride,[2] who has given considerable thought to this problem, includes functional, vocational, and cosmetic loss, earning-capacity, and adverse influence in securing employment.

Scudder's Method.—Scudder[3] does not rate his fracture cases until the end of one year, and employs the following method:

The use of the letters A.F.E. signifies { Anatomic result / Functional result / Economic result }

The use of the figures 1, 2, 3, 4 ($A^3F^4E^1$) after each letter gives the percentage value to the final estimation, allowing a range of 25 per cent for each figure.

1 = 25 per cent 2 = 50 per cent 3 = 75 per cent 4 = 100 per cent

Accurate Rating.—There are four factors which make up the total of an *anatomic* result:

1. Length
2. Alignment (total) by inspection and measurement
3. Apposition: judged by roentgenograms taken in two planes
4. Angulation

Suppose a patient has normal length	100 per cent
Suppose a patient has poor alignment	75 per cent
Suppose a patient has half apposition	50 per cent
Suppose a patient has 10° to 15° angulation .	50 per cent
	4)275
	68 ¾ per cent

This patient would receive A^{-} because 68— per cent is nearer 75 per cent in the gross rating.

There are four factors which make up the total of a *functional* result:

1. Total functional result, subjective (asking the patient)
2. Total functional result, objective (by observation) muscle strength and staying power
3. Joint movement above the fracture, as compared to the other side
4. Joint movement below the fracture, as compared to the other side

Suppose the patient says he is as good as before	100 per cent
Suppose on observation, he has less power and movement on fractured side; can move the part (walk, or use his arms) about $\frac{1}{2}$ of that which he did before	75 per cent
Suppose his joint above has limited motion, estimated at $\frac{1}{3}$ off normal	66 per cent
Suppose his joint below is normal	100 per cent
	4) 341
	85 per cent

This patient would be rated as F^{3}.

There are four factors which make up the total of an *economic* result:

1. Same work as before, lighter, or heavier work
2. Same pay as before, more, or less
3. Same hours of work, more, or less
4. Same volume of work, more, or less

Suppose a patient has a lighter job	50 per cent
Suppose a patient gets $\frac{3}{4}$ former pay	75 per cent
Suppose a patient works half as long each day	50 per cent
Suppose a patient turns out $\frac{1}{3}$ the volume of work	33 per cent
	4) 208
	52 per cent

This patient would receive E^{2}.

Added up, that patient would receive $A^{3}F^{3}E^{2}$.

McBride's Method.—McBride believes that it is not enough to measure extension, flexion, pronation, supination, rotation, and the like. He considers also the ability of the injured one to perform the acts of pointing, reaching, pinching, grasping, holding, pushing, pull-

ing, catching, throwing, pressing, kicking, turning, and lifting. These functions, in addition to the estimated anatomical and physiological limitations, should permit the trained physician to arrive at a fair computation of percentage of disability. The elements of motion as listed above are considered under the functional factors of:

1. Quickness of action
2. Coordination of movements
3. Strength
4. Security
5. Endurance
6. Safety as a workman
7. Prestige of normal physique

McBride gives the relative value of each of the functional factors in respect to 100 per cent incapacity in a general way as follows:

1. Delayed action	10 per cent
2. Awkwardness	20 per cent
3. Weakness	20 per cent
4. Insecurity	10 per cent
5. Diminished endurance	20 per cent
6. Lowered safety factor	10 per cent
7. Adverse influence of conspicuous impairment	10 per cent

The total of these respective percentages of loss incurred will represent the partial loss of the part as a whole, and is to be estimated as a percentage of the loss in respect to physiological and anatomical limitations.

Permanent Disability in California

In California, when the physician determines his patient is ready for a permanent rating, a standard form, furnished by the Industrial Accident Commission, is filled out and mailed directly to that body (Form 9). Upon the information furnished, the rating committee of the commission bases its award. It is apparent that this form does not furnish as complete a basis for determination of disability as that suggested by McBride. It has the advantage, however, of securing more standardized information from a large group of physicians, many of whom are ill-trained in this phase of medicine, than the method advised by McBride. Possibly our medical schools of the future will graduate men trained in a uniform method of estimating disability.

For a broader and more general consideration of the method of estimating permanent disability, I am indebted to Douglass V.

Form No. 7

NOTICE—This original report must be sent by the examining surgeon direct to the Industrial Accident Commission of the State of California and not to any other party.

DO NOT write in here

NO.

STATE OF CALIFORNIA
DEPARTMENT OF INDUSTRIAL RELATIONS

INDUSTRIAL ACCIDENT COMMISSION

PERMANENT DISABILITY RATING DEPARTMENT

STATE BUILDING, CIVIC CENTER, SAN FRANCISCO
602 STATE BUILDING, CIVIC CENTER, LOS ANGELES

SURGEON'S SPECIAL REPORT—UPPER EXTREMITIES

This Blank to Be Filed Only When Maximum Restoration of Function Has Been Attained

Name of patient — Address.
Age — Occupation — Name of employer
Date of accident — Insurance company

1. Give description of injury, stating the condition of the patient when first called to your attention.

2. Give description of treatment employed in this case in full, stating what operations have been performed and with what results.

3. Name and address of surgeon who operated and assistant.
Surgeon
Assistant

4. Has repair been delayed from any cause? If so, what?

5. Was there any previous permanent disability or deformity? If so, what?

6. Is there evidence of luetic, gonorrheal or tubercular infection or alcoholism? If so, what?

7. Temperament of patient, any evidence of hysteria, neurasthenia, or hypochondria? If so, what?

8. Date of last treatment — Is present condition permanent?

9. What is the PERMANENT DISABILITY in this case, if any? Discuss fully from the following standpoints:

a. AMPUTATION

b. DEFORMITY.

c. ANKYLOSIS or LIMITED MOTION

d. ANESTHESIA or PARESTHESIA

e. LOSS OF FUNCTION

NOTE.—Record as far as possible the loss of function in degrees and as the term of a fraction in comparison with the corresponding uninjured member. Example: Injury to finger, index, major, proximal joint. Motion is found to be 60°. Motion in the index, minor, proximal joint is found to be 30°. The fraction found then is 60/90, which represents existing function. Estimates will be made on VOLUNTARY, NOT PASSIVE, motion. In finger immobilities give distance by which finger tips fail to reach palm of hand on active flexion and to what part of palm.

Signed this — day of — 19—

Surgeon executing blank sign here

Campbell,[1] a referee of the California Industrial Accident Commission, at Los Angeles.

Permanent Disability Defined. While the act does not define this term, a disability is generally regarded as "permanent" where further change—for better or worse—is not reasonably to be anticipated under usual medical standards. *It may be that no further treatment is possible, or that the only treatment suggested is so problematical of success as to warrant the employee's refusal to undergo it.* In such an event, it is permanent within the meaning of the act. In practical legal results, the healing period is over, and a permanent aftermath of disability exists. In connection with this result of injury, disability primarily means impairment of function in whole or part of a member or of the entire body.

Ordinarily the term "permanent," when applied to a personal injury means "lasting during the future life of the injured party." But, under certain insurance policies, it has been interpreted as meaning "lasting or continuous," as distinguished from transitory or temporary. Under United States War Risk Insurance, ". . . if it be reasonably probable that this status will long continue, . . ." it is permanent in legal contemplation. Influenced by the requirement of liberality in interpretation, under some compensation acts a disability is regarded as permanent when it is shown that the injured employee will suffer such disability for an indefinite period.

Because of the absence of a statutory definition, the California standard has been a literal interpretation of the term. This makes it a question of medical fact and involves an occasional insoluble problem of proof, complicated by the limitation upon the period of temporary disability. As a matter of substantial justice, any disability which lasts beyond the statutory period of temporary disability should, *ipso facto*, be regarded as permanent.

Permanent Disability in General.—Where an employee, aged sixty, has been totally disabled for several years, the condition is permanent. The mere possibility of slight improvement is insufficient to change the character of the disability. Loss of use of a part, as reduction of vision to merely light- and dark-perception, is total loss of the part.

Loss of Fingers and Toes.—In cases involving but one finger, for most occupations, there is a smaller rating for the amputation at the middle joint than for immobility at that joint. This is based on the theory that a finger which is stiff at the middle joint is in the way of the full use of the remaining fingers, and that one is actually better off without such a finger than to have it stiff and protruding. In general, there is no rating given for the loss of terminal phalanges

of the minor middle, ring, or little finger for most occupations. Certain occupations in mechanical lines do get a rating for loss of terminal phalanges of the major middle, ring, or little fingers, but most occupations do not get a rating for these phalanges, even on the major hand. There is no rating given for the loss of the distal phalanx of the second toe, for a laborer; the distal phalanges of the second, third, fourth, or fifth toes for most occupations; or the little toes for most occupations. Where the permanent disability rating has been exhausted by the temporary-disability payments, the making of the rating releases the carrier from further payment to the injured. A hernia remediable by operation is not regarded as a permanent disability.

Loss of Sight.—The loss of the sight of a single eye, the other remaining normal, is not so severe a handicap as the loss of an arm or leg, since the ordinary working-man or mechanic will teach his remaining eye to do the work of both eyes with an efficiency comparable to that of a two-eyed man. Consequently, the disablement inflicted by the loss of one eye cannot usually be rated as high as the loss of an arm or leg. However, the loss of both eyes is as severe a handicap as the loss of both arms or legs.

Loss of Teeth.—The loss of teeth is usually not rated, because it is possible to reconstruct such loss and provide a man with a satisfactory substitute. It is only when the jaw is so badly injured that artificial teeth cannot be supplied, in which the loss of teeth is both permanent and of such an extent that it interferes with the employee's ability to eat to maintain his strength, that a rating is given for the loss of teeth. Loss of sexual power does not constitute a ratable permanent disability under most acts.

Actuarial Background of Permanent Disability.—In California it is believed that permanent total disability is worse than death. In the case of permanent total disability the injured man not only is as unproductive as he would be if dead, but, in addition, is a burden upon his family to the extent that they must bear the cost of his personal expense. By a study of average expenses among wage-earners, it was determined that 40 per cent of a worker's wage could fairly be said to represent the cost of his own upkeep. On this basis, the worker is dead so far as his contribution to the support of his family is concerned when he becomes permanently disabled to the extent of 60 per cent and is thus, theoretically at least, just able to earn enough for his own support.

Life Pensions and Death Benefits.—The California schedule, therefore, provides the same compensation for a 60 per cent permanent disability as for death. From this point to 100 per cent, or total in-

pairment, the act provides a life pension of the difference between what is left of the employee's earning capacity and 40 per cent. Thus, in case of an 80 per cent impairment, the employee can theoretically earn only 20 per cent of his former wage, and the act therefore provides him with a pension of 20 per cent. In case of a 90 per cent impairment, the pension is 30 per cent, and where the disability is total the pension is 40 per cent. The compensation for permanent partial disability thus grades gradually into that for permanent total disability. The full death benefit is payable in all cases where the disability is rated 60 per cent or over in addition to the life pension, which does not start until the death benefits have been exhausted, 240 weeks from the date of the injury.

Basis of Permanent Disability.—If the injury results in permanent disability, the indemnity begins on the eighth day after the injury and is payable in the same manner as prescribed for temporary disability. The permanent disability rating is based upon the age and occupation of the injured man at the time of injury, and also upon the nature of the physical impairment or disfigurement at the time of rating, with due consideration being given to his diminished ability to compete in an open labor market. This is then worked out under the provisions of the Permanent Disability Rating Schedule, which results in a percentage of disability, entitling the employee to four weeks of indemnity for each 1 per cent of permanent disability.

For all ratings in excess of 70 per cent the employee receives 240 weeks of indemnity at 65 per cent of his average earnings within the limits provided, and thereafter, during the remainder of his life, 1 per cent of such average weekly earnings for each 1 per cent of permanent disability in excess of 60 per cent. If the rating does not exceed 69.75 per cent, it is paid at the usual weekly rate until received in full. The employee is entitled to and must be rated upon his handicap in the occupation in which he was engaged at the time of his injury, irrespective of his previous work. Where two forms of occupation are combined under the same employer and under the same general engagement of hire, the rating is based upon the occupation for which the greater disability exists.

Effect of Age Differences.—The permanent-disability ratings reflect age differences, since adaptability to a changed condition is dependent upon age. A boy of fifteen is believed to possess full powers of rehabilitation from an injury. He has not yet learned his life's work. If he receives an injury, he can change his life's work with reference to that injury and therefore be considered but little handicapped. When an employee reaches the age of seventy-five, he is regarded as possessing no power of rehabilitation. If the injury dis-

ables him and requires a change of work, he is industrially barred for the rest of his life. The different ratings for age are, therefore, apportioned through a range of from 7 to 7.5 per cent between the ages of fifteen and seventy-five, the greatest being for a man of seventy-five and the smallest for a boy of fifteen. The fact that there may be exceptions to this principle is ignored. The average man of that age, occupation, and disability is the necessary basis.

Schedule on Statutory Ratings.—In some of the states, typified by the Longshoremen's Act, and the schedules of Missouri, Colorado, and New York, there is a statutory schedule of indemnity for specific permanent disabilities. Under such a scheme, no distinction is made on account of the age of the employee, his intelligence, efficiency, strength, health, condition in life, or the like. The act bases the indemnity solely upon disability due to the loss either of a member, part of a member, or its function.

Such a plan is comparatively simple in operation, since it takes no cognizance of situations which may reflect a loss of earning-capacity greater than the average to employees engaged in specific operations. Where the average loss to workmen generally is the basis of indemnity, it can truly be said that there is little necessity for the receipt of testimony on the extent of such loss of earning or of functional capacity. The trained personnel of the compensation authorities is as well qualified to pass upon such matter as any other persons. The better practice, however, requires that the record contain either lay or expert testimony on the degree of disability.

The compensation provided by such a schedule is exclusive for the named disabilities. However, injuries resulting in partial incapacity not among the named disabilities are not controlled by the schedule.

"The Legislature evidently believed the loss of a specific member or organ deserved the compensation stated, whatever else occurred. If, however, additional injury should increase the workman's partial disability, either permanently or temporarily, he should receive additional compensation.[6] . . .

"If there is an additional or independent injury which produces disability, it is compensable, even though it is a consequence of the (scheduled disability). It is an independent injury that produces its own disability, separate and distinct from that resulting from the loss of the member. . . .

"Where, in consequence of amputation, injuries result which are distinguishable from those immediate results of the amputated limb, for example, if a nervous disorder ensue, or blood poisoning sets in, or a phlebitis develops, affections such as these were not intended by the . . . Act to be compensated in the loss of this member."[7]

Permanent-Disability Rating Schedule in California.—The degree of permanent disability is determined by reference to the rating schedule. It is based on the law of averages and is the result of intensive actuarial investigation and analysis of bodily functions particularized to the various occupational activities. The present schedule is that adopted in 1914 and has been modified but little since its adoption.

Provision for its use was made in the act, which did not include the schedule, however, since it was desired that the latter remain flexible and be subject to whatever change experience should indicate. The schedule is but *prima facie* evidence of the actual disability present. The schedule can be changed at any time by the commission and be effective for all pending claims, even though they were filed before the schedule had been amended.

Theory of Permanent-Disability Rating.—The theory of permanent-disability rating is that an employee of any stated age, occupation, and disability will be able to rehabilitate and readjust himself to his new earning-capacity within an average period. The ratings are so computed as to provide disability indemnity during that estimated period.

A former commissioner[6] has outlined the controlling principles incorporated into the schedule:

"The California schedule is based upon two theories: (1) that permanent injuries should be compensated for, not upon the basis of loss of future earnings, but upon the loss of earning capacity; and (2) upon the necessity of providing an adequate period of rehabilitation. . . .

"The underlying theory of the schedule is that there is a certain standard occupation and . . . age which can be used as a basis in determining the percentage of disability caused by various injuries; that all other occupations can be rated in accordance with the standard occupation, depending upon the physical requirement and skill; and that the age of the injured person can be made a function of the standard age, based upon the diminished ability to rehabilitate himself as he becomes older. . . .

"The standard occupation is taken as that of an ordinary unskilled workman and the standard age as 39. The occupation chosen makes an admirable standard because of the simple nature of the physical requirements placed upon the different parts of the body. Age 39 was taken as the standard age because statistical records at that time showed this age to be the approximate average age of persons injured in California."

Concurrent Indemnity for Temporary and Permanent Disability.—Due to the fact that the same injury may cause a series of grades of

disability, and each may have a statutory provision relating to it, beginning with temporary total, which merges into temporary partial and terminates with a permanent partial or total disability, some problem arises as to the amount of indemnity to be paid. This is primarily, of course, a matter to be determined by the particular act involved. Even so, confusion arises because of the declaration in some of the statutes that the schedule allowance for a stated permanent disability is "in lieu" of all other compensation.

The acts fall into two definite groups on this subject. Either because of the absence of the "in lieu" clause, or because of the liberal interpretation of the section by the courts, a large number of jurisdictions allow both temporary and permanent indemnity for the same injury. The statutes of this type vary from full payment for each grade of disability to but partial recognition of each within prescribed limits. The other group makes the schedule award exclusive. A provision for specific injuries does not necessarily provide for a combination of specific injuries.

How to Use the Schedule.—The California Permanent Disability Rating Schedule is divided into groups under roman numerals, each referring to some portion of the body, starting with the head and ending with the toes. For example, I refers to disabilities resulting from skull injuries; II pertains to eye injuries; III to ear injuries. The degree of disability is a subdivision under the respective headings, *e.g.*, complete loss of both eyes is II-1; complete loss of one eye is II-3.

1. A carpenter, aged forty-one, with amputation of the right, major hand at the wrist is to be rated. Under Table I (nature of the injury), page 6, heading "VIII—Hands," loss of the major hand is given as "VIII-1, line 41." Under Table II (occupation), page 13, carpenters are listed as "Form 20." Referring to Table II, Second Division, under the disability number "VIII-1" and under Form 20, we find the letter "D," which is the table to which the case belongs. At page 42 we drop to line 41 of Table D, and along line 41 to the proper age, forty-one years. This gives "52:0" or 52 per cent as the rating. In formula, the rating reads: "VIII-1-41-20-D-52:0." Otherwise stated:

Disability	*Line*	*Occupation*	*Table*	*Per Cent*
VIII-1	41	20	D	52

The percentages are stated 28:1, 12:2. These figures are not decimals, but quarters. That rating would be 28.25 per cent and 12.5 per cent. Only odd ages are given. The rating is taken at the nearest odd age. At age thirty years and four months, the man is nearer age thirty-one than twenty-nine, requiring a rating at age thirty-one.

2. A laborer, aged forty, has lost one half of the gripping power of the left, minor hand, with the thumb normal. In Table I, under disability heading, "XVI—All fingers not including thumb," we find "XVI-8, line 24," covering immobility of the minor hand. Proceeding as before, we develop the following formula:

Disability	*Line*	*Occupation*	*Table*	*Per Cent*
XVI-8	24	1	A	30:1

Disability in Silicosis and Allied Conditions

Estimating a disability resulting from an occupational disease, such as silicosis, has to be done without the use of concrete measurements and figures which serve as a basis of estimation in traumatic injuries. As has been many times intimated, permanent disability from occupational diseases is rare. However, silicosis and the allied conditions of anthracosilicosis and asbestosis constitute a problem of disability determination which remains unsolved. For a review of this phase of the problem, the reader is referred to the author's discussion under the medicolegal aspect of silicosis. It is apparent from the general nature of silicosis that a subsequent disability may never be anticipated by the victim, and yet years after having removed himself from the hazard he may break down as a result of his previous exposure and remain unrewarded. One thing is certain, that the numerous problems presented by the dust diseases can best be handled by the industrial accident commission or a specially appointed board in each state, rather than by lay juries.

Fixing Responsibility.—One of the chief problems has been the placing of responsibility for the disease, which involves fixing the date of disability. In many states the general rule both in workmen's compensation cases and in common-law cases is that the date of the accident is the date the disability commenced. This tends to place full responsibility upon the last employer. In California liability is *prorated upon the various employers*, according to the length of exposure with each. By a sort of general agreement among the insurance carriers, little emphasis is placed upon *dust counts* and the working conditions at the various places of employment. Formerly a defendant carrier would present dust counts and descriptions of the working conditions at the place of his insured in order to prove his liability was minimal, or none. But it was often contended that a dust count today or the working conditions today cannot be assumed as exactly representing those of some years previously at the same place of employment. Today, in California, liability is estimated largely upon the basis of length of exposure in each employment. To illus-

trate, a claimant has worked in a silica hazard for 5000 days, 2000 of which were spent in another state. Since this state has no power to secure action or decision from another state, these 2000 days are deducted. In this state, the claimant worked for 3000 days and for three different employers, for employer A, 1000 days; for B, 1500 days; and for C, 500 days. Each employer is, therefore, held for his proportionate amount. To the writer this method appears fair. First of all, it dispenses with the cost of liability in cases where the exposure occurred in other states. If a coalminer from the anthracite district of Pennsylvania drifts into goldmining in California, a California employer should not be held liable for the days spent in Pennsylvania. It dispenses with quibbling regarding the extent or manner of exposure.

Disability Prior to Enactment of Laws.—A problem which confronts the states recently assuming liability for disability from silicosis is the matter of easement of payment for a total disability which has accrued prior to the enactment of provisions for compensation in that particular state. New York, Ohio, Pennsylvania, Michigan, and West Virginia, to mention only a few, have had to face this situation. The intention and attitude of the Bureau of Workmen's Compensation of the state of Pennsylvania illustrate this phase. The following is based on Challener's discussion[7]:

The claimant must have had an aggregate employment of at least two years in the Commonwealth of Pennsylvania in an occupation having a silica or asbestos hazard, during a period of eight years next preceding the date of disability.

Under the Rules of Procedure of the Workmen's Compensation Board (Rule 41) the two-year period may be calculated in any one of three ways:

1. When during any two calendar years in the preceding eight years he has been employed in such an occupation; or
2. When he is employed in such an occupation during the eight-year period for a total number of working days equal to twice the number of days the men employed in that industry work during an average year; or
3. When he has been employed in such an occupation during any twenty-four calendar months during the eight-year period.

Compensation by Employer.—Section 5*b* of the Occupational Disease Act provides that, "The total liability of the employer unto the employee or his dependents under this section shall not exceed the sum of $3,600.00."

The Chairman of the Board of the Pennsylvania Bar Association, in discussing the silicosis section, calculated that a man 44.6 years old, who became totally disabled, had a maximum life expectancy of 25 years, and if he lived his full expectation of 25 years, he would receive $14,400. A reverse calculation indicates that his theoretical man received $18 per week for 500 weeks, or $9000 plus $30 per month for the balance of his expectancy (15 years) or $5400 or a total of $14,400.

This method of calculation is apparently based upon Section 4 of the act, which provided:

> When an employer and employee shall be subject to the provisions of Article 3 of the Workmen's Compensation Act as therein provided, compensation for occupational disease shall be paid in all cases by the employer according to the schedule provided in such Act, subject however, to the special terms and conditions relative thereto, as set forth in this act.

Compensation by State.—In determining whether the employer's liability is $3600 or greatly in excess of that amount, as intimated by the Chairman of the Board, it is also necessary to consider Section 7*a* of the act.

This is the section that provides for part payment by the Commonwealth, starting at nine tenths the first year and decreasing one tenth each year until in ten years the employer will have paid the compensation in full.

To take a concrete illustration, let us assume that a man is totally disabled on January 1, 1938, from silicosis. The Occupational Disease Compensation Act does not specify his weekly rate of compensation, and it is necessary to refer to the Workmen's Compensation Act which fixes it at $18 per week, assuming that his earning-power justifies the maximum rate. We would then have this situation. His compensation at $18 per week would amount to $936 a year; under Section 7*a* the state in the first year would pay nine tenths of that amount or $843 and the employer would pay one tenth of that amount or $93 a total of $936.

	State	*Employer*	*Total*
First year	$843	$ 93	$936
Second year	750	186	936
Third year	657	279	936
Fourth Year	476	316	792
	$2726	$874	$3600

By the end of the third year he has received $2808, so that if $3600 is the maximum compensation to which he is entitled, the payments would run out in the fourth year. But while the employee has received $3600 the employer has paid only $874. Does the employer owe anything more? It should be noted that Section 5b does not create a liability of $3600, but on the contrary states that the liability of the employer shall not exceed $3600. The language seems to be significant, and the result is, therefore, in accordance with the terms of the act. Any other interpretation would produce an anomalous result.

If this were not so and it should be held that an employer must pay $3600 in all cases, then our theoretical man who became disabled in January, 1938, would continue to receive compensation for nine years as follows:

	State	*Employer*	*Total*
First year	$843	$ 93	$936
Second year	750	186	936
Third year	657	279	936
Fourth year	564	372	936
Fifth year	471	465	936
Sixth year	378	558	936
Seventh year	285	651	936
Eighth year	192	744	936
Ninth year	20	252	272
			$7760

The net result would then be to give an employee who became totally disabled in 1938 a payment of $7760, but another employee who sustained the same disability ten years later would receive only $3600, as in his case there would be no contribution from the state.

In a personal communication from Challener[5] regarding his discussion of the above phase of the Workmen's Compensation Act in Pennsylvania, he writes, "At the time I wrote this paper, there had been no decisions determining the amount of liability." Implying that a recent interpretation had been handed down, Challener says further in his letter that the Workmen's Compensation Board has interpreted the act ". . . as restricting the liability of an employer and the Commonwealth of Pennsylvania to the total payments of $3600.00 in all cases of silicosis, anthraco-silicosis and asbestosis. Under the Board's decision, the liability of an employer to pay compensation, where the silicosis results in total disability during the first year, is restricted to one-tenth of $3600.00 or $360.00 for his entire disability. The Commonwealth's obligation, where the silicosis results in total

disability during the first year is $3240.00. . . . However, there have been no appellate court decisions deciding this matter."

Similar restrictions are found in the statutes of other states:

Ohio (Laws of 1937, Section 1465, 68a) *New York* (article 4a, Chap. 887, Laws of 1936, Section 66, effective June 6, 1936), and *Michigan* (O.D. Amendment 1937, Section 4) restrict the liability of the employer by fixing a maximum benefit of $3000, with a limitation of $500 for disability occurring during the first month after the act became effective plus $50 additional for each month thereafter until the maximum of $3000 is reached.

The West Virginia act, following the precedent set in South Africa, divides silicosis into three parts and awards $500 for the first stage, $1000 for the second, and the full benefits of the Workmen's Compensation Act for the third.

OPERATION OF INDUSTRIAL ACCIDENT COMMISSION

To this point, the discussion in this chapter has considered the manner in which the intentions of the Workmen's Compensation Act are fulfilled. A brief further discussion is necessary of that body which has the power to enforce the act. This body in California is known as the Industrial Accident Commission, as it is in most states, and its duties in this state closely parallel those of the other states.

"The Act creates a board of three members known as the Industrial Accident Commission. Claims for compensation are filed with the Commission; hearings are held and awards are made.

"The Commission may make and enforce safety rules and prescribe safety devices. Its procedure is largely judicial. It has powers to administer oaths, issue subpoenas, take testimony and punish for contempt. Its proceedings commence by application in writing followed by notice of hearing. The statutory rules of evidence and procedure do not govern. The Act gives the Commission a continuing jurisdiction over its awards for 245 weeks, and it may during this period alter or amend them for good cause. Appeal lies to the Supreme Court or District Court of Appeal by a writ of review. The findings and conclusions of the Commission on questions of fact are conclusive and final and not subject to review. The Appellate Court is limited in the appeal to questions of whether the Commission acted without or in excess of its powers, or the order, decision or award was procured by fraud or was unreasonable; or that the findings of fact did not support the order, decision or award under review.

"The Industrial Accident Commission is a division of the Department of Industrial Relations. Its functions are performed by various departments within this division.

"The judicial division of the Industrial Accident Commission is called its 'Compensation Department.' Headquarters are at San Francisco, with a substantial branch at Los Angeles. A staff of more than twenty judicial officers known as referees decide the claims brought before the Commission by employees, with all decisions subject to confirmation or approval by the Commission itself.

"Referees travel in various parts of the State each week to hold the hearings necessary to permit the presentation of claims by injured men or women in all of the State's 58 counties. With a relatively small staff of officials and employees, the Compensation Department thus covers the State, hearing and passing upon the amazing total of 10,000 original claims of compensation each year. Supplemental matters usually requiring an equal amount of procedural work account for about 4,000 more decisions per year.

"This extremely busy department awards annually an estimated total of nearly $7,000,000 in compensation in addition to medical benefits. Its decisions form the basis for disposition of thousands of unlitigated claims totaling many millions more.

"Hearings are conducted by referees and juries are dispensed with. Medical evidence is nearly all submitted in writing and points of dispute are reduced to a minimum by stipulation. Parties need not be represented by an attorney unless they so desire, since the referee will examine all parties and witnesses regarding the facts of each case. These procedural short-cuts greatly reduce the time needed for the presentation of a controversy. The average case is pending less than eight weeks, as compared with former periods of from six months to six years in civil courts.

"**Medical Department.**—In the consideration of cases, the Compensation Department is assisted by two related bureaus, the Medical Department and the Permanent Disability Rating Department. The Medical Department examines thousands of applicants annually without charge to the injured person, and the medical files of thousands of other cases are reviewed and expert medical opinions are given orally or in writing to the referees.

"Bills for medical services to injured men are approved or revised. Cases of an especially difficult nature are referred to independent medical experts chosen by the Medical Department. The entire work of this department is directed primarily toward assisting the Compensation Department to decide cases before the Commission.

"The Permanent Disability Rating Department is also of great assistance in formal matters before the Compensation Department, as well as in many cases where no claims have been filed. The determining of the extent to which an employee has been permanently

crippled by an injury is frequently an involved problem. These problems, however, have been greatly simplified by means of a schedule which takes into consideration the workman's age, occupation and the nature of the injury. The adjudication of the amount of compensation as determined by the schedule for the disability suffered is the work of the Permanent Disability Rating Department.

"**Legal Department.**—The Legal Department, through its attorneys, represents and appears for the Industrial Accident Commission in all actions and proceedings arising under the provisions of any of the law administered by the Commission. The decisions of the Commission are defended by the Legal Department in all appellate proceedings. This involves preparing briefs and making oral arguments in the State District Court of Appeal, the State Supreme Court, and the United States Supreme Court.

"It is also the duty of the Legal Department, through a specially appointed law enforcement officer, to enforce the compulsory features of the Workmen's Compensation Law. Employers who are reported as not carrying compensation under the present practice are at first warned that they are committing a criminal act, and if the warning is disregarded and violation continued, or if evidence of previous violation is found, prosecution proceedings against the employer are instituted. A permanent file of all employers reported as uninsured is kept by this department and a program of enforcement through cooperation with local law enforcement agencies throughout the State is maintained.

"The law also gives the Commission, through its Legal Department, the right to file injunction proceedings against any employer failing to abide by the provisions of the law requiring compensation insurance.

"In addition to the foregoing, the Legal Department has the duty of bringing criminal proceedings for violation of the safety laws and orders concerning air tanks and boilers, elevators and motor boats. It acts as legal counsel to the Commission, rendering legal opinions when required, and represents it in all litigation involving the Commission.

"**Accident Prevention.**—The Commission has the power and supervision over any employment and place of employment in this State for enforcing its safety orders. This function lies with the Bureau of Industrial Accident Prevention.

"The Commission may require and prescribe what safety devices, safeguards or other means of protection are adapted toward the safety of employees. It may fix reasonable standards and enforce reasonable orders for adoption of reasonably uniform safeguards.""

Finally, after consideration of all that has been previously written in this chapter, it can be summed up by stating that the purpose of workmen's compensation is adhered to by the physician who renders his opinions after a complete study upon an unbiased basis. He practices this phase of medicine best who disregards the insurance carrier, the patient, the court, and the social or economic consequences of his decision. Upon his integrity and sanity rests the structure of one of the finest laws ever to be promulgated.

REFERENCES

1. *Information Regarding the California Workmen's Compensation and Safety Law*, Industrial Accident Commission, San Francisco.
2. McBride, Earl D.: *Disability Evaluation*, J. B. Lippincott Co., Philadelphia, 1936, p. 41.
3. *Ibid.*
4. Campbell, D. A.: *Workmen's Compensation*, Parker, Stone, and Baird Co., Los Angeles, 1935, Vol. I, pp. 719–732.
5. Franko vs. Wm. Schoffhorn Co., 93 Conn. 13, 104 Atl. 487, 1918.
6. Naftzer, A. H.: U. S. Labor Bulletin No. 281, pp. 64–66, 1931.
7. Challener, Wm. A.: *Lecture on Compensation Laws*, Dept. of Ind. Hygiene, School of Medicine, University of Pittsburgh, 1939.
8. Personal communication from Wm. A. Challener to author, May 2, 1941.
9. *California Safety News*, 24: 4 (June), 1940.

CHAPTER III

THE FUNCTION OF THE PHYSICIAN AS RELATED TO WORKMEN'S COMPENSATION

THE *recognition and prevention of disease*, or the proper application of a remedy when disease presents itself, has traditionally constituted the duty of a physician. But the practice of medicine under the administration of the Workmen's Compensation Act imposes additional obligations. In addition to a knowledge of the law there exist certain other requirements necessary for the adequate disposition of this work. The employee, employer, insurance carrier, and the court assume that the physician understands his relationship to them. But since physicians generally have received little or no instruction on this relationship, there exists a lamentable state of misunderstanding. Where shall the doctor or student obtain the fundamentals sufficient to render him competent in this work? In this chapter the intention is to enumerate these fundamental requirements and to suggest acceptable methods of meeting them.

Scope of Act in One's Own State.—The first duty of the doctor is to acquaint himself with the scope of the act as adopted in the state in which he practices. It behooves him to secure a copy of the act from the State Department of Labor, or the Industrial Accident Commission. In the previous chapter it was intimated that it is not necessary that a physician be informed on all the technicalities of the law. It is not expected that he be a quasi-attorney. Many points of the law do not concern him, nor should they affect his conclusions on the medical aspects of the case; but on the other hand there are certain sections of the act which involve both *medical and legal interests* and which should be understood by the physician if he is to avoid errors in the conduct of an industrial practice. For instance, the patient who enters a claim for the first time nine or ten months after an alleged injury supposedly occurred is barred, in most states, by the *statute of limitations*. This is primarily a point for legal decision, but a physician should be cognizant of it so that he will deny treatment until properly authorized. It is not infrequent to find this factor of time interval ignored. It should be understood what constitutes treatment. As an example, a patient who desires to reopen his case, which has been closed some months before, is sent to a doctor for examination only. The examiner should offer no advice, suggestions,

nor give a prescription, for in most states this is considered by the court as "treatment." The statute of limitations is thereby waived, and tolling of the statute by the doctor has been indulged in.

Groups Excluded from the Act.— The industrial physician should know what groups of people, if any, are excluded from the benefits of the act. In some states, farmers and domestics are not covered by the act. In California, a domestic is covered if she works fifty-two hours a week. Some states specify what disease will be compensated, thereby excluding all others, while certain states have unlimited coverage. In some of the commonwealths there exists a limitation in time and cost of medical and hospital benefits in compensable occupational diseases; in others it is unlimited. Special provision is made relative to the status of hernia in twenty-five states. "Aggravation of a pre-existing condition" receives a varied interpretation, and in Connecticut and Florida disability or death due to the aggravation or acceleration of venereal disease is not compensable. These are but a few of the peculiarities of which the doctor should be informed.

Advantages in Knowing the Law.—An understanding of the law not only aids the physician in protecting himself but permits him to be of greater service to the workman. To illustrate, instituting proceedings for compromise and release settlements is a function ordinarily confined to the legal interests or the claim department. Most states require the approval of the court before this can be entered into. A few permit this practice without interference. Not often, but occasionally, the physician who appreciates the benefits of this alternative may suggest it for the good of the patient, especially when he has studied the ability, character, and capabilities of the disabled one.

A branch manager for a camera concern on the West Coast received a compensable skull fracture which entitled him to a weekly compensation of $25. While not physically disabled it was apparent he could never resume his executive work. It was suggested to him that he accept a lump sum of money, release the carrier of all further responsibility, and invest this money in a small photographic supply store, a business in which he had had experience, and one in which the responsibilities could be shared by his wife. This he did, and today at a lake resort he has a business which yields a greater return than his compensation. Furthermore, he is useful and happy. Similar examples could be cited. Many times a good industrial physician who is aware of this existing possibility in the law can serve in rehabilitating the working-man. However, he should discriminate between the responsible and the irresponsible. Some might dissipate a lump sum of money, as did the dissolute soldier with his bonus.

References.—A discussion of many other aspects of the compensation act cannot be indulged in at this time. For those interested, it is recommended that they obtain for reference a book entitled *Workmen's Compensation Acts in the United States*, published by the National Industrial Conference Board, New York; also, *Administration of Workmen's Compensation* by Walter F. Dodd, the Commonwealth Fund, New York. Another excellent reference is an article by E. Ransom Koontz, M.D., in the *Journal of the American Medical Association, 114*: 563 (Feb. 17), 1940, entitled "Compensation for Industrial Injuries and Occupational Diseases." An intelligent comprehension of the law constitutes the first step in becoming an efficient industrial physician and often demarcates one from the routine first-aid man.

Importance of Keeping Records.—The next important step is the establishment of a well-systematized office. "Paper work" is abhorrent to most doctors, but the handling of compensation cases requires meticulous adherence to details. First reports must be followed by periodic progress reports. Records must be kept in a manner acceptable to the courts and accessible months or years later. Case histories and especially *x*-ray films should not be destroyed for at least five years. Such a procedure not only is important from a legal standpoint, but is of value to him who, at intervals, wishes to read a paper based upon a series of cases. The office should be run much as a hospital record room with emphasis upon the nomenclature used and the methods of filing adopted. As this is being written, the Council on Industrial Health is preparing a recommendation on standard terminology for industrial medicine. A universal conformity to this would seem advisable.

The Doctor's Fee.—A doctor's bills are often disputed by insurance companies, therefore charges for service, supplies, or physiotherapy should conform to the accepted fee schedule of the district, and extra charges should be explained. The author knows of a young physician who for more than a year made long trips to see patients, charging only the accepted fee for calls, not knowing that he was entitled to additional remuneration for mileage beyond a certain zone. It is recommended that the young physician or beginner in this field inspect the methods of service and office management used by men long experienced in this work. In addition he should visit the claim departments of several insurance companies in the nearest metropolitan area in order to learn their methods of handling the claim and to ascertain what is expected of him. The old family doctor managed to live in spite of his indifference to bookkeeping through contributions of eggs, butter, and chickens, but such procedure is not avail-

able to the industrial physician. His financial success is not alone dependent upon his professional ability. The installation of a good system and the employment of a good bookkeeper are factors of competence in the service rendered and the returns gained.

Court Testimony.—A despised but required function of an industrial physician is his testimony in court or before an industrial referee. The writer wishes to enlarge upon this duty. Competent testimony in the compensation case is rare. In general medicine, the knowledge of the disease in question is fairly well understood by every physician. The etiology, symptoms, complications, and treatment of pneumonia, pernicious anemia, syphilis, etc., form a part of the experience of all practitioners of medicine. This cannot be said of silicosis, benzene, carbon tetrachloride, dichlorethylene, or other poisons. It is not infrequent to find divergent opinion offered by physicians who have had no experience with the causative agent involved. Honest mistakes are bound to arise in any medical testimony at times, due to confusion or emotional imbalance of the witness; or to the failure to be properly prepared; or to being unwittingly led into erroneous commitment by a lawyer. The great majority of errors made by the medical witness in an industrial court are the result of ignorance and of inadequate information regarding occupational diseases. For example, a young physician made certain misstatements regarding the effects of sulfur dioxide, during a trial and when cross-examined, he admitted his statements were guesses, since he had had no experience with this gas. Another physician stated on the witness stand that the bronchiectasis shown in the films of a pottery worker's chest was due to silicosis which had resulted from the patient's exposure to silica. The facts were that the patient had worked six months in a "wet process," and the films showed no evidence of silicosis. Off the stand and off the record the physician later admitted that he had read of silicosis but had never seen an *x*-ray film depicting this disease. Testimony of this type is damaging to the true facts of the case, but unfortunately the referee or courts do not always recognize the inexperience or lack of authority behind such testimony.

Difference between Possibilities and Probabilities.—What the medical witness needs to appreciate, and what the referee or judge should be careful to discriminate, is the difference between possibilities and probabilities. Too often possibilities exist but the probabilities are remote, and yet an inexperienced physician will permit a clever attorney to secure an admission of unlikely probabilities. An example of such an incidence is the case of a truck-driver who picked up the end of a narrow, light piece of pipe and stated that at the time he noticed a mild twinge in his right lower abdomen. The incident

was forgotten. Three nights later he was awakened with pain, nausea, and vomiting. He was operated upon for appendicitis, and following the operation he recalled the occasion of lifting the pipe and subsequently filed a claim for appendicitis resulting from lifting. His physician testified that the act could have produced the pathology which necessitated an operation. The author was asked upon the stand by the claimant's attorney, if it were not possible to produce or aggravate appendicitis by the act described. His answer was that he personally might become president of the United States, but that it was vastly improbable. It is imperative in these medicolegal disputes that the physician adhere to the likely and more reasonable elements rather than those extremely unlikely and improbable. What is needed is a more uniform basis for evaluating the relationship between the hazard and the disease. Undergraduate and postgraduate training will partially fulfil this need, but it can never be fully obtained unless the doctor ignores his affiliation with the employer, or carrier, or divorces his tendency to sympathize with the patient.

It is obvious that there is a great national urge to care for the unfortunate. Such a trend is inevitable and cannot be associated with, or attributed to, any particular political group. Unemployment insurance, old-age pension, and other legislative acts are born of economic necessity. Their provisions must not be confused with the benefits of workmen's compensation. To fulfil faithfully the requirement of court testimony, the physician should be prepared with data relative to the history of the case, the physical and laboratory findings, a knowledge of the disease and of the accepted postulates concerning that disease. Fallacious assumptions are repeatedly cited in the following pages.

The Medical Report.—All employers or insurance carriers have a standardized form, "The Surgeon's First Report," which they ask to be filled out immediately and forwarded to the nearest office of the company. Such a succinct report is sufficient only when a minor injury is sustained, such as cuts, burns, or minor fractures. It is insufficient in severe degrees of trauma and never satisfactory in a case of occupational disease. The occupational diseases are rarely acute, seldom immediately apparent, and usually require detailed study and investigation. Obviously it is impossible to impart the result of such endeavor on a stereotyped, one-page printed form. These cases require letters of some length. The reason a letter is essential is explained in the following paragraphs. Regardless, however, of the type of report submitted, it should be done with as little delay as possible. This sets into motion the machinery relative to the case, enabling the workman to receive his compensation when due if the case is

compensable; or permitting the carrier to investigate the facts surrounding the claim before accepting or denying their responsibility.

In a well-written letter the occupational environment will be described, the non-occupational factors cited, the symptoms, physical and laboratory findings listed, and the entire mass of information evaluated in the conclusion or discussion. If the disability is due to occupation, then the extent is estimated. These important parts of such a letter deserve further description.

The History.—The art of case-history taking cannot be imparted to others. The student may be instructed upon forms, methods, or outlines to follow, but the ability to ferret out the essential facts either resides in him or doesn't. It is the idiograph of a good clinician. Much of the importance in the narrative of the patient may be read between the lines. To follow the trail as it wends its way without losing it by deflecting questions elicits a clear-cut story. During ten years of teaching physical diagnosis, the author constantly emphasized that the patient should be permitted to *recite his history before the historian writes down a word.* The habit of beginning the interview by immediately setting down on paper the statements of the patient and having him wait until the historian catches up with him results in a stultified, incoherent story. This practice is to be especially condemned in industrial medicine for the following reason. Frequently the patient has been previously interviewed by a claimsman, investigator, or insurance representative. To these he has given a statement which has been written down word for word and then signed by the patient. If then the doctor to whom he has been subsequently referred immediately starts to record every statement made, the patient is most apt to assume that the history he is giving is further legal information, not medical, and therefore he "freezes" or guards his every utterance. How much better it would be if the physician sat back and listened to the story, guided it as he saw fit, and then recorded it at its conclusion, going back if necessary to clarify certain points!

The history should be written in the words of the patient and not in the third person. It should ordinarily classify the case as obviously compensable, obviously not compensable, or one where compensability can be determined only after a complete study. In the writer's opinion this history should not be too short. An alleged occupational illness demands the recording of seemingly irrelevant facts. It not only must include the customary details concerning the onset as would be of interest in any illness, but must delve into factors of occupation such as the materials handled, their composition, their noxious or inert form, the content of the atmosphere, the length of time of an acute or

chronic exposure, the use or non-use of respirators or other protective devices, the adequacy or inadequacy of ventilation, the previous occupation of the patient, his previous medical and family history. Some of this information cannot always be obtained from the patient, nor always from his employer. Solvents vary in their chemical composition, and the concentration of metals or dust in the air may not be known to the workman or to his employer. In such instances, reliance must be placed upon the hygienist, engineer, or chemist. It is inadvisable to assume the correctness of the patient's statement regarding the environment of his work or the composition of the materials he handles.

The Physical Examination.—It would seem impertinent to suggest that this be most complete, yet frequently it is not complete. This negligence occurs more often in industrial medicine than in general medicine, apparently because of faulty assumption. In general practice the patient presents his symptoms to the doctor, seeking an explanation of the cause. In industrial medicine the patient *tells* the physician that his symptoms are the result of a certain exposure or incident. There exists, therefore, a tendency to accept the patient's theory without an investigation or complete physical examination. The alleged back sprain is an excellent example of this type of assumption. In the chapter on "The Industrial Back," the author cites his study which reveals that nearly 70 per cent of the cases previously accepted by physicians as back sprain were, upon investigation, due to conditions not of industrial origin. In most instances pain was due to obvious but overlooked focal infection. Cancer of the spine was treated as a back sprain; a patient with well-marked pyonephrosis received physiotherapy for a sacro-iliac slip; the pain of aortic aneurysm was attributed to an inhalation of formaldehyde, and the patient received many weeks of compensation. Malingering has been attributed to patients with fractured vertebrae or nucleus pulposus without benefit of a roentgenological or neurological examination. Additional instances would further serve to emphasize the urgency of a complete physical examination upon the presentation of even a trivial complaint.

The Laboratory Investigation.—There is a prevailing opinion among the members of the profession that insurance companies will not pay for special laboratory procedures. This is not true. The failure to investigate the case properly from a laboratory standpoint, combined with inadequate histories, incomplete physical examination, and indifferent cooperation, constitutes the chief reason for the tendency of insurance companies to centralize the examination and treatment of their patients in the hands of a few doctors upon whom they can rely.

Any physician who attempts to establish his diagnosis by the aid of special tests will receive favorable recognition from these companies and will find them willing to permit the case to remain in his hands. The reason is obvious. In any alleged case if the laboratory investigation substantiates the physical examination in establishing the authenticity of the claim, money is saved to the carrier and litigation is avoided by instituting treatment to hasten the patient's return to normal health. As an illustration, consider the following two methods of reporting a case of alleged lead poisoning. In a small town, one hundred miles from a metropolitan area, a workman develops an illness which he believes is the result of exposure to lead. Dr. A. examines him, does a blood smear for stippled cells, and reports his diagnosis accompanied by a bill for $7.50 or $10. In this same town, Dr. B. examines the same type of case. In addition to the blood smear he does a complete blood count, collects by accepted methods a twenty-four-hour urine specimen, and in a proper container mails it to a testing laboratory in the city for evidence of normal or abnormal amounts of lead in the urine. Upon receiving the report, he forwards his diagnosis accompanied by a bill for blood count, blood smear, special examination of urine, postage, and his examination fee. His bill may be $25 or more. The second and more comprehensive report is the more valuable, for it saves further consultation or repetition of tests if litigation arises. Charges for needless laboratory study are soon recognized as padding the bill, but procedures carried out to establish the diagnosis, and accompanied by an explanation of their expediency, are rarely questioned.

Conclusions or Discussion.—All letters to the carrier pertaining to an occupational-disease claim should end with a discussion of the positive and negative findings. In it the physician evaluates the factors which have led him to his decision or diagnosis. In a medical report from one physician to another, the physical examination and laboratory reports are of predominant importance. But in a letter which is to be read and understood by a claimsman or the court, the final discussion receives the greatest attention. This discussion should be devoid of scientific terms, or if they must be used, they should be immediately explained in lay terms. In the recital of findings in the physical examination or special investigation it may be impossible to avoid scientific nomenclature, such as Hoffman's sign, Babinski's sign, or that 0.08 mg. of lead was found in the urine. In the discussion, these must be explained as to their significance in establishing or denying the patient's claim. Findings and tests have value to the layman only when they are understood. That portion of the medical report devoted to discussion is similar to the rebuttal in a debate. In

it the clinching statements to all that has been related previously are made. It should be well organized and endowed with reason, judgment, and truth.

Estimation of Disability.—It is incumbent on the industrial physician that he state in a case that is proved to be non-compensable that there "exists no disability arising out of the occupation." If a case is acceptable he should estimate the length of temporary disability and the possibility of permanent disability. It should be appreciated that the employer or insurance carrier is required by law to set up a reserve for all cases in his files. This reserve is similar to that carried by life-insurance companies and signifies the stability of the company to meet the claims that might be made. The self-insured or insurance carrier is subject to an examination of his books at any time and without notice by the state auditors. He is dependent therefore upon the medical reports to set up this reserve. In addition to estimating the length of time for a temporary disability and the probability, or improbability, of permanent disability, the period of treatment should be predicted.

The Attitude of the Industrial Physician.—In a day of radicalism, communism, and altered beliefs in all walks of life, an exhortation to hew to the line is not amiss. Medicine long ago emancipated itself from the shackles of priestcraft. While various schools of thought and philosophy have come and gone, medicine has maintained its unbroken continuity of methods and ideals. Industrial medicine has freed itself of the unwarranted stigma of charlatanism. Never has its future been so bright, and no branch of the profession offers the young medico a more brilliant career. The development of new industrial hazards offers a problem for solution not paralleled in any other type of medical practice. The field is new, not crowded, and the call is urgent for the right type. But he who answers must be qualified. It is not given to every physician to be a surgeon, or to everyone to have research ability. Just so it should not be expected that everyone can practice industrial medicine successfully.

Toward the Patient.—Among the important attributes is his attitude toward the patient. His demeanor toward the injured workman should be no different from that of the general practitioner toward his private patient. If anything, it should be more indulgent, solicitous, and sympathetic. While there are individual cases of malingering or falsification of claims, these are not frequent. When present, they must be denied. The majority of injured or ill workmen are desirous of obtaining relief and being rehabilitated. Frequently they misunderstand the relationship of their illness to their occupation, or they misunderstand the purpose of compensation. The

examining physician, through kindliness and indulgent explanation of the conditions, may win the patient's gratitude and understanding, so that he offers no problem in the equitable disposition of his case. One outstanding fault existing among industrial physicians is the tendency to consider the patient a "case number" and hurry him through in order that the next one may be quickly served. This results in errors in diagnosis and, as previously intimated, a too frequent acceptance of a case as compensable when in reality it is not. Again let it be emphasized that the occupational-disease claim requires laborious effort and is rarely ever to be hurried through as an emergency. Finally, in regard to the attitude toward the patient, it must be remembered that the intention of the law is that in doubtful cases the benefit of the doubt is to be given to the patient. The least one can do in these cases where uncertainty exists is to state honestly that the diagnosis is not certain and a definite fixation of liability cannot be made. In such instances the court or Industrial Accident Commission will recognize the honesty of the report and will decide as the findings warrant.

Toward the Employer or Insurance Carrier.—The attitude of the physician toward the employer or insurance carrier is one which, at the present time, causes grave concern. The philosophy of this era has tended to make compensation insurance, health insurance. It is not infrequent to find a physician who will say, "The poor devil has no money, and the insurance company can well afford to care for him even if the condition isn't due to his work." This is altruistic and commendable from a standpoint of humanitarianism, but it is not honest. It is unwittingly an insidious undermining of the basic structure of compensation. This thought is repeatedly stressed throughout this book. In the previous paragraph it was urged that the virtues of sympathy and understanding be extended to the patient, but this sympathy should not alter one's judgment. The industrial physician must constantly guard against favoritism. Once the condition is apparent as being of non-occupational origin, the opinion must be so expressed. The plea for rigid adherence to the honest acceptance or denial of a case is not made in order that the insurance companies may be favored, but that the purpose of the compensation act may be observed and preserved. The industrial physician should respect the rights of the general practitioner or the family physician. If the case is determined to be of communal origin, the patient should be referred to the family doctor. It cannot be too strongly emphasized that there be no competition between these two phases of medicine.

Toward Industrial Practice.—Finally, the physician should never consider an industrial practice a side issue in his professional duties. He should not feel aloof to this type of work. Most of the gross errors in this field come from the doctor who conducts a general practice and, for pecuniary reasons, indulges in a partial industrial practice. If he cannot gain the full vision of the purpose of industrial medicine that is so necessary to the successful pursuit of this type of practice, he would better refrain from any participation in it. The first step toward success in any occupation is to become interested in it. This requires of the industrial physician not only a knowledge of the diseases common to industry, but in addition an understanding of the law under which these diseases are compensated. The disciple of Chauvin can find no harmonious niche in industrial medicine. The requirements demand practical adherence to a simple, workable legal enactment evolved for the good of the workingman.

ACCEPTABLE METHOD OF REPORTING AN OCCUPATIONAL DISEASE

The following case history may serve as a model to those unfamiliar with an accepted method of reporting an alleged occupational disease.

Gentlemen:

At your request, Mr. Jack X—— reported to this clinic January 2, 1940, for an examination. He is fifty years old, and gave his address as Mojave, California. He had previously been examined May 17, 1939, and certain facts in the history and certain physical and laboratory findings of that time will be incorporated in this letter. The patient relates the following history:

History.—"About one year ago I was exposed to fumes as a result of some dynamiting. They blasted in the mine at 2.45 in the afternoon but the men did not return to work until 7.00 A.M. the following day. In the meantime they had used the blowers to draw out the fumes, but when we returned to work the gas was still present. I and my partner were sent down to this particular level where they had been dynamiting, and while in there my partner dropped dead. I don't remember exactly how I felt, but it did not seem to bother me much just at that time for I went up to the surface unaided and made my report to the boss. I stayed away from work for about one week following this accident and then returned to work. After that, however, I was off a number of times because I felt sick.

"In the past year I have lost a good deal of weight, have become very short of breath, have pains in both shoulders, and I cough a great deal. No; I don't spit up with this cough very much. As far as I know I have never had any fever. I seem to be getting worse,

and I think it is due to that gas and also due to the dust. I think both of them have affected my lungs."

Past Medical History.—Usual childhood diseases. Influenza, 1918. Denies any other serious illness since reaching adult life.

Family History.—Father died at the age of eighty-six from general senility.

Mother died at the age of seventy; cause unknown.

Three brothers living and well; two brothers dead, both because of accidents.

Four sisters living and well; two sisters dead; cause unknown.

Wife and four children, living and well.

Occupational History.—The patient's first employment was with the Iron Blossom Mining Company in Utah at the age of eighteen, at which time he was engaged in laying a pipe line. He also worked for the Chief Consolidated Mine in Utah, being engaged in mining silver and lead. He was engaged in mining gold and copper for a short period with a company called, he thinks, the Keystone Mine.

His mining experience in Utah was from 1907 to 1937. This was not all underground, for at times he was employed above the surface dumping cars, repairing tracks, mucking, caring for mules, etc. He came to California in 1937 and began working for the Golden Queen Mine in August, 1937. With this company he has worked as a tram operator, as a mucker, and as a stopper and liner. He states that the operations for this company are conducted under conditions of wet mining.

Physical Examination.—The patient is a poorly nourished adult male, whose present weight is 127 pounds. Two years ago his weight, he alleges, was 155.

His temperature was 98.3 degrees. The pupils reacted normally to light and accommodation. The eye grounds were negative. The ear canals and drums were normal. No nasal obstruction. No cervical adenopathy.

Teeth.—Marked pyorrhea involving the gums of the incisors and bicuspids. Many teeth missing. Tonsils atrophied.

Chest.—Inspection revealed emaciation of the intercostal spaces. The inspiratory excursion appeared normal anteriorly, but posteriorly there was a definite lag at the right base. In the upper left chest anteriorly there was a high-pitched percussion note which was markedly in contrast to that of the opposite chest space. The breath sounds on the left showed a harsh type of breathing throughout. Over the left base, both anteriorly and posteriorly, could be heard a coarse friction rub. Over the right base posteriorly the breath sounds were absent. Heart rate of 88, regular; no murmurs heard. Blood pressure 130 84. No apparent enlargement of the heart. The rest of the physical examination was negative.

Laboratory. *Blood Count.*—Erythrocytes 4,200,000; hemoglobin, 84 per cent; leukocytes, 11,150; polymorphonuclears, 73 per cent; lymphocytes, 22 per cent; mononuclears, 3 per cent; transitionals, 2 per cent.

Urinalysis.—Specific gravity 1.024; alkaline reaction; albumin- and sugar-negative. Microscopic: negative.

x-Ray Examination.—May 18, 1939. An *x*-ray film of the chest reveals a deviation of the trachea to the right in the lower third, and a large elongated area of increased density in the left upper, extending laterally from the mediastinal area. The edges are smoothly rounded. The area of increased density is 9 cm. long and approximately 5 cm. wide, centrally. There is no apparent increase of density in the hilum below this area. There are two areas of increased density on the right, one centrally located in the base, 4 by 6 cm. The edges are feathered. There is a small elongated area of increased penetrability centrally located. There is another area of increased penetrability, irregular in contour, extending laterally from the superior aspect of the right hilum, with no apparent increase in hilum density. The diaphragm is tented, bilaterally, with sharp costophrenic angles. The heart is small. The right apex is clear. There is pleural thickening in the left. There is an accentuation of linear markings on the left, particularly in the downward axis.

x-Ray pictures taken at this most recent examination, January 2, 1940, show the previously mentioned mass in the upper left lung to be considerably enlarged but still well defined and well demarcated. Pleural adhesions not noted in the former films are now present in both bases. The mass in the right lung and the one adjacent to the hilus are likewise enlarged. In neither the pictures taken in May, 1939, nor these most recent pictures is there any evidence of nodulation or emphysema.

Discussion.—It was learned that the explosive used was that of dynamite containing nitroglycerine to approximately 40 per cent. The toxic effects of gases liberated by such explosives in confined spaces are naturally greater than the effects of these same gases in the open air. The two chief gases resulting from such explosions are carbon monoxide and nitrogen dioxide. These deserve momentary consideration.

Nitrogen Dioxide.—Exposure to this type of fume may result in one of three types of reaction. There may be immediate death, or delayed reaction, or no effects whatever. It is not uncommon for a person who has been exposed to nitrogen dioxide to notice no particular effect other than a mild upper respiratory irritation for an hour or two, or even longer. Following this interval of from an hour to ten or twelve hours, there may set in a violent reaction or death. At this time the patient coughs violently, has a sensation of suffocating, and has shortness of breath. The cough is often markedly productive and the spu-

tum may be blood-streaked. If the patient survives this episode, he may recover within a day or two, or pneumonia may complicate the picture. There is rarely any permanent sequela from such an exposure.

Carbon Monoxide.—Carbon monoxide gas is absorbed through the lungs into the blood and affects the hemoglobin of the red blood cells. It thus affects the ability of these cells to carry oxygen from the lungs to the tissues which demand it. Here again death may be instantaneous, or the patient may suffer severe headache, nausea and vomiting, shortness of breath, a feeling of constriction in the chest, and may lose consciousness. If the gassing has been severe, edema of the lungs may be present, and within a few hours or a day or two, pneumonia may develop. However, if the patient does not die, recovery is usually complete. If any permanent sequelae develop, they are usually of the nervous system and appear within a week or two following the accident.

From this discussion of these two gases it is apparent that exposure to neither carbon monoxide nor nitrous dioxide would cause permanent pathological changes within the lung. While this patient had a definite exposure to a combination of noxious gases he, by his very own statements, was only slightly affected by them, and his recovery from this minimal affection was complete.

Silicosis.—In the patient's claim of ill health he believes that in addition to exposure to gases, dusts from mining have affected his lungs. This belief is easily understood, since most laymen have the idea that the dusts from any mining process permanently affect the lung tissue. However, in the occupational history it will be noted that he had very little exposure to silica dust, and that at no time did the *x*-ray pictures reveal the presence of silicosis in the lungs. Therefore, this cause of illness can be definitely ruled out.

On the other hand, the *x*-ray pictures, the history of existing ill health, and the symptoms and signs present indicate a malignancy of the lung tissue. The signs and symptoms resulting from this disease were first noted in the patient at or about the time of his exposure to the fumes from dynamite. Therefore, the onset and development of the cancer must have antedated the period of gas exposure by many months.

Conclusion.—Investigation of this case would rule out any of the occupational factors and would indicate that the patient's disability is due to cancer and that this cancer is non-occupational in origin. There exists, therefore, no temporary disability and no permanent disability arising out of the patient's occupation.

Very truly yours,

Comment on the Presentation of the Above Case.—The above case was chosen from the files of this clinic to illustrate the presentation of

facts, especially the discussion as it was treated by the author. It should be noted that the claim of the patient, his history of exposure, his past and most recent occupational history, the physical examination, and the laboratory findings are all detailed in a manner that the lay or the legal mind can readily understand. Then, the substances alleged to be the cause of this patient's illness are considered in the discussion. The known effects from exposure to these substances are set forth in contrast to the pathology actually found. From all of the presented facts a conclusion of non-occupational origin of the man's disease is made. Such a presentation, with the weighting of all facts, better enables the court to arrive at an equitable decision.

PART II

GASES, SOLVENTS, AND FUMES

Across the pathway to normal health of the industrial worker lies the mysterious shadow of the gases, solvents, and fumes. A criminal may be fingerprinted and photographed so that he *is* known to the police, but he may easily escape recognition by the general public. Similarly, the danger from exposure to certain substances is known to the toxicologist but the profession as a whole has not as yet learned to distinguish the characteristics of such conditions. The lethal effect of carbon monoxide or cyanide has been well publicized, but the insidious damage done by lesser known substances receives little consideration.

General Effects.—It is important that we know which of these substances may be *acutely fatal*, which produce only *temporary effects*, and which may produce *permanent damage*. The very term "gas" conjures up false impressions of the noxiousness of various gases in the minds of most people. In this respect a lesson can be drawn from the first World War. War gases were designed to kill or cripple. How effective were they? The Surgeon General's office reports 70,552 gas casualties with only 1221 deaths, or a rate of 1.73 per cent (American troops). What about the sequelae? In the immediate years following the cessation of fighting, asthma, hay fever, bronchitis, and especially tuberculosis were alleged as resulting from "being gassed." The Veterans' Bureau was deluged with claims. Investigation revealed the vast majority of these cases to be neuroses, malingering, or incorrect diagnosis. Colonel Adelno Gibson,[1] of the Chemical Warfare Service, in a study eighteen years later, states, "These gases produced relatively little after effects." The consensus both here and abroad is that gassing left no large aftermath of tuberculosis, nor did it predispose to tuberculosis.

Effect on the Blood.—Since the solvents and gases have an effect in many instances upon the blood cells, and since anemia of some type is invariably present, do these anemias become chronic and do these noxious substances produce leukemia? Overwhelming evidence points to complete recovery from secondary anemia with few exceptions. Regarding leukemia, only fragmentary evidence exists. Falconer[2] reports a case of lymphatic leukemia four years after an acute exposure to benzene. He *suggests* that leukemia may have developed as an overcompensatory measure for previous destruction of leukopoietic

tissues. Alice Hamilton[1] refers to a report of Martland who noted a blood picture characteristic of myelogenous leukemia in a patient with benzene poisoning. She states that she has seen two such cases herself. It would appear as sane reasoning to deny compensation on any of the anemias if they occur after an interim of non-exposure or if they occur many months or several years after an acute exposure. However, as noted in the chapter on benzol, the recent work of Hunter, Mallory, and others indicates the possibility of latent effects from benzol poisoning. If they are alleged to have resulted from chronic exposure, the only criterion to accept would be the proof of the exposure. The case mistaken for idiopathic aplastic anemia, reported by Askey and referred to in the section on petroleum distillates, illustrates this point.

Effect on the Liver.—In addition to the disturbed blood picture, the acute or chronic assault to the body metabolism by toxic substances vitally affects the liver. Not only does this organ metabolize the carbohydrates, fats, and proteins, maintain a normal blood-sugar level, carry on the diaminization of the amino acids, excrete bile and perform other duties, but it serves to detoxify toxic substances. These may be endogenous or exogenous. It is responsible for the handling of these by-products so that they are to be excreted from the body in a non-toxic or less toxic form. For instance, benzol may be conjugated by the liver with the formation of the organic sulfates, aromatic acids, and alcohol, which are converted into glycuronates. Other noxious agents are detoxified by the liver and passed off in the excreta in a manner as yet not fully understood. However, there is a limit to its capacity to handle enormous amounts or a tolerance to constant, repeated injury. Certain substances in particular appear to have destructive or damaging effects upon the liver. In industry the halogenated compounds such as carbon tetrachloride, the chlorinated naphthalenes, trinitrotoluene, tetrachlorethane, and also cadmium, manganese, arsine, and others are known to affect this organ. It should be realized that the ability of the liver to detoxify these poisons varies with individuals and that some have marked susceptibility to these noxious substances. This variation in susceptibility is seen every day in the routine use of chloroform or avertin.

Effect on the Kidney.—The kidney is often damaged when acute poisoning occurs from certain solvents. It is not uncommon to see uremia in workmen overcome from carbon tetrachloride.

Nature and Concentration of the Contaminant.—In predicting the outcome of a case of industrial poisoning, the nature and concentration of the contaminant, as well as the usual pathology produced, must be taken into consideration. While each of the gases or solvents has its own peculiar reaction, certain general facts may be accepted: (1) Any

substance introduced into the body in excess is harmful. (2) Few, if any, of the gases or solvents are stored in the body. Living in uncontaminated atmospheres for sixteen out of every twenty-four hours results in an interchange with oxygen that ordinarily maintains the blood free from saturation. (3) Short, severe exposures usually result in but short, temporary disability. (4) Constant daily exposure to amounts slightly in excess of the recognized limit, may, and often does, result in chronic illness. (5) Odor as well as irritation to the eyes, nose, throat, or lungs should be ordinarily construed as an atmosphere that is unsafe for continuous exposure. There are a few exceptions to these axioms; but if they are constantly kept in mind, better medicolegal decisions will result.

A stumbling-block to honest diagnosis is the subterfuge that often exists in the manufacturing of these products. Trade names conceal the properties, and frequently the formula is not on the label. Requested information from the manufacturers does not always meet with cooperation. Therefore, if the suspected offending ingredient cannot be determined because of any one of these conditions, it behooves the physician to submit a sample to a testing laboratory. A further difficulty is the fecundity of carbon compounds. They appear to reproduce with ease.

Individual Susceptibility.—In the final estimation of any case, consideration must be given to the individual's make-up. *Obesity, alcoholism, endocrine imbalance,* and personal and home *hygienic factors* may play a part in susceptibility. In this present period of the practice of medicine, allergy is being greatly emphasized. Enthusiastic proponents account for any reaction not specific in its etiology upon this basis. To the author, this is a dangerous procedure. Until allergic reactions to gases or solvents are more definitely established, a physician should be wary of accepting a case on such a basis. Observation during the next few years should clarify the situation.

Classification.—

A. Aliphatic solvents
   1. Sources
      (*a*) Petroleum
      (*b*) Wood distillation
      (*c*) Fermentation
      (*d*) Fats
      (*e*) Synthesis
   2. Toxic effect
      (*a*) Less than aromatic solvents
      (*b*) Hydrocarbon derivatives more toxic

B. Aromatic solvents
   1. Sources
      (*a*) Distillation of coal tar
      (*b*) Distillation of pine tar
      (*c*) Fat
      (*d*) Synthesis
   2. Toxic effect
      (*a*) Greater than aliphatic solvents
      (*b*) Hydrocarbon derivatives more toxic

Symptoms Produced Dependent Upon.—

A. Nature of solvents
   1. Toxic effect greater as their power of solution in fats is greater
   2. Toxic effect less as their solubility in water is greater
   3. Solvent mixtures
B. Severity of exposure
C. Length of exposure
D. Susceptibility of individual
   1. Preexisting organic disease
   2. Nervous instability
   3. Alcohol
E. Specific action of solvent
   1. Action on fats and lipoids, particularly of the central nervous system
   2. Action on specific organs
      (*a*) Methyl alcohol on the optic nerve
      (*b*) Chlorohydrocarbons on the liver
      (*c*) Carbon disulfide on central and peripheral nervous systems
      (*d*) Benzene on the blood-forming organs
      (*e*) Action of certain solvents on the skin, removing fats

The following pages do not attempt a complete discussion of all the solvents. By and large the newer preparations present problems similar to the ones studied. Before this ink is dry, other substances will find their way into industrial use, but it can be expected that they will be evolved from the hydrocarbon compounds and that the reaction in general will be similar to the ones now understood.

REFERENCES

1. Gibson, A.: "Chemical Warfare as Developed during the World War and Postwar Future Development," *Bull. N. Y. Acad. Med.*, 2d series, *13*: 397–421, July, 1937.
2. Falconer, E. H.: "Instance of Lymphatic Leukemia Following Benzol Poisoning," *Am. J. M. Sc.*, *186*: 353–361, Sept., 1933.
3. Hamilton, A.: *Industrial Toxicology*, Harper and Brothers, New York, 1934, p. 163.

CHAPTER IV

BENZENE (Benzol)

This coal-tar derivative should be referred to as *benzol* to distinguish it from the petroleum distillate, *benzine*. Rarely coming in contact with pure benzol, the workman when exposed is subjected to a commercial variety containing also toulene, xylene, olefins, paraffins, and carbon disulfide. Because of the long-recognized dangers of benzol, efforts and legislation have been in effect to suppress its use or to have it properly labeled for easy recognition. Yet, like crime, it continuously reappears under a new guise. Frequently solvents will be labeled "non-hazardous" because they are non-inflammable, or non-explosive, yet they may contain a high percentage of benzol.

The demand for this substance as a solvent of gums, resins, fats, rubber, and alkaloids, and as a fuel creates a field of wide exposure. The dangers of benzol decrease when men are informed of the exposure and adopt proper precautions.

Occupational Hazards.—Workers with airplane dope, makers of aniline dyes, artificial leather, and batteries (dry), bronzers, rubber-cement mixers, compounders of rubber, degreasers, dryecleaners, electroplaters, enamelers, engravers, extractors of oil and fats, lacquerers, lithographers and rotogravers, painters, rubber-tire builders, shellackers, varnishers, vulcanizers, welders, and so on, are liable to this hazard.

Pathology.—An extensive review of the literature reveals a general agreement that chronic benzol exposure produces a disturbed hematopoiesis, but there appears no uniformity of opinion regarding the pattern the blood picture assumes. There exists no definite laboratory basis for a diagnosis of this poisoning, but not because there has been a lack of clinical reports; for since Santesson's description in 1897 of nine cases of purpura haemorrhagica in girls working with benzol-rubber cement, the literature has been replete with various clinical findings in persons exposed to this solvent. Likewise much animal experimentation has been engaged in. Such experimentation tends largely to reduplicate the conditions of acute exposure rather than the chronic. What has been needed is a controlled investigation of a large series of cases in which the degree and length of exposure were known and which entailed a hematological, pathological, and postmortem study. Very recently this has been done by several investigators, and there

is every reason to believe that from this work, and a further study of this type, a more accurate basis for diagnosis will be derived.

Bone-Marrow Reaction.—Before considering the hematological phase, some attention should be given to the actual pathology. The most marked lesions occur in the bone marrow. For years it has been assumed that a medullary aplasia occurred. Cases are on record in which patients with a known exposure to benzol have been denied compensation because they demonstrated hyperplastic marrow, which was considered not typical of benzol poisoning. Some years ago Hamilton,[1] realizing the variations in the pathology of all blood diseases, predicted that further study would alter the viewpoint then held. Her prophecy has been substantiated in the work of Mallory[2]; Thompson, Richter, and Edsall[3]; and Rhoades and Miller.[4]

Recently, Mallory and his co-workers have studied nineteen cases, fourteen of which were completely autopsied, and histological material was obtained from each of the nineteen. In the autopsied cases, material was obtained from several specimens of bone marrow, including the long and flat bones, as well as from the thoracic and abdominal viscera, liver, and spleen. None of these patients had been exposed for less than six months, four being exposed for less than a year; eight from one to four years; and seven from four to twelve years. Excluding two cases of possible leukemia, the remaining number could be divided into two groups—six showing marrows less cellular than normal, and nine revealing hyperplasia. In comparing his findings with the history of each case, Mallory was of the opinion that intensity of exposure was not a determining factor in the bone-marrow reaction, but that duration of exposure was. Cases of less than a year's exposure showed a *hypoplasia*, whereas those with prolonged exposure evidenced a *hyperplasia*, indicating that the initial effects of benzene upon the bone marrow were to depress it but that the prolonged effect was to stimulate it.

On the other hand, from these observations it might be reasoned that since persons react differently to benzene exposure, the occurrence of a hyperplasia enables the worker to continue for many years, whereas the development of an aplasia results in rapid disability or fatality unless the patient is removed from the exposure.

Effect on the Liver.—The chief findings in the liver were the prominence of the Kupffer cells, which were loaded with hemosiderin, and central necrosis, possibly due to terminal infection rather than to the benzene assault. In Mallory's fourteen autopsied cases, the spleen was rarely noted to be enlarged clinically, but was frequently found enlarged at the autopsy table. Increased cellular activity was usually present. Well-defined foci of hematopoiesis were seen even in cases of

minimal general changes. These became more marked and numerous with increasing severity of the lesion. These investigators noted a general parallelism between bone-marrow and splenic hematopoietic activity. In spite of frequent purpura of the skin, mucous membranes, and serous surfaces, they noted no gross brain changes except in one case showing several subarachnoid hemorrhages.

Latent or Delayed Effects.—Since one of the primary functions of this book is to show the *medicolegal* relationship of the disease, the latent or delayed effects of benzol poisoning deserve special consideration. This phase of the disease concerns itself with the leukemias and certain neoplastic changes. There appears to be increasing evidence that this solvent may initiate a pathological alteration of the bone marrow which can slowly progress for months or years after exposure. The sequence of events in the *anemias* due to benzol is comparable in many ways to the changes which occur following exposure to *x*-rays or radium, as reported by Martland[5] in 1931. He observed in a benzol worker marrow typical of pernicious anemia, and in another the blood picture was characteristic of myeloid leukemia. Penati and Vigliani collected ten cases from the literature of leukemia in workers with verified histories of exposure to benzol. Mallory and his co-workers had two cases of leukemia in their series; Erf and Rhoades[6] had one in their group. It is not unreasonable to believe that many more such cases have not been reported.

Regarding *neoplastic* changes, Mallory[4] states:

> Also suggestive to anyone who has studied even superficially cases of the hyperplastic type, are the evidences of what for a better term may be called a neoplastic tendency. The degree of anaplasia, the rapidity of growth as judged by the number of mitotic figures, the development of cells having no counterpart in normal tissue but common to a variety of malignant tumors are phenomena characteristic of neoplasia which the authors have never met heretofore in such a marked degree in non-neoplastic states.

Complications in Establishment of Compensation.—These two possible sequelae just mentioned offer complications in the establishment of compensation. Hunter[7] following a study of eighty-seven cases, stated that the most unexpected observation made in his series was that the first clinical symptoms and signs of chronic exposure may appear *long after exposure has ceased.*

Assuming that these observations are accurate, one can visualize for the future *two types* of cases: One is the person with leukemia or neoplasm who has had adequate benzol exposure, but because of the interval of time existing between exposure and illness, no claim is

made or relationship recognized. The other is the worker with a blood dyscrasia who, months or years after a varying degree of benzol exposure, alleges his condition to be due to that solvent but whose degree of exposure was probably insufficient, and a coincidence is very likely.

Most claims for an occupational illness are made during the period of exposure or shortly afterward. This observation regarding the latent effects of benzol promises to confuse the administration of compensation. Time and adequate evidence from abundant material will be necessary before acceptance or denial of this possibility can be made.

Blood Picture. *Leukopenia Not Diagnostic.*—It has been accepted that the earliest evidence of the effect of benzol exposure is leukopenia. Largely this has been the only test relied upon, and in many plants a routine white-cell count constitutes the basis for permitting the workman to continue his work or removing him from the exposure. From the recent investigations such a practice is questionable and probably unreliable. It seems odd to this writer that although Hamilton warned a number of years ago that dependence should not be placed upon leukopenia for diagnosis and cited a case in which the white cells were 6800 but the red cells only 1,500,000, yet textbooks continue to cite leukopenia as the characteristic finding in benzol poisoning.

Recent investigations by Hunter, at the Massachusetts General Hospital; Greenburg, Mayers, Goldwater, and Smith, of the New York Department of Labor; and Erf and Rhoades, of the Rockefeller Institute, indicate that leukopenia is not a prevalent isolated finding. In Hunter's series, leukopenia was not found without other abnormalities in one of his eighty-nine cases. Greenburg and his co-workers found a deviation of the white count in only 14.5 per cent of 332 workers examined. Of his patients who were daily exposed to benzene vapors, 85 per cent showed a normal white-cell count. Furthermore, there appears no basis for assuming that leukopenia is produced in the presence of a severe anemia and purpura. In Hunter's series a switchboard operator developed a fatal case of anemia, the cause of which was not evident at first. It later developed that she daily cleaned her board with a solvent containing 50 per cent benzene. At the time Hunter saw her, she had marked pallor, generalized ecchymosis, and questionable enlargement of the spleen. The rest of the physical examination was negative. Her blood count was: hemoglobin, 22 per cent; red blood cells, 1,300,000; white blood cells, 7400; polynuclear cells, 48 per cent; lymphocytes, 40 per cent; mononuclears, 7 per cent; basophiles, 1 per cent; and unclassified cells, 4 per cent. Biopsy of the bone marrow showed marked hypoplasia.

Of Hunter's eighty-nine cases, fifty-seven showed multiple deviations from the normal as noted in Table 8. He particularly stresses the fact that, omitting a case of leukemia, the leukocyte count was normal in two and the polynuclear percentage was normal in two, *but in every single instance of fatal poisoning*, the absolute number of polynuclear leukocytes was decreased. Hunter also points out that a decrease of the polynuclear percentage is a better index of *early* poisoning than either a leukopenia or an absolute polynuclear decrease.

TABLE 8.—MULTIPLE DEVIATIONS FROM THE NORMAL*

Erythrocytes	No. of Cases	Leukocytes			Polynuclear Cells Percentage			Absolute Polynuclear Cells Percentage			Erythrocytes Percentage Young Polynuclear or Mar row Cells	
		N	+	−	N	+	−	N	+	−		
Increased	7	3	3	1	4	0	3	4	1	2	4	0
Normal	21	5	15	1	11	2	8	9	10	2	9	4
Decreased	29	10	7	12	14	0	15	4	6	19	7	15
Total	57	18	25	14	29	2	26	17	17	23	20	19

* Hunter, F. J.: *J. Indust. Hyg. and Toxicol.*, 21: 339 (Oct.), 1939.

Macrocytosis.—What appears to be a valuable contribution to the study of benzene not noted in other investigations is the finding of Greenburg and his workers regarding the mean corpuscular volume, mean corpuscular hemoglobin, and the mean corpuscular hemoglobin concentration. In their series of cases they found a distinct tendency towards macrocytosis. The average cell size was greater than 94 cu. μ in 9.3 per cent of the control group, as compared to 46.7 per cent over 94 cu. μ among those exposed to benzene. The average size was less than 90 cu. μ in 73.4 per cent of the control group, as compared to 30.1 per cent of the benzene-exposed group. They had fifty-five cases in one of the plants inspected which showed a white-cell count above 5000. Upon the basis that benzene causes leukopenia, these cases might have been classified as negative for benzene poisoning. Yet 41.8 per cent of them were found to have red corpuscles larger than 94 cu. μ. In only three of the sixteen cases in this plant, with less than 5000 leukocytes, were the erythrocytes normal in number. This indicated to them that with benzene absorption the mean corpuscular volume is definitely increased in a large proportion of cases.

Regarding the *mean corpuscular hemoglobin*, they estimated the hemoglobin concentration by the constant method of Wintrobe rather

than by the usual method calculated in terms of color index. They found that the erythrocytes of benzene workers are likely to contain more than normal amounts of hemoglobin. Calculation of the mean corpuscular hemoglobin concentration shows that, in addition to the greater content of hemoglobin, there is also an increased concentration.

Significant Hematologic Abnormalities.—The incidence of significant blood abnormalities found in the series studied by Greenburg and his co-workers is somewhat differently classified as revealed in Table 9.

TABLE 9.—INCIDENCE OF SIGNIFICANT BLOOD ABNORMALITIES IN MEN EXPOSED TO BENZENE.*

Test	Criteria of Abnormality	Total Tests Made	Abnormal	
			No.	*Percentage*
Erythrocytes	Less than 4.5 million	159	76	47.8
Mean corp. vol.	More than 94 cu. μ	103	48	46.6
Platelets	Less than 100,000	107	35	32.7
Hemoglobin	Less than 13.0 gm. per 100 cc.	235	36	15.3
Leukocytes	Less than 5,000	332	48	14.5

* Greenburg, L., Mayers, M. R., Goldwater, L., and Smith, A. R.: *J. Indust. Hyg. and Toxicol., 21:* 413 (Oct.), 1939.

Erf and Rhoades investigated benzene poisoning in six rotogravure printers, one chemist, one beauty-parlor worker and one rubber-cement worker. Concerning the *leukocytes*, they noted that early, when the bone marrow was hyperplastic, the presence of myelocytes and irritated leukocytes was noted, but later, when the marrow became aplastic, there was a decrease in the white cells.

In fifty-two patients tested for *bleeding time* by Erf and Rhoades, they noted an excessively long bleeding time in only two cases and moderately prolonged bleeding time in only three, although the blood platelets were below 100,000 in thirty-five cases and below 50,000 in twelve others. This group noted no significance in the *sedimentation rate* of men exposed to benzene. Erf and Rhoades made a noteworthy observation when they studied the *free hydrochloric acid content* of the stomach of their nine patients and found it to be present in every one. This finding should have some value in the differential diagnosis of pernicious anemia from benzol poisoning.

Urine-Sulfate Ratio.—It is held by some that a valuable test for the evidence of benzene absorption is to determine the urine-sulfate ratio. This is done by dividing the amount of inorganic sulfates present in the urine by the total sulfates. The specimen should be obtained

while the worker is exposed and not after he has been absent for several days. Thus, if the test is to be of value, it must be done only on those actively engaged; it has no place in the diagnosis of those once removed from the exposure.

One senses from the opinions in various reports that the present status of this test is that it has a confirmatory value but cannot be relied upon specifically as a diagnostic one.

Summary.—A review of the investigations previously mentioned, as well as the work of others not cited in these paragraphs, leads to this conclusion: that chronic benzol exposure brings about a varied clinical and hematological picture, and that anemia, leukopenia, thrombocytopenia, reticulocytosis, high color index, increased fragility of erythrocytes, urobilinuria, and prolonged coagulation time may or may not be present. The bone marrow may be normal, aplastic, hyperplastic, or leukemic. The variation of the findings is due probably to the degree of exposure, length of exposure, and individual susceptibility.

In spite of the varied picture two facts remain evident: (1) that anemia invariably results from exposure and (2) that leukopenia is not a common early finding. Furthermore, if leukopenia is present, other abnormalities of the blood picture are also seen. Such observations should discourage the confinement of diagnosis to a white-cell count alone.

DIAGNOSIS

Signs and Symptoms.—In acute benzol poisoning, the symptoms are due to an irritant and possible destructive effect upon the central nervous system, whereas chronic exposure affects the hematopoietic system.

Fatigue, lethargy, and dryness of the mucosa are the most common findings. These are accompanied eventually by some or all of the following: loss of weight, headache, anorexia, dizziness, insomnia, epistaxis, bleeding gums, nervousness, palpitation, and intestinal colic.

Basis for Diagnosis. *Differential Diagnosis.*—Obviously benzol poisoning must be differentiated from the various anemias, chiefly *pernicious anemia*. This is extremely difficult at times when it is realized that not only does benzol poisoning assume frequently atypical forms, but likewise do the various blood dyscrasias. One cannot place definite reliance upon the high color index of pernicious anemia, for it will occur in benzol poisoning. Until recently it was not known whether achylia was present in benzol poisoning, but the work of Erf and Rhoades would indicate that there is no achylia in this chemical poisoning.

The establishment of a diagnosis depends upon the following points:

1. *History of Exposure, (a) Concentration, (b) Duration.*—A short, abrupt, severe concentration is apt to produce immediate intoxication from which the patient ordinarily recovers quickly. Prolonged exposure to insidious amounts has the more damaging effect, but the actual exposure should be proved.

2. *Analysis of the Atmosphere.*—This must be done under the exact conditions of the alleged exposure.

3. *Analysis of the Exact Solvent and the Conditions of Concentration in Which It Was Allegedly Used.*—It has been previously intimated that solvents change their benzene content from time to time as well as their trade names. In an effort to substantiate or deny exposure, the analysis for the benzene content should be of the substance in use at the time of the alleged exposure, not at the time of the trial. Many employers assume that the chemical content of a solvent they buy is standardized and constant, whereas in reality it frequently is not.

4. *A Complete Blood Count.*—A white blood count alone is insufficient proof for or against the diagnosis of this poisoning. Diminution of the *red cells* and increase in their size are better criteria than dependence upon the reduction of the leukocytes, particularly early in the disease. Regarding the change in the *size* of the red blood cells, one must be sure to rule out pernicious anemia, since they show this phenomenon in that condition as well as in certain other toxic poisonings, as has lately been realized in the blood picture of those exposed to methyl cellulose in the "fused collar" industry. The mean corpuscle volume and the concentrated hemoglobin content are additional confirmatory findings.

5. *Signs and Symptoms.*—The average case should show the variations of these as enumerated. The atypical case will show a clinical picture with no significant laboratory findings, nor will it reveal an abnormal and indicative blood picture without signs or symptoms. Here again the history of exposure is all-important.

6. *Biopsy.*—Biopsy may be resorted to in medicolegal controversy. It must be evaluated with the blood study and the history of exposure. This may likewise be said of autopsy findings.

MEDICOLEGAL ASPECTS

Temporary Disability.—Most patients tend to recover upon removal from exposure. No set time can be given, for this depends upon the recuperative powers of the patient and the severity of his ex-

posure. The majority of patients recover after from a few months to a year.

Permanent Disability.—If there has been unresolved hemorrhage into the brain or involvement of the nervous system, the disability may be *total* or *partial* and should be so rated. This should not be done for at least two years after the initiation of the disability. Rarely does there occur a complete permanent disability, but occasionally there is a partially permanent one. The possibility of delayed or latent disability or fatality has already been discussed.

Illustrative Cases.—The first three of the following cases are taken from an investigation of chronic benzene poisoning by the labor department of New York, the Massachusetts General Hospital, and the Rockefeller Institute for Medical Research.

Case I.—Fatal, exposed for twelve years. Blood picture suggesting aleukemic leukemia; marrow hyperplastic; no necropsy. Male, aged forty-eight, first seen November 1, 1936. Had worked as a matcher of colors for twelve years in a factory producing artificial leather. Doing the same work during the whole period, he had been heavily exposed to benzene fumes. He was referred for an opinion because a recent blood examination had shown an anemia, a white-cell count of 9000 per cu. mm., 51 per cent of polynuclear cells, and a moderate number of myelocytes and normoblasts. Three years previously, however, his blood had been reported as normal.

Present Illness.—The patient had tired easily for the past year. He complained of numbness and cramps in the legs on getting out of bed in the morning. There had been dyspnea on exertion for the past year, but his symptoms were not getting worse. For some years he had occasionally noticed blood on his toothbrush. There was no history of bruises, petechiae, or blood loss. The past history was noncontributory.

Physical Examination.—Patient well developed and nourished but definitely pale. Pupils slightly irregular. There was slight edema of both shins. The knee jerks were active, the vibration sense well felt. The rest of the examination was entirely negative.

Laboratory Examination.—Urine, normal. Hemoglobin, 55 per cent; red blood cells, 4,420,000 per cu. mm.; white blood cells, 4500 per cu. mm.; polynuclears, 13 per cent; lymphocytes, 24 per cent; mononuclears, 2 per cent; eosinophiles, 3 per cent; basophiles, 1 per cent; normoblasts, 6 per cent; young and late myelocytes, 26 per cent; young polynuclears, 16 per cent; unclassified cells, 6 per cent; eosinophilic myelocytes, 1 per cent. There was marked anisocytosis and poikilocytosis, many microcytes, macrocytes, and faded forms, occasional stippling, and a rare myeloblast; platelets, normal. Bleeding

time, three and one-quarter minutes; tourniquet test, negative; icteric index, 8; cell volume, 72 *c*.

Course.—A week after this examination the patient entered the hospital for study and, at this time, the blood was essentially the same except that the white count was 13,500 per cu. mm. It was thought that the patient had early myelogenous leukemia. *x*-Rays of the pelvis and lumbar spine and a blood Hinton test were negative. A bone-marrow biopsy was performed. A diagnosis of benzene poisoning with hyperplastic marrow was made. The patient was sent home to recuperate. A short time later, however, he developed influenza and an abscess of the ear. He entered his local hospital, and anemia became marked (red blood cells, 1,900,000 per cu. mm. with many normoblasts in the blood smear). He became rapidly worse and died on January 14, 1936, six weeks after having been first seen. No autopsy was performed.[7]

In this case there is no question of a history of exposure or of the probable relationship between benzene and the eventual anemia. The important suggestion to be pondered is that benzene has been the *provocative agent* in the production of a leukemia. The three cases in this series, added to the ten already reported in the literature, are sufficient to cause a reconsideration of the toxic effects of benzene.

Case II.—W. D. S., fifty-four, male, a factory worker, entered the hospital complaining of bleeding from the gums. For seven years the patient had operated a spreading-machine in an artificial-leather factory, in the process of which he was constantly exposed to benzene fumes. About a year before entry he first noted occasional bleeding from the gums and a slightly productive cough.

Present Illness.—For about two months there was slight swelling of the ankles and puffiness of the eyelids, associated with polyuria and polydipsia. For six weeks there was constant bloody oozing from the gums.

Physical Examination.—Examination on admission showed pallor, marked hemorrhagic oozing from the gums, and numerous purpuric spots on the skin. The tip of the spleen was barely palpable, and the liver edge extended 6 cm. beneath the costal margin.

Laboratory Examination.—Urine was normal. Red blood count was 1,900,000, with 45 per cent hemoglobin. The white cells numbered 2800 with 16 per cent polymorphonuclears, 52 per cent lymphocytes, 8 per cent monocytes, 4 per cent eosinophiles, and 14 per cent basophiles. There was marked stippling of red cells, occasional nucleated red cells, and rare myeloblasts. Platelets were markedly reduced in numbers. Reticulocytes were 2 per cent. Bleeding time was over twenty minutes, and there was no clot retraction in forty-eight hours. The icterus index was 8 units.

The patient was given five transfusions without improvement. He died on the twenty-eighth hospital day.

Necropsy. (*a*) *Skin.*—Icteric, multiple petechial hemorrhages. Edema of both lower extremities. (*b*) *Lungs.*—Pulmonary edema and hemorrhagic bronchopneumonia with red blood cells and fibrin but relatively few polymorphonuclears. (*c*) *Liver.*—2095 gm. Kupffer cells markedly prominent and filled with hemosiderin. Islets, normal. Within the sinusoids are scattered immature blood cells and moderate numbers of clasmatocytes. (*d*) *Spleen.*—480 gm. Corpuscles in normal numbers but compressed by surrounding collars consisting of dense aggregations of indeterminate stem cells accompanied by a few normoblasts and phagocytes. Splenic cords are widened and contain hemorrhagic extravasation and hemosiderin-laden phagocytes. Lining cells of sinusoids are prominent. Many of the sinusoids are filled with stem cells, erythroblasts, and occasional normoblasts. There are also some clasmatocytes with both red and white cells ingested. Architecture is generally preserved save for a few foci in which masses of stem cells obscure the structure. (*e*) *Bone Marrow.*—Markedly increased cellularity in both rib and femoral marrow. No fat remains, and there is no evidence of fibrosis. Architecture is obliterated by large clusters of stem cells, erythroblasts, and normoblasts, although moderate numbers of myelocytes and polymorphonuclears may also be observed. Megakaryocytes are rare, mitotic figures abundant, and occasional giant cells with monstrous nuclei may be seen. Phagocytes with hemosiderin are abundant.[2]

It is rare that autopsy is performed in alleged benzene poisoning, and, therefore, this case has that significance. Particular attention is called to the findings in the bone marrow of abundant mitotic figures and the occasional giant cells with monstrous nuclei, which serve to support Mallory's contention of neoplastic tendencies in certain instances of benzol poisoning.

Case III.—Male, aged forty-seven. Admitted May 19, 1938, complaining of weakness and ecchymosis. The patient had worked for a rotogravure printing company for three years, and only during the month preceding admission had he noticed increasing weakness and fatigability. He had used solvents containing benzene and was constantly exposed to the fumes. Ten days before admission, subcutaneous ecchymoses appeared on the extremities.

Physical Examination.—The patient was a well-developed and well-nourished male, showing no evidence of recent ecchymoses or purpura. The oral mucous membranes appeared normal, and the liver extended two fingerbreadths below the right costal margin. There were no other pertinent findings.

Laboratory Examination.—Erythrocytes, 2,700,000; hemoglobin, 72 per cent; leukocytes, 2450; platelets, 50,000; reticulocytes, 6.2 per cent. Differential count: polymorphonuclears, 70 per cent; eosinophiles, 2 per cent; lymphocytes, 20 per cent; monocytes, 8 per cent. Fragility, normal. Icterus index, 8. Fasting gastric contents contained free hydrochloric acid. Urea clearance 135 120 per cent of normal. Urobilinogen excretion: fecal, 410 mg. and 336 mg. excreted daily during two periods; urine, 3.8 mg. and 1.1 mg. daily for two periods. A sternal bone-marrow puncture was done, and the marrow was found to be normal. Supravital differential count of the living marrow cells: neutrophilic myelocytes, 22 per cent; myeloblasts, 2 per cent; polymorphonuclears, 21 per cent; normoblasts, 45 per cent; erythroblasts, 10 per cent. Wassermann test, negative.

Course.—Uneventful. The patient received liver extract and thiamine intramuscularly for three weeks in the hospital and three months in the Out-Patient Department.

Laboratory Examination (four months after admission).—Erythrocytes, 3,830,000; hemoglobin, 95 per cent; leukocytes, 7700; platelets, 256,000; reticulocytes, 4.8 per cent. Differental count: polymorphonuclears, 52 per cent; eosinophiles, 4 per cent; lymphocytes, 38 per cent; and monocytes, 6 per cent. Serum bilirubin, 0.85 mg. per cent. Urobilinogen excretion: fecal, 191 mg. and 192 mg. excreted daily during two periods; urine, 0.8 mg. and 0.6 mg. daily during two periods. The patient recovered fully and was followed over a period of one year with no recurrence of symptoms.[6]

This case illustrates the complete manner of investigation from the laboratory standpoint, the treatment, and the eventual recovery.

Case IV.—After working in a benzene tank for about twenty minutes a workman joined his fellow workers at lunch, complaining of dizziness and severe headache. One hour after returning to his work, he was found unconscious. He died two hours after being removed.

This tank had but recently contained a composition of 80 per cent benzene and 15 per cent toluene. The patient's history of previous good health was established. The autopsy showed scattered hemorrhages in the brain, pleura, and intestinal mucosa. The blood was very dark and fluid. The death was adjudged compensable.

Case V.—A migrant to California had been engaged for two weeks in an airplane plant spraying a solution containing 30 per cent benzene. One morning he suddenly fell to the floor in convulsive seizures, frothed at the mouth, and was taken to a nearby physician. The patient denied ever having had similar attacks. The physician diagnosed the case as epilepsy and stated that probably the exposure to benzene

had provoked the attack. The workman then filed for compensation for a disease resulting from his employment.

It was only after some weeks of difficult investigation that the workman's former family physician in Texas was contacted. He provided proof that this man had been subject to epilepsy for years. The case illustrates: (1) the idle remark of a physician without due consideration for the import of his statement, and (2) the fact that epilepsy is common to all walks of life and is not the result of an occupation. While a single attack may be precipitated by a complementary factor arising out of a physical or chemical exposure, it would be absurd to assume that such exposure initiated the syndrome. If, in this case, the occupational exposure brought about the attack, the temporary disability was about one hour.

Case VI.—A twenty-eight-year-old Negro had been employed for three years in a paint and lacquer plant where there existed a moderate exposure to both lead and benzene. He presented symptoms of nausea and vomiting, abdominal cramps, pains in the back of the legs, and weakness. The community physician diagnosed his condition as due to benzene, and he was on compensation for eight weeks. Subsequent examination ruled out any evidence of abnormal lead in the excreta; the blood examination showed 3,100,000 red blood cells, 55 per cent hemoglobin, 9200 white blood cells (with 82 per cent polynuclears). The Wassermann test was negative. In view of the then current opinion that leukopenia had to be present to substantiate a diagnosis of benzene poisoning, he had been subjected, fortunately, to other tests. Examination of a slide after it had been sealed for twenty-four hours revealed sickle cells.

Only persistent effort revealed the actual cause of this patient's condition. The patient was denied further disability compensation.

Many more cases could be cited where physicians have accepted cases as compensable due to benzene poisoning simply upon the patient's history of exposure, without laboratory investigation. Dyspnea in a fifty-five-year-old electroplater who actually had cardiac failure, or chlorosis in a young girl, is an instance of this.

TREATMENT

Prophylaxis. *Frequent Physical and Laboratory Examinations.*—Constant vigilance by blood examinations and *air analysis* should be the established order wherever benzene constitutes a hazard. The routine white-cell count commonly resorted to should be supplanted by a *complete blood count*, for reasons emphasized in the preceding paragraphs. Only those in fine physical condition should be placed

Fig. 1

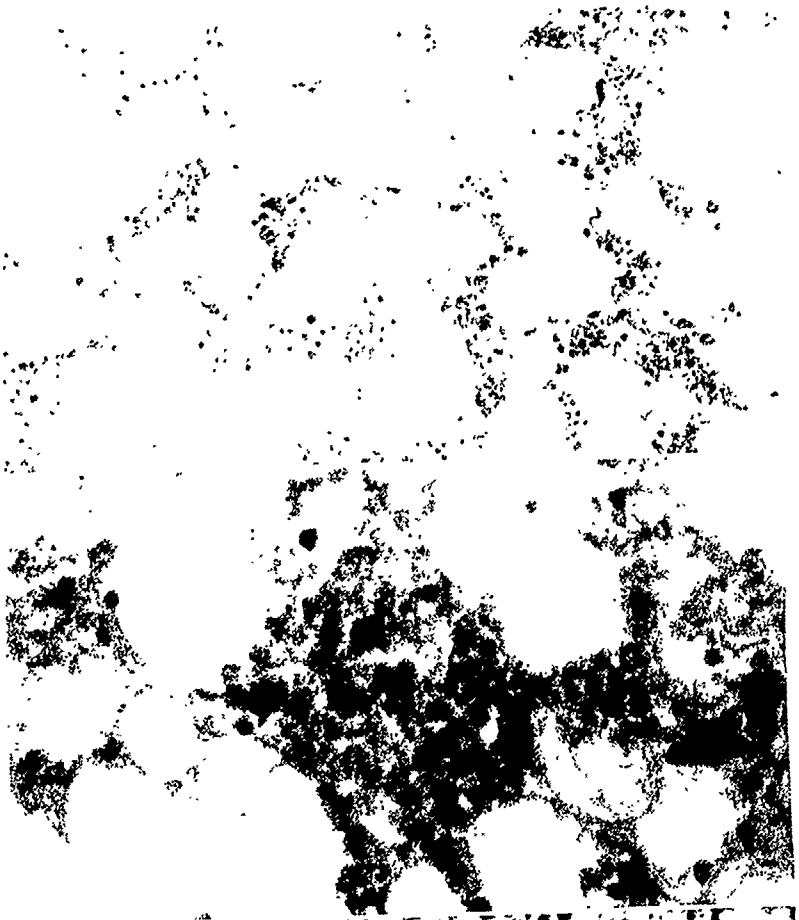

Fig. 2

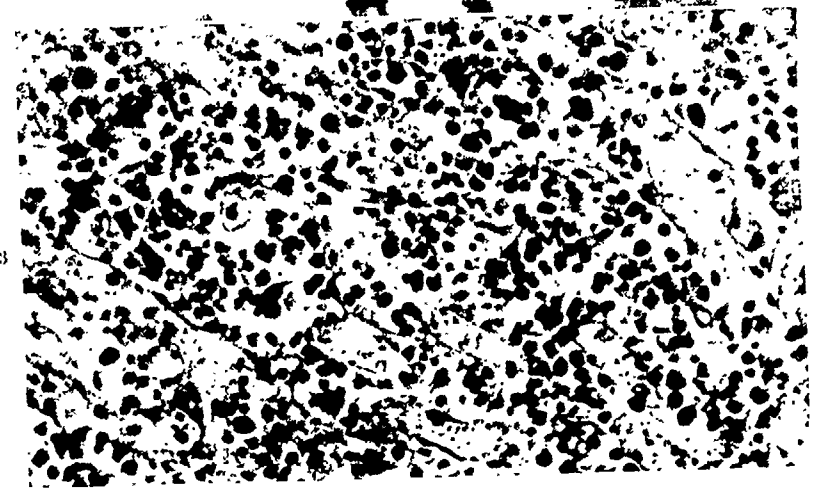

Fig. 3

where a benzene hazard may arise, and frequently physical, as well as laboratory, examinations are indicated.

Men should be *rotated*, and upon perceiving the least variation from the normal, the worker should be removed from his exposure. Likewise, workers should be instructed to report for examination upon noting *bleeding* from the nose or gums or other mucous membranes, or if unaccountable subcutaneous hemorrhages or discolorations are noted. Safety engineers have done an excellent job of preventing accidents by educating the employees regarding the dangers which exist in certain types of employment. It is high time for a similar educational campaign to be conducted in the various occupational diseases. Many serious cases of poisoning from benzene and other noxious agents could be prevented if the employees themselves knew, or were informed, of certain early signs or symptoms. To find a case of far-advanced anemia in one who had for some time noted bleeding and certain symptoms of general malaise, denotes not ignorance on his or her part, but a failure of education or instruction.

Proper Ventilation.—Obviously, proper ventilation is extremely important. Since benzene fumes tend to form pockets and are very diffusible and heavier than air, the ventilation should be general and from the floor by means of suction.

Dermatitis.—Dermatitis is prevented by the rubbing into the skin of olive oil or other animal or vegetable fat, or by the use of a wax ointment prior to handling the substance. Rubber gloves may be used but do not ordinarily withstand the action of benzene, so synthetic rubber, which resists the solvent action much better, should be substituted.

Anemia. *Blood Transfusions.*—In those workers who have developed anemias of varying degrees together with other symptoms of benzene poisoning, the most valuable form of therapy is blood transfusions repeated frequently if necessary.

In attempting to carry out this type of therapy, Gray, Greenfield,

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Fig. 1.—*Bone Marrow (Vertebral)*: There is pronounced hypoplasia with replacement of marrow substance by fat. Most of the sinusoids are collapsed. Those which remain patent contain only small clusters of hematopoietic cells, the predominant elements being non-nucleated erythrocytes. (200 ×) (Mallory: *J. Indust. Hyg. and Toxicol., 21:* [Oct.] 1939.)

Fig. 2.—*Bone Marrow (Vertebral)*: A higher power view from the same section as Figure 1. A group of sinusoids distended with erythrocytes is seen. Comparatively few nucleated elements are evident, and these are almost all normoblasts. (500 ×) (Mallory: *J. Indust. Hyg. and Toxicol., 21:* [Oct.], 1939.)

Fig. 3.—*Bone Marrow (Vertebral)*: The sinusoids are all widely dilated and filled with hematopoietic elements with normoblasts, erythroblasts, and stem cells predominating. Only a few giant cells are seen. The marrow fat has completely disappeared. (300 ×) (Mallory: *J. Indust. Hyg. and Toxicol., 21:* [Oct.], 1939.)

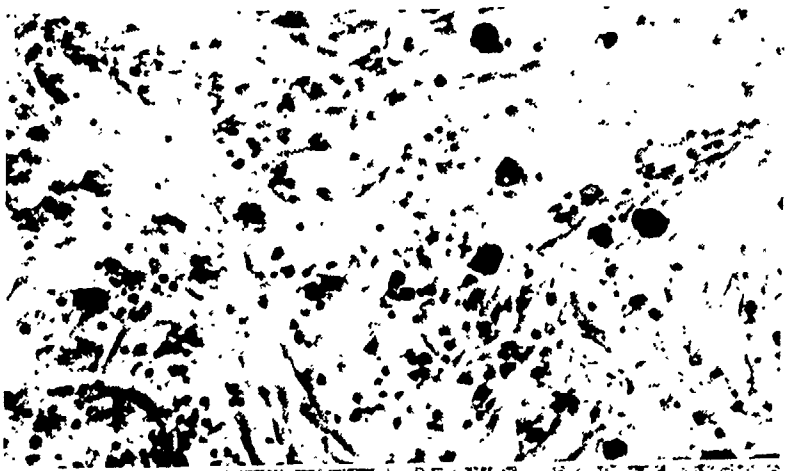


Fig. 4

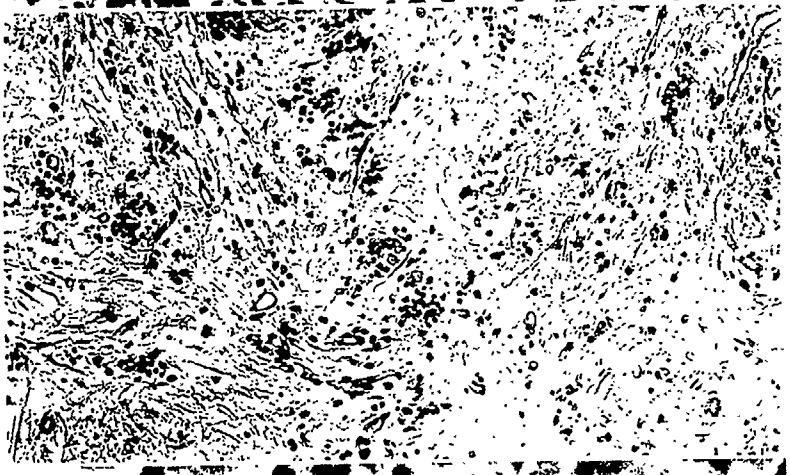


Fig. 5

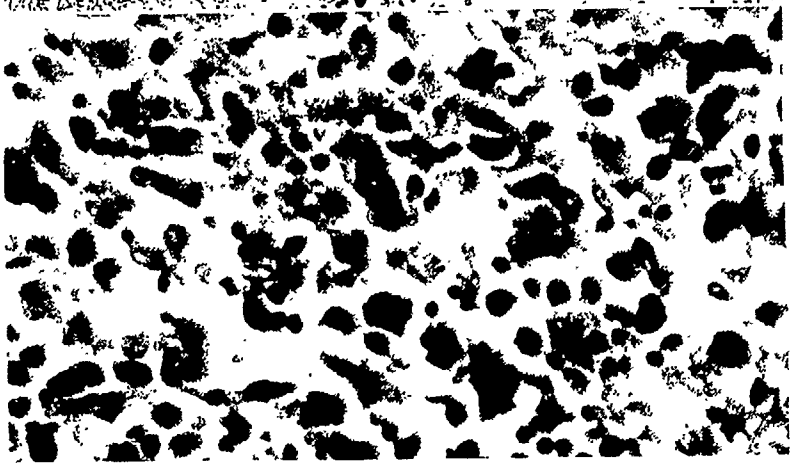


Fig. 6

and Lederer[7] were confronted with a case of autohemagglutination, although this patient had had three previous transfusions. They point out that autohemagglutination is the interaction of the agglutinin of the serum with the agglutinogen of the red blood cell and can occur only at a temperature below that of the body. They eliminated the difficulty by heating the patient's serum and cells to body temperature.

Liver Extract and Iron.—Since the anemia is frequently of the macrocytic hyperchromic type, liver extract intramuscularly in large dosages (15 to 30 units daily for four days, then every two or three days), or ventriculin orally (15 to 30 gm. daily), sometimes both, should be tried. If the anemia appears to be of a secondary type, the use of iron in the form of ferrous sulfate (5 grains; 0.32 gm.), two or three times a day, is advised. Leukopenia indicates the trial of pentnucleotides intramuscularly (10 to 40 cc. daily), although marked improvement under this form of therapy has not usually been demonstrated in these cases.

Roentgen Rays.—For further stimulation of the blood-forming organs, the use of roentgen therapy to the long bones and spleen and bone-marrow extracts has been suggested. Others have used ascorbic acid in moderately large doses with reported success.

Diet.—The diet should contain nourishing food with an excess of animal fats and a high calcium content. Calcium preparations may be given orally as supplements to the diet. Intravenous infusions of from 1000 to 3000 cc. daily of from 5 to 10 per cent dextrose in saline are of value. Good results from the use of ascorbic acid in daily doses of from 200 to 400 mg. intravenously and orally, until a normal vitamin C level is attained, have been claimed.[19]

Mouth, Nose, Throat, and Skin Hygiene.—Because these patients frequently die of a terminal infection, particular attention should be paid to mouth, nose, throat, and skin hygiene.

Fig. 4.—*Bone Marrow (Vertebral)*: Fat is no longer present. There is early fibrosis with thin strands of fibrous tissue. The sinusoids are all dilated but some are filled with normoblasts and scattered stem cells and others only with non-nucleated erythrocytes. The large dark-staining elements are hemosiderin-laden phagocytes. (300 ×) (Mallory: *J. Indust. Hyg. and Toxicol.*, 21: Oct., 1939.)

Fig. 5.—*Bone Marrow (Vertebral)*: The marrow shows dense whorl-like fibrosis with no fat and only a few persistent distorted sinusoids. These are filled with small islands of regenerative hematopoiesis among which normoblasts predominate. (200 ×) (*Arch. Path.*, 25: 322, 1938.)

Fig. 6.—*Bone Marrow (Femoral)*: Fat is completely replaced by cellular marrow. The wide range of polycellularity and the resemblance to Hodgkin's sarcoma are apparent. There is an admixture of stem cells, phagocytes, normoblasts, and multinucleated giant cells. Only a rare granulocyte can be detected. A background of fine fibrous stroma may be observed. (750 ×. Mallory: *J. Indust. Hyg. and Toxicol.*, 21: Oct., 1939.)

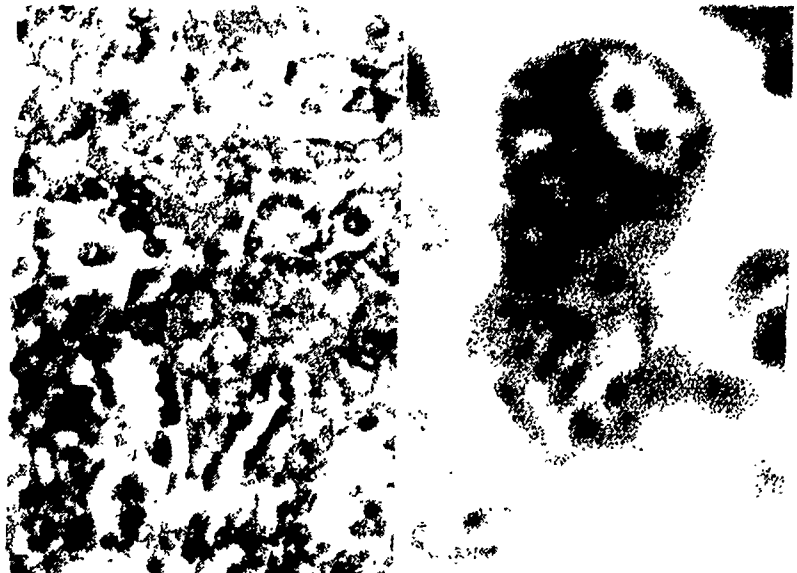


Fig. 7

Fig. 8

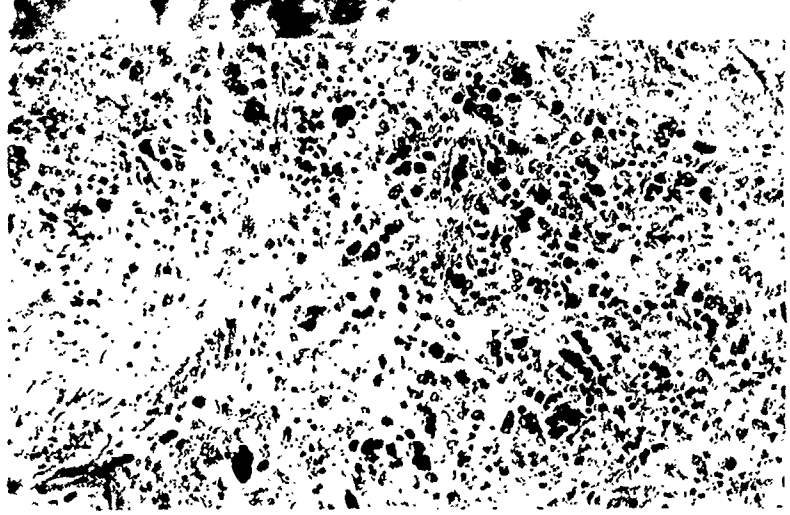


Fig. 9

Fig. 7.—*Spleen:* Pulp lymphocytes have almost completely disappeared. Billroth's cords exhibit marked hemorrhagic extravasation and contain only a few scattered lymphoid elements. Sinusoids are small, but littoral cells protrude prominently into their lumens. (500 ×) (Mallory: *J. Indust. Hyg. and Toxicol., 21:* [Oct.], 1939.)

Fig. 8.—*Spleen:* A multinucleated giant cell is shown almost completely filling a splenic sinusoid. Stem cells and normoblasts may be seen in immediate propinquity. (1000 ×) (*Arch. Path., 25:* 324, 1938.)

Fig. 9.—*Spleen:* A section demonstrating the resemblance to Hodgkin's disease. There is dense fibrosis and apparent loss of architecture although a few distorted sinusoids filled with stem cells may be seen. Two arterioles are evident as the sole remnants of sclerosed malpighian corpuscles. In the scarred stroma are a variety of cells among which multinucleated giant cells are particularly noteworthy. The small dark-staining round cells which simulate lymphocytes at this magnification are actually normoblasts. (300 ×) (Mallory: *J. Indust. Hyg. and Toxicol., 21:* [Oct.], 1939.)

Fig. 10

Fig. 11

Fig. 10.—*Lymph Nodes:* Lymphoid cords are shrunken and irregular in appearance. The total lymphocytic content is diminished. Sinuses are widely dilated and filled with interlacing strands of spindle-shaped phagocytes. (×250. ×½. (Mallory. *J. Indust. Hyg. and Toxicol.*, 21: Oct., 1939.)

Fig. 11.—*Lymph Nodes:* A section through the cortex shows apparent obliteration of the subcapsular sinus and invasion of the capsule. Radial sinuses are compressed and distorted as the result of hyperplasia of the pulp. The latter contains, in addition to densely packed lymphocytes, large numbers of all varieties of hematopoietic cells. Multinucleated giant cells may be readily identified. (×300. ×½. (Reproduced through the courtesy of Dr. Ellis Kellert, Ellis Hospital Laboratory, Schenectady, New York.)

Fig. 12

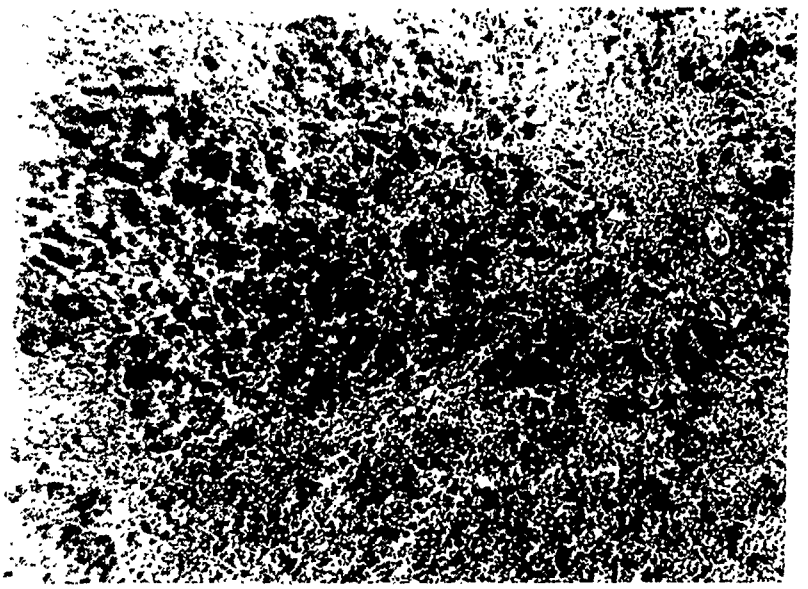


Fig. 13

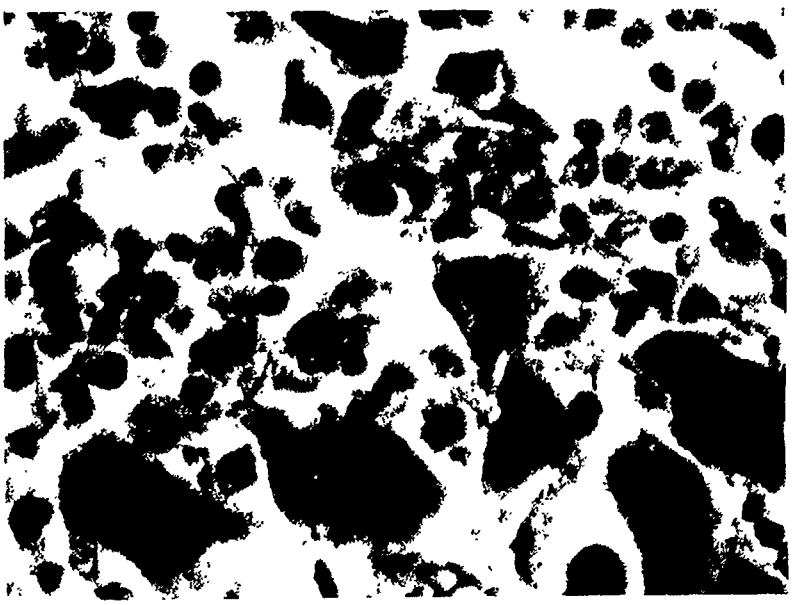
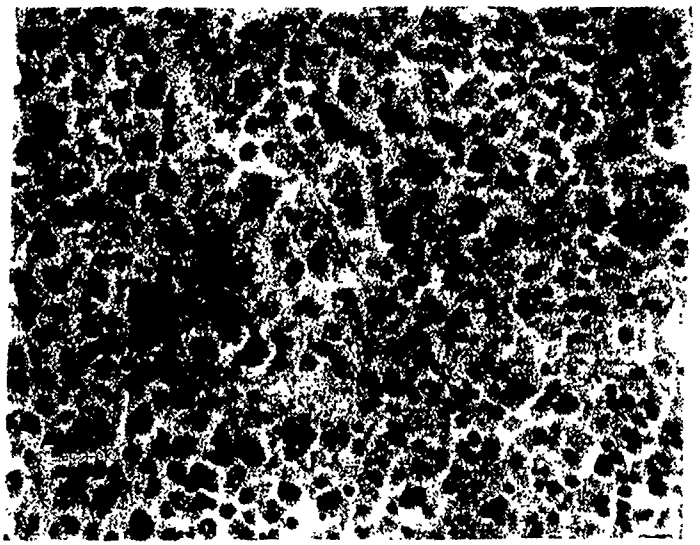


Fig. 12.—*Liver.* The illustration represents the edge of the 4 cm. tumor nodule noted in the gross. Broad sheets of leukemic cells may be seen invading and destroying liver parenchyma. (150 ×. (Mallory: *J. Indust. Hyg. and Toxicol.*, 21, Oct., 1939.)

Fig. 13.—*Liver.* A higher power view of the section shown in Fig. 12. Invasion beyond the confines of the liver sinusoids by leukemic cells may be observed. These cells are almost all myelocytes although identification is not possible in the illustration. Among them, however, small numbers of normoblasts may be identified. Parenchymatous cords show obvious disruption. (600 ×. (Mallory: *J. Indust. Hyg. and Toxicol.*, 21: Oct., 1939.)

Fig. 14.



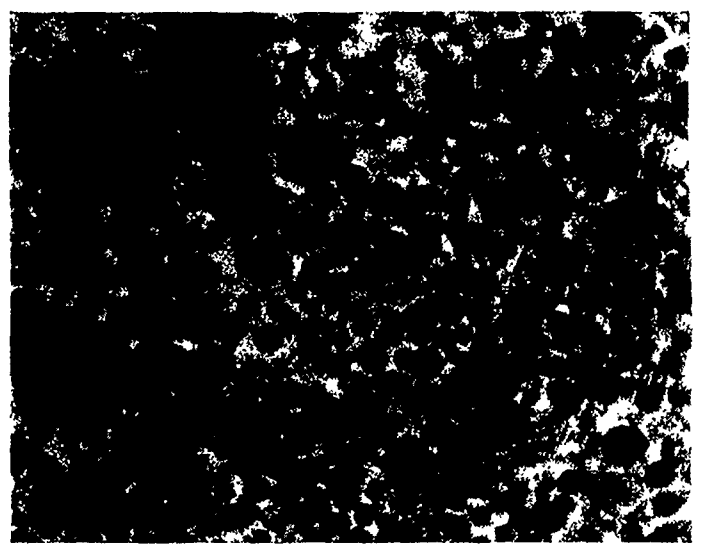
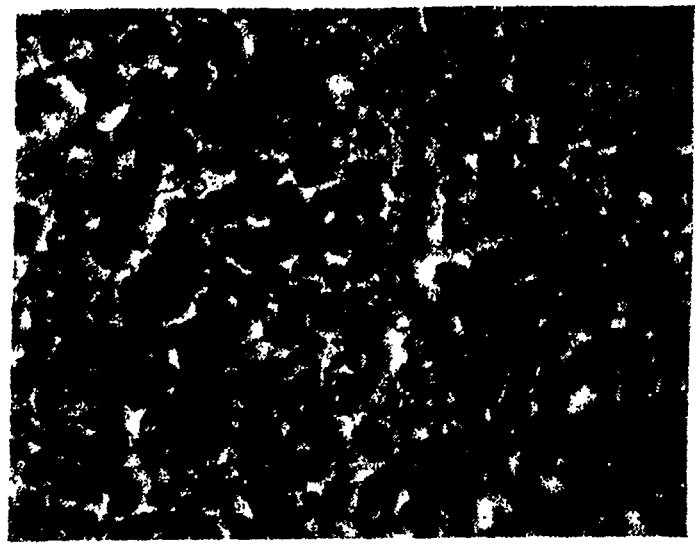


Fig. 15.

Fig. 14.—*Bone Marrow* (*Femoral*): Sinusoidal margins are obscured. There is marked cellularity with erythrogenic elements predominating. Normoblasts and erythroblasts are readily identified. There are also moderate numbers of phagocytes and megakaryocyte-like cells, one of which contains a mitotic figure. (300 ×. (Mallory: *J. Indust. Hyg. and Toxicol.*, 21: Oct., 1939.)

Fig. 15.—*Spleen*: Architecture is partially obscured by a marked accumulation of stem cells and normoblasts. These appear to be (for the most part) within barely visible distorted sinusoids. A large multipolar mitotic figure is evident. (600 ×.) (Mallory: *J. Indust. Hyg. and Toxicol.*, 21: Oct., 1939.)

Fig. 16.



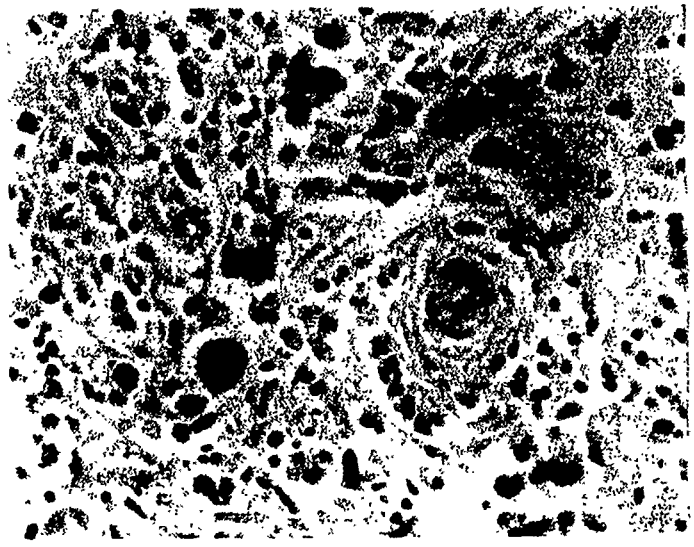


Fig. 17.

Fig. 16. —*Spleen:* Large numbers of stem cells fill the sinusoids and appear also in the intervening pulp. The cells are arranged in irregular clusters. Nuclear vesicularity and relative lack of differentiation are apparent. 1000 ×. (Mallory: *J. Indust. Hyg. and Toxicol.*, 21: Oct., 1939.)

Fig. 17.—Spleen: A sclerotic malpighian corpuscle is demonstrated. Only the central arteriole is recognizable, the lymphoid substance of both corpuscle and parenchyma having been replaced by fibrous tissue. In the region of the arteriole the collagen is concentrically arranged, and a few polymorphonuclears and residual lymphocytes are seen. Elsewhere there are scattered normoblasts, stem cells, plasmatocytes and multinucleated giant cells. 1000 ×. (Mallory: *J. Indust. Hyg. and Toxicol.*, 21: Oct., 1939.)

Fig. 18.

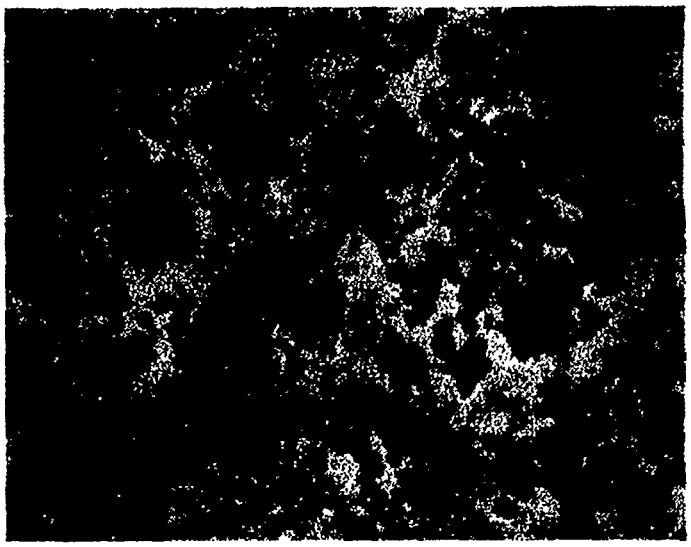


Fig. 19.

Fig. 18.—*Liver:* The sinusoids contain clusters of hematopoietic cells. A group of stem cells and a multinucleated cell indistinguishable from a megakaryocyte are seen. (750 ×) (Mallory: *J. Indust. Hyg. and Toxicol.*, 21: Oct., 1939. Williams and Wilkins Co.)

Fig. 19.—*Lymph Node:* A higher power view from the section shown in Fig. 11. Large numbers of multinucleated giant cells may be seen in the midst of a relatively fibrous stroma. Some of these cells are indistinguishable from megakaryocytes. One contains a large mitotic figure. (500 ×) (Reproduced through the courtesy of Dr. Ellis Kellert, Ellis Hospital Laboratory, Schenectady, New York.)

REFERENCES

1. Hamilton, A.: *Industrial Toxicology*, Harper and Brothers, New York, 1934.
2. Mallory, T. B., Gall, E. A., and Brickley, W. J.: "Chronic Exposure to Benzene (Benzol). III. The Pathologic Results," *J. Industr. Hyg. and Toxicol.*, *21*: 355–377 (Oct.), 1939.
3. Thompson, W. P., Richter, M. N., and Edsall, K. S.: "Analysis of So-Called Aplastic Anemia," *Am. J. M. Sc.*, *187*: 77–88 (Jan.), 1934.
4. Rhoades, C. P., and Miller, D. K.: "Histology of Bone Marrow in Aplastic Anemia," *Arch. Path.*, *26*: 648–663 (Sept.), 1938.
5. Martland, H. S.: "Occurrence of Malignancy in Radioactive Persons: General review of data gathered in study of radium dial painters, with special reference to occurrence of osteogenic sarcoma and inter-relationship of certain blood diseases," *Am. J. Cancer*, *15*: 2435–2516 (Oct.), 1931.
6. Erf, L. A., and Rhoades, C. P.: "The Hematological Effects of Benzene (Benzol) Poisoning," *J. Industr. Hyg. and Toxicol.*, *21*: 421–435 (Oct.), 1939.
7. Hunter, F. T.: "Chronic Exposure to Benzene (Benzol). II. The Clinical Effects," *J. Industr. Hyg. and Toxicol.*, *21*: 331–354 (Oct.), 1939.
8. Greenburg, L., Mayers, M. R., Goldwater, L., and Smith, A. R.: "Benzene (Benzol) Poisoning in the Rotogravure Printing Industry in New York City," *J. Industr. Hyg. and Toxicol.*, *21*: 395–420 (Oct.), 1939.
9. Gray, L., Greenfield, L., and Lederer, M.: "Benzene Poisoning," *J.A.M.A.*, *114* (April 6), 1940.
10. Meyer, A.: Communication to the Editor under "Queries and Minor Notes," *J.A.M.A.*, *108*: 911 (March 13), 1937.

CHAPTER V

THE PETROLEUM HYDROCARBONS

THE petroleum distillates are chiefly gasoline, naphtha, petroleum ether, and benzine. Those whose boiling points range from 30° to 90° C. are used as motor fuels, while the solvents range from 70° to 150° C. Above 270° C. occur heavy oils, vaseline, and paraffin.

Occupational Hazards.—Automobile mechanics, chauffeurs, degreasers, drycleaners, buffers, and housewives are liable to this hazard.

DIAGNOSIS

Signs and Symptoms.—Headache, vertigo, nausea, vomiting, drowsiness, apathy, mild intoxication indicated by unsteady gait and giddiness (naphtha jag), irritation of the skin, visual disturbances, and sometimes twitching of the muscles are symptoms in less severe cases. In the severely acute cases, clonic spasm, convulsions, and death have ensued.

Entering the body, usually through the lungs, the fumes from these substances have an anesthetic action affecting the central nervous system. Instances of ingestion of gasoline or kerosene have been reported by Nunn and Martin and others. St. Leger,[1] Schneider,[2] Battley,[3] and Johnstone[4] have reported instances of gasoline entering the lungs in liquid form. Chronic exposure may cause a loss in weight, tachycardia, and secondary anemia. A slight leukocytosis is not unusual.

Considerations in Diagnosis. *Differential Diagnosis.*—Disease of the heart, lungs, liver, kidneys, and the blood-forming organs have been attributed to the toxic effects of the petroleum products without, as yet, adequate proof. While any of the symptoms mentioned above may occur, there is a need for more abundant correlation between the manner of exposure, the nature of the substance, and the resulting pathology. When an employee working with these distillates develops primary or secondary *anemia* or *leukemia*, it does not follow that the cause resides in his work.

Exposure to Combination of Products.—Most petroleums are relatively non-poisonous. Straight-run gasolines are relatively safe. Cracking of gasoline as done commonly in California results in unsaturated compounds which are more toxic than the saturated. Furthermore, other solvents are sometimes added to what appears to

be a straight distillate. This suggests the possibility that whenever disease does arise from exposure to these petroleum products it is because of an unrecognized combination existing. This is illustrated by the case Askey[4] reports. When seen the man had purpura haemorrhagica and profound anemia with leukopenia. Believing it to be an idiopathic aplastic anemia at first, Askey subsequently considered the possibility of benzene poisoning. The patient was a painter occupied in washing paint off metal plates in a bath of gasoline. This discovery did not solve the problem, but Askey later was informed that California gasoline may contain from 12 per cent to 20 per cent benzene (benzol).

Variety of Findings.—Experimental work has not arrived at a conclusive unanimous opinion. Papers from Russia state that it is possible to produce leukopenia in animals with petroleum benzine if enormous doses are used. Granulopenia was not marked. They report a decrease in the red blood cells and hemoglobin early in the exposure, but with subsequent return to the normal occurring. Their work would indicate an increase of nervous diseases and secondary anemia, but no nutritional disturbance in those exposed to these substances. On the other hand, Hayhurst[6] conducted observations on a series of patients subjected to chronic exposure. He states that disability began from three weeks to several years after exposure began. He lists the symptoms as headache, dizziness, loss of appetite, dyspepsia, restlessness, insomnia, paresthesias, pains in the back, legs, and heart region, weakness and dyspnea. Objectively they revealed from a 10 to 50 per cent loss of weight, tachycardia, secondary anemia with leukocytosis, mental depression, stupor, twitching, and tremors.

Gasoline as Hazard. *Among Gasoline-Station Attendants.*—Frequently gasoline-station attendants confront the industrial physician alleging an illness due to daily contact with the fumes. The weight of evidence is against the validity of their claims. Few such cases are reported in the literature, and when they occur analysis of the facts leads one to suspect *carbon monoxide* (from within the garage) and not the gasoline as the noxious agent. Furthermore, the paucity of these claims in comparison with the thousands of gasoline-pumpers throughout the country who work at this trade for years argues against its likelihood. Any existing illness is probably coincidental. On the other hand, men exposed to large amounts of the concentrated fumes, especially with an admixture of oxygen, have been overcome or have died.

Among Workers with Gasoline Engines.—Johnson[7] reports the cases of forty-two men working with gasoline engines in a tunnel who were overcome by these fumes. Two were completely asphyxiated.

remaining unconscious for twenty minutes. On regaining consciousness, they could move their limbs but could not speak. They complained later of frontal headaches, but no nausea or vomiting. The following day they showed no effects. The day following the exposure, sixteen of the other workers were affected. On another day, eighteen were affected. The blood contained no CO, and the air in the tunnel contained no CO.

Among Those Using Gasoline for Cleaning.—The United States Public Health Service examined a group of twenty-two workers operating coupon-canceling machines who were exposed constantly to fumes of gasoline used in cleaning stamps and belts. Their dispensary-attendance rate for sickness was higher than a controlled group. When perfumed kerosene was substituted, the sickness rate was lowered and their output increased. It is to be suspected that the ventilation was poor.

Tetraethyl Lead as Hazard. *Toxicity.*—The addition of tetraethyl lead to gasoline results in a noxious substance, the hazard of which is confined chiefly to those engaged in blending or producing this type of motor fuel. In 1924 a number of deaths occurred among workmen at plants where this fuel is manufactured. As a result, a special committee investigated the toxicology of tetraethyl lead and concluded that:

1. Tetraethyl lead constitutes no undue hazard to the garage worker, gasoline-pumper, or the general public.
2. A relatively small industrial group is actually exposed to the hazard. Of this group, the chief ones are those who produce or blend this substance or those who come in contact with its products of decomposition, such as cleaners of storage tanks or railroad tank cars.
3. Minute amounts spilled upon the skin in handling the liquid produce no ill effects since the quantity of this substance per gallon of gasoline is so low as to be toxicologically insignificant.
4. The preventive problem can be reduced to the minimum by requiring tetraethyl lead to be colored by a dye to distinguish it from other types of gasoline and by having it properly labeled wherever it is sold, pumped, or stored.

It is not likely that any of us will ever be confronted with a case of tetraethyl lead intoxication occurring in one engaged in its production. But it is not unlikely that any physician may have occasion to diagnose and treat a patient who has been exposed to the decomposition products of tetraethyl lead, and therefore a brief description of the pathology and symptoms is justified.

Pathology.—The pathological changes which occur are widespread but are not specific of this poisoning. Congestion of all the viscera is common, as well as hemorrhage into all parts of the body, although hemorrhage into the gastro-intestinal tract has not been noted in man. In the white matter of the brain occurs the greatest amount of hemorrhage, together with engorgement of the cerebral vessels. The next site to be most affected is the lung, where hemorrhage, congestion, and edema are found. Degenerative changes in the liver, kidneys, heart, spleen, and pancreas have been reported.

Symptoms and Signs.—These vary with the degree of exposure. They appear within a short time in one severely exposed, while in the one who has received a small dose symptoms are usually delayed. This, according to Machle,[7] is the result of two factors: (1) "The decomposition of lead within the body with the liberation of water-soluble lead compounds; and (2) the delayed absorption from either the pulmonary epithelium or the skin."

(a) *Prodromal Period.*—Investigators of this type of intoxication state that there is a prodromal period of from eighteen hours to eight days prior to the onset of real symptoms, varying inversely with the magnitude of the exposure and the severity of the poisoning. The victim appears to become increasingly nervous; sleep is difficult and disturbed by terrifying dreams. Apprehension and anxiety progress into states of excitement accompanied by headaches and vertigo. Occasionally the patient becomes depressed and evidences delusions of various types. Generalized weakness and fatigue are common complaints.

Physical Examination.—Upon examination the reflexes are hyperactive, although those reflexes not normally present are not seen. The eyegrounds reveal no abnormal findings, although visual weakness may be complained of because of weakness of the extrinsic muscles of the eye. Tremor, especially of the upper extremities, is noted in the majority of the severely exposed. The heart rate is slow and the systolic and diastolic blood pressure is lowered. Laboratory studies yield little diagnostic significance.

Complications, Sequelae, and Convalescence.—What is the end-result of the non-fatal cases of tetraethyl lead intoxication? Machle[7] studied a series of cases over a long period of time. He states:

In mild and moderately severe cases in which there were brief periods of delirium no complications developed. As a consequence of circulatory weakness and exhaustion in severe or fulminating cases a variety of complications may conceivably develop, such as acute cardiac dilatations, cerebral edema, pulmonary edema or similar

lesions associated with terminal states. No complications occurred in the present series, however.

Sequelae have not been seen. Convalescence may be protracted with symptoms persisting as long as from eight to ten weeks, but no residual symptoms or evidences of damage have been observed. A group of fourteen patients not included in this series have been under medical observation at monthly intervals continuously since the time of intoxication in 1925 and have not given any evidence of subnormality, physical alteration or increased susceptibility to intoxication. They have been continuously at work in the manufacturing operation, the majority of them having been in supervisory positions during the past two years where the exposure is often greater than that experienced by day workmen. Such evidence, plus the absence of focal brain lesions at necropsy, indicates the reversible nature of the injury from tetraethyl lead and reduces the likelihood of damage from cumulative subclinical exposures.

MEDICOLEGAL ASPECTS

Acute cases are not common in industry. Housewives, cleaning with naphtha have become dizzy, drowsy, or have fainted. Dorner reports a case where the patient fell into a tank of crude benzine and remained comatose for three days before regaining consciousness. A motor paralysis was the sequela.

Temporary Disability.—In mild cases, temporary disability may exist from a few hours to several days. Complications may prolong the temporary disability to several weeks.

Permanent Disability.—If a severe, acute exposure leaves a permanent defect, it is usually a disability of the nervous system. Epilepsy has been reported as occurring several months after the acute exposure. Indictment of the exposure in such instances must be questioned. Retrobulbar neuritis has followed acute exposure, and nervousness over a period of months is frequently alleged. Chronic exposure may result in a temporary total disability in which the workman requires months to several years to regain normal health. Permanent changes in personality, mental confusion, generalized weakness and muscular twitching have been accepted as results of chronic exposure. Such statements are to be critically questioned.

Illustrative Cases:

Case I.—This case was reported by the author[1] in May, 1938. Up to the present writing it is the only one found in the American literature.

Present Illness.—A white male, aged thirty-one, was brought to the Golden State Hospital complaining of severe chest pain, and with dyspnea and cyanosis. His employer who accompanied him stated that

several hours previously the patient was engaged in buffing an auto fender. Desiring some gasoline to remove the fine dust particles, he attempted to siphon gasoline from the tank of the car. Inserting one end of the "airline hose" into the tank, he stretched out on the floor below and started to draw. Due to the diameter of the hose, the fellow had difficulty in removing the hose from his mouth when the gasoline gushed through. This time-interval and the patient's position enabled a large quantity of gasoline to enter the patient's mouth. By the time the victim got to his feet he was strangling violently. He stated, "All I spit out was fumes." His mouth and throat were

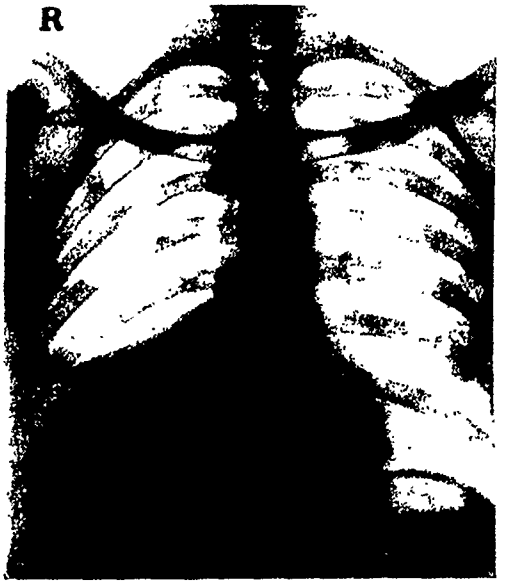


Fig. 20.—(May 20, 1937.) Films taken fifty-two hours after exposure show fluid level in right chest with some clouding of left lung. (Author's article in *Indust. Med.*, 7: 243–251 (May), 1938.)

burned, and within a half hour he experienced pain in the right chest, especially around the nipple area. This became so distressing that he was taken first to the Georgia Street Receiving Hospital and then to our clinic.

Physical Examination.—Examination revealed ashen gray facies with general cyanosis. He assumed a sitting position, leaning to the right, and refused to lie down, claiming that his breath would be cut off. Respirations were 32 and shallow; pulse, 90 with fair volume; temperature, 100; blood pressure, 102/66. Examination of the chest revealed a pronounced lag on the right; percussion sounds on the left were slightly less resonant than normal; on the right they were

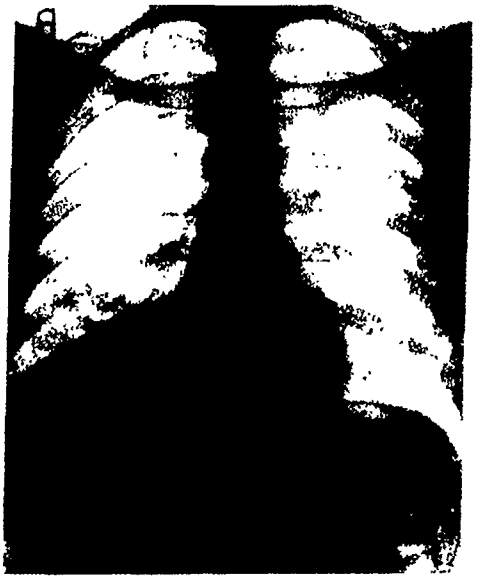


Fig. 21.—(May 24, 1937.) Films taken four days later show partial recession of fluid level with cloudiness existing above fluid level. (Author's article in *Indust. Med.*, 7: 243–251 [May], 1938.)

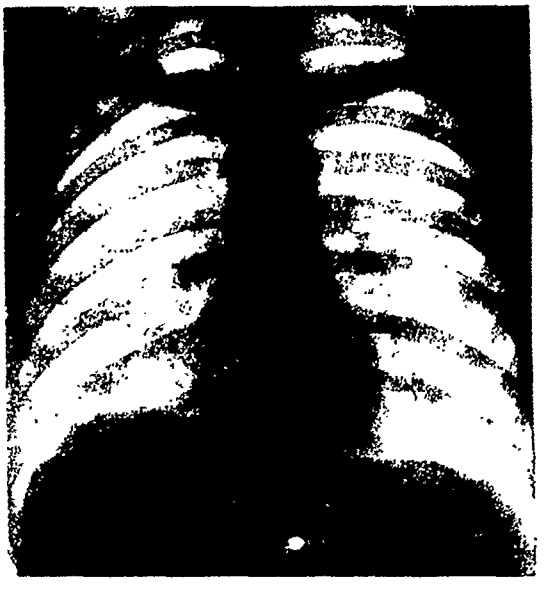


Fig. 22.—(May 27, 1937.) Greater recession of fluid level and more normal appearance of right lung field. (Author's article in *Indust. Med.*, 7: 243–251 [May], 1938.)

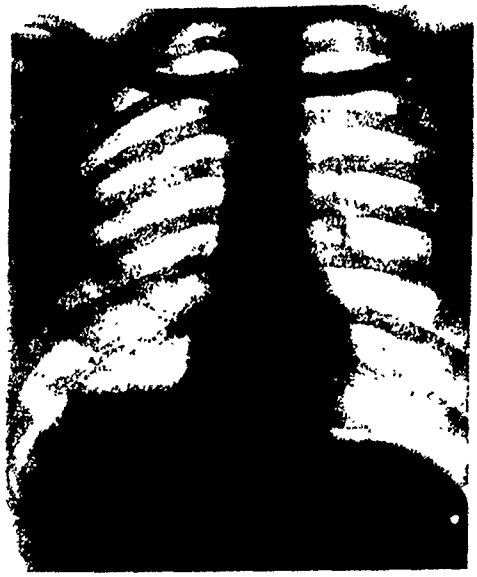


Fig. 23.—(June 3, 1937.) Disappearance of evidence of fluid. Evidence of pleuro-diaphragmatic adhesion. (Author's article in *Indust. Med.*, 7: 243–251, May, 1938.)

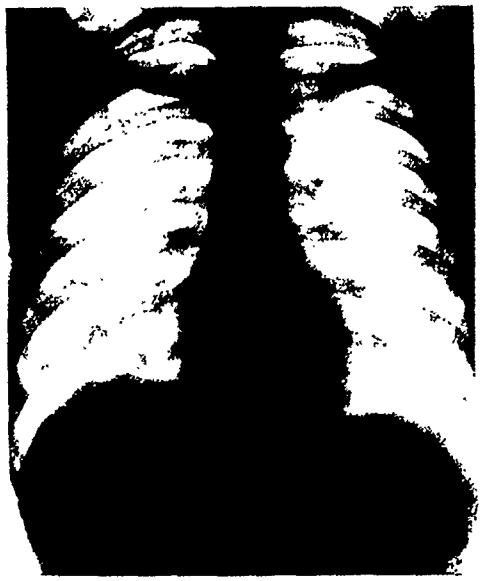


Fig. 24.—(June 11, 1937.) Lung fields in accord with the normal, well-aerated lungs. Adhesion now reduced to a very small area. (Author's article in *Indust. Med.*, 7: 243–251, May, 1938.)

noticeably diminished. There were scattered rales throughout both lungs. The rest of the physical examination and the details of the progress of the case will not be given at this time. He developed a hacking, harassing cough which was productive of a brown sputum. Within thirty hours, evidence of a pleural effusion existed. Roentgenograms confirmed this suspicion. Further confirmation was obtained by the aspiration of 10 cc. of amber-colored fluid. The patient made an uneventful recovery, and was discharged on the sixth day (Figs. 20, 21, 22, 23, 24).

Laboratory Examination.—The laboratory study on this patient was entirely negative. The urine showed a trace of albumin, the blood chemistry was normal, the sputa revealed no evidence of tubercle bacilli and only a few scattered pneumococci. Examination of the sample of effusion showed it to be reddish yellow and slightly cloudy. Microscopic examination revealed numerous lymphoid and blood cells. Inoculation of various media revealed no growth, and the guinea-pig inoculation produced no pathology in two months.

Testimony of the employer and a fellow-employee as eyewitnesses of the accident was obtained. The gas was a cheap grade of cracked gasoline. The patient had been in good health, had lost no time from his work for years, and personal and family history denied the presence of tuberculosis. A sterile effusion argued against the possible etiology of acute infection. The sudden onset and the rapid return to normalcy were in favor of the alleged cause. While no cases had been reported in our own medical literature, several instances of similar disturbances occur in foreign publications. He returned to work within ten days of the accident. The case was considered occupational in origin.

Case II.—A movie director engaged three men to renovate a yacht he had just purchased. One man was assigned to clean and polish the engine-room, which was a rather small confined space. An unidentified gasoline was employed in removing the grease. Failing to appear for lunch, one of the men was found on the floor of the engine-room in a stupor. The glass bottle which contained the gasoline was broken, and the room reeked of gasoline. Brought to the deck, the patient regained consciousness but complained of severe frontal headache and smarting of his eyes. Medical examination two hours later found no objective disturbance, and the laboratory findings were negative. He alleged headache and dizziness for a period of five weeks, at which time he was denied further compensation.

No physical or pathological cause could be found for the man's sudden abnormal condition. The circumstances appeared sufficiently typical to permit a diagnosis of gas intoxication. It was found that

the man had not been employed for several months, and this particular job was for only three days. The question of the actual existence of his symptoms for a period of five weeks entered into the case. Compensation for that period was granted. Upon discharge he maintained that he could not work because of his headache, but four days later he secured a job and worked uninterruptedly.

Case III.—A white male, aged fifty-eight, was employed by a garage as a handyman. One day he was found slumped over in a car he had just parked within the garage. He was taken home and put to bed by order of his family physician. The patient complained of weakness of the right arm, headache, and the doctor noticed a thickness of speech. After four days in bed, he arose one night to go to the bathroom, stumbled, and was found in a stuporous condition. His physician found him to be semi-comatose; the right arm was paralyzed and the left arm fractured. A diagnosis of gasoline poisoning was made, and the claim was received by the insurance company. The insurance carrier dispatched a physician who noted the following: a blood pressure of 240 120, marked peripheral sclerosis, vascular engorgement of the fundi, albumin and casts in the urine. Subsequent study revealed 60 mg. of non-protein nitrogen per 100 cc. of blood. The blood count was not significant. Inquiry into the man's occupation showed that he had had no exposure to gasoline. For the past year he had complained of occipital headaches and tinnitus. He had stopped reading because his glasses "did not fit."

It was established that no undue exposure existed. The physical examination definitely indicated hypertension. Compensation was denied.

Case IV.—A maid employed in a home was asked on numerous occasions to clean wearing apparel with naphtha. One cold day she cleaned several articles in the kitchen. The doors and windows were closed. The patient was found on the floor bleeding from a laceration of the posterior scalp. A garment was soaking in the dishpan of naphtha, and the patient was removed from the kitchen to her bedroom where she regained consciousness, but was unable to account for her accident. Physical examination and laboratory studies were negative. She recovered and resumed her work in three weeks.

In the absence of any other cause, the fact that the patient was leaning over naphtha in a confined room was accepted as sufficient evidence of naphtha intoxication. The resulting head injury disabled her for three weeks. The case was accepted as occupational.

Case V.—A sixty-year-old male was employed in a drycleaning establishment. He reported a loss of weight, increasing fatigue, lessened appetite, and obstipation, to his family physician. The blood

count showed a marked secondary anemia. He was advised to discontinue work, and a diagnosis of "anemia due to a solvent" was made. The diagnosis was accepted, and compensation was paid for the first five months. Subsequent examination showed marked anemia, increased loss of weight, abdominal pain, a mass in the abdomen and blood in the stools. Gastro-intestinal examination revealed evidence of cancer of the large bowel.

Investigation of the man's occupation established the fact that he was a checker of goods as they were loaded onto trucks. He had worked at that occupation for only six months and was never exposed to fumes or gas. Carcinoma accounted for all of his symptoms. Further compensation was denied.

TREATMENT

Acute Cases.—Invariably these cases are acute, and if the patient survives the exposure no specific treatment is necessary.

The Unconscious Patient.—If the patient is seen while unconscious, removal to the fresh air and inhalations of from 5 to 7 per cent carbon dioxide in oxygen are indicated. Saturated clothing should be removed and the skin cleansed of any of the material. The patient should be wrapped in blankets and kept warm. At this time *circulatory failure*, if present, should be treated by any of the following drugs: coramine (1.5 cc.), caffeine sodium benzoate ($7\frac{1}{2}$ grains; 0.5 gm.), metrazol ($1\frac{1}{2}$ to $4\frac{1}{2}$ grains; 0.1 to 0.3 gm.) or camphor in oil (1 to 2 cc.) intramuscularly. The coramine and metrazol may be used intravenously and will also act as respiratory stimulants. Artificial respiration usually is felt to be contraindicated because of the existing lung damage, and adrenalin administration has been found to give poor results.

Prevention of Pulmonary Complications.—The lungs especially need to be watched for complications, such as bronchopneumonia. After the first portion of the acute stage where respiratory stimulation chiefly is needed, oxygen therapy may be substituted for the carbon dioxide and oxygen inhalations. For cough, codeine phosphate or sulfate ($\frac{1}{2}$ to 1 grain; 0.0325 to 0.065 gm.), perhaps in a cough-syrup vehicle, is useful.

Nervousness and Restlessness.—These conditions may require sedation by any of the following drugs: chloral hydrate (10 to 30 grains; 0.65 to 1.95 gm.), phenobarbital ($1\frac{1}{2}$ grains; 0.1 gm.), sodium bromide (10 to 30 grains; 0.65 to 1.95 gm.), or paraldehyde (2 to 4 drachms; 8 to 16 cc.). The paraldehyde may be mixed with elixir lactate pepsin, poured over crushed ice, and administered by mouth, or it may be given rectally in olive oil with 1.5 cc. benzyl alcohol. Epsom salts (1 ounce; 30 gm.) should be given.

Venesection, followed by blood transfusions, to rid the body of methemoglobin, has been advocated.[9] When ingestion of the substance has occurred, lavage with epsom salts solution is of value.

Conjunctivitis.—Due to irritation from petroleum distillate fumes, conjunctivitis may be treated by dropping 1:1000 adrenalin solution into the eyes four times a day, followed by the use of cold applications for from ten to fifteen minutes. Bland boric acid ophthalmic ointment should also be used.

Chronic Cases.—Patients in chronic cases chiefly need removal from their exposure. The *diet* should be high in calories with added vitamin preparations. For the *secondary anemia*, ferrous sulfate (5 grains; 0.32 gm.), two tablets three times a day, may be used. The ferrous sulfate preparations seem effective in relatively small doses, and they also seem to cause less gastro-intestinal discomfort than other iron compounds.

REFERENCES

1. St. Ledger, A. W.: "Inhalation of Petrol," *Med. J. Australia, 1:* 500 (March 18), 1922.
2. Schneider, H.: "Über akute Benzin-vergiftung," *Med. Klin., 29:* 1168–1169 (Aug. 25), 1933.
3. Battley, J. C. S.: "Effect of Liquid Gasoline on Pulmonary Tissue," *J.A.M.A., 94:* 1570–1571 (May 17), 1930.
4. Johnstone, R. T.: "Pleural Effusion from Gasoline Inhalation," *Indust. Med., 7:* 243–251 (May), 1938.
5. Askey, J. M.: "Aplastic Anemia Due to Benzol Poisoning, Report of Two Cases," *California and West. Med., 29:* 262–263 (Oct.), 1928.
6. Haythurst, E. R.: "Poisoning by Petroleum Distillates," *Indust. Med., 5:* 53–63 (Feb.), 1936.
7. Johnson, J. G. W.: "Toxic Effects of Gasoline Fumes," *Canad. M. A. J., 3:* 118, 1913.
8. Hamilton, A.: *Industrial Poisons in the United States*, The Macmillan Co., New York, 1929, p. 404.
9. Machle, W. F.: "Tetra-ethyl Lead Intoxication and Poisoning by Related Compounds of Lead," *J.A.M.A., 105:* 578–585 (Aug. 24), 1935.
10. Barbour, O.: "Kerosene Poisoning," *J.A.M.A., 87:* 488 (Aug. 14), 1926.

CHAPTER VI

CARBON TETRACHLORIDE

Carbon tetrachloride is a clear, colorless liquid, having an odor resembling that of chloroform. It is insoluble in water but mixes with alcohol, chloroform, ether, benzene, benzine, and dissolves most of the oils. The fumes are heavy, being 5.3 times heavier than air.

Occupational Hazards.—Carbon tetrachloride is used widely as a degreasing agent for metals, textiles, and in drycleaning; as a solvent

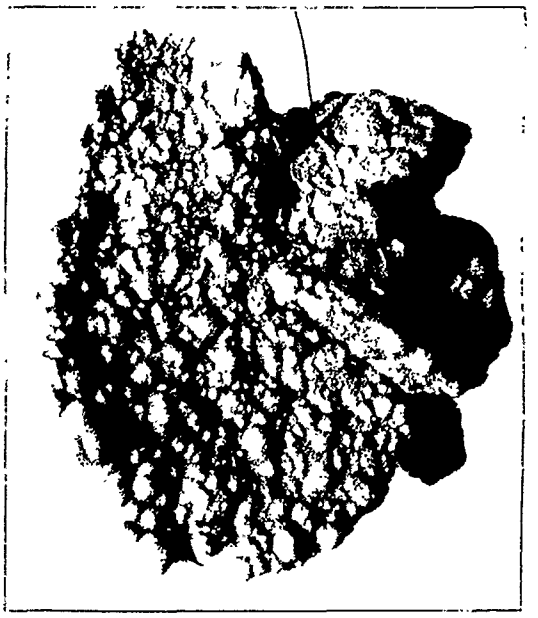


Fig. 25.—Liver of Rat 377, showing advanced cirrhosis; color, yellow; 119 exposures to 100 parts per million. (Smyth and Smyth: *J.A.M.A.*, Nov. 21, 1936.)

for rubber, lacquers, resin, and animal fats; in the doping of airplane wings; in the manufacture of fire-extinguishers; as a delousing agent; and is commonly found in the home as an agent to remove grease spots.

Pathology.—Intentional ingestion of this substance for medicinal purposes (hookworm) does not concern us.

Fig. 26.—Liver of Rat 395, showing regenerated cirrhosis; color, normal; 68 exposures to 400 parts per million, and 156 days without exposure; section resembles Fig. 27. (Smyth and Smyth: *J.A.M.A.*, Nov. 21, 1936.)

Fig. 27.—Liver of Rat 393, showing regeneration from cirrhosis, with contracted septums; magnification, 250; 76 exposures to 400 parts per million; 63 days without exposure. (Smyth and Smyth: *J.A.M.A.*, Nov. 21, 1936.)

General Effects.—Industrially, inhalation of the fumes through the respiratory tract produces changes in the tissue of various organs, alteration of metabolism and disturbances of the blood chemistry. The gas, like chloroform, exhibits powerful anesthetic properties. Sudden severe exposure results in a depressive action upon the heart. Sudden death is due to failure of the heart or ventricular fibrillation, rather than respiratory failure.

Specific Effects.—Animal experimentation reveals a necrosis of the liver (Figs. 25, 26, 27). This undoubtedly occurs in human exposure but it would appear that the kidneys in the human are more involved than the liver. There is an increase in the protein metabolism, the by-products of this increase being sulphuric acid, phosphoric acid, oxalic acid, beta-oxybutyric acid and others, leading to a condition of acidosis. Poindexter and Greene[1] report an autopsy of a case of long-standing exposure which showed cirrhosis of the liver with ascites, infarction of the right kidney, and compensatory hypertrophy of the left.

DIAGNOSIS

Signs and Symptoms.—Nausea, vomiting, weakness, vertigo, burning sensation in the epigastrium, headache, excitement, cough, and narcosis are present in the early stages, followed by oliguria, albumin and casts in the urine, symptoms of uremia, convulsions, deep coma, and death. Chronic exposure will cause headache, vertigo, decreased appetite, gastric distress, and allegedly jaundice without ascites.

Some investigators believe that an *individual susceptibility* exists resulting from lowered nutritional states, alcoholism, and deficient intake of calcium. They maintain that carbon tetrachloride combined with alcohol exerts a synergistic action upon the liver.

Test. *For Concentration in Air.*—Examination of the air where the patient works may be necessary as well as expedient. For a working day, 100 parts per million parts of air is considered a safe level; for short exposure, a concentration of below 500 parts per million.

For Presence in Body.—In cases of death, chemical analysis of the various tissue will affirm or deny the presence of carbon tetrachloride. While there exist no simple, specific tests to prove the presence of this substance in the body, certain examinations will serve as a reliable aid when considered with all other factors in a given case. It has been shown that long exposure or severely acute exposure may produce amblyopia characterized by concentric restriction of the visual fields, damage to the liver, and alteration of the calcium metabolism; and therefore *measurements of the visual fields*, the *icteric index*, and the *van den Bergh reaction* should be made. *Tests of the blood calcium*

should further aid in the diagnosis of suspected cases. Smythe[2] believes that repeated examinations of this nature at intervals, in those working in known exposures, would serve to detect early injury.

MEDICOLEGAL ASPECTS

Exposure outside Industry.—Because of the cheapness of this solvent and the fact that it is non-inflammable, its frequency of use is mounting daily. Its increased use is bound to produce numerous claims for disability. It is of utmost importance, therefore, that emphasis be stressed at this time upon several points. Today, acute or chronic reactions to carbon tetrachloride are actually rare. The majority of cases happen outside industry where ignorant use of the substance has occurred. Likewise, many of the cases referred to in medical literature took place before the present-day knowledge of the solvent induced proper ventilation as well as proper precautions. During the first World War, production was at a premium, and both precaution and ventilation were neglected. Much of the present-day impression regarding its dangers is a result of that period of unsupervised use.

Fatalities in Industry.—The almost negligible effect of this agent can best be illustrated by referring to the work of Smythe.[3] His review of the literature up to 1935 showed only twenty-seven fatal cases of acute poisoning. Fourteen were the result of the administration of this substance as an anthelmintic. Of the remaining thirteen cases, only six occurred in the United States. He found only thirty-three non-fatal, acute, or subacute industrial cases in this country, but not all were typical, since thirteen patients were exposed to mixed preparations. Ninety-six patients working in a carbon tetrachloride hazard were studied by him. Correlating his animal experimentation, men working for a long period of time in this exposure developed a resistance showing fewer abnormalities than the men just starting to work in such a hazard. No man considered materially or appreciably weakened by the exposure was found, nor was there found among them pallor or jaundice. One enlarged liver was seen in a man in this group who was admittedly a chronic alcoholic. The men uniformly reported that the subjective symptoms of headache, nausea, vomiting, and fatigue were present only during brief periods when the ventilation was bad, or when machine-cleaning, spilling, or other intermittent exposures were present. Contrary to common belief, anemia was not ascribed to. This investigation is reviewed here to offset prevailing misconception of the action of this solvent *in industry*. The symptoms previously outlined do occur in persons exposed

to carbon tetrachloride, but they usually occur in non-industrial exposures.

Temporary Disability.—This is short, a few days at the most. Acute, severe exposure may produce symptoms lasting for from several weeks to several months, although complications such as nephritis may prolong this period.

Permanent Disability.—This is extremely rare. Removal from exposure results in recovery. When liver cells are damaged, regeneration usually occurs. Impaired vision usually returns to normal.

Illustrative Cases:

Case I.—A workman dropped a container of the carbon tetrachloride in the basement of a drycleaning establishment. While attempting to improvise a cork for the neck of the container he was overcome. His employer awaiting him upstairs became impatient and went down to this small, dank basement to find the employee completely unconscious. The employer first shook the patient, then dragged him to the stairway, where he too collapsed. The office girl, wishing to inform her employer that he was wanted on the telephone, found them and summoned help. The employer revived in the ambulance, and the employee shortly after he arrived at the hospital. Both patients made uneventful recoveries.

The exposure was definite and the reaction typical. Recovery was in keeping with the usual run of cases.

Case II. *Present Illness.*—A fifty-two-year-old janitor noticed an increasing fatigue, generalized weakness, dyspnea, palpitation, and edema of the ankles. After he had complained for some weeks, his wife noticed that he seemed quite pale. During this time one of his helpers became ill one night while handling a cleaning solution. It occurred to the janitor that possibly this substance which he used on the brass fixtures almost daily was bothering him. He consulted his physician who, following a physical examination, "felt inclined" to believe that the patient's suspicions were correct. He was ordered to stop work, rest, and to get plenty of fresh air. Several weeks later, a claim for compensation was made. One month later he was examined at our hospital.

Physical Examination.—Upon examination it was noted that his color was slightly lemon-tinged. The tongue was smooth and sore. Extreme weakness was evident, and exertion in getting on and off the examination table produced marked shortness of breath. He had a bilateral reading of 20 on the gripping machine. Pulse was 88, heart tones weak, blood pressure, 110 80. Abdominal examination was negative. Prostate normal. Neurological examination essentially negative. No particular numbness of the limbs was complained of.

Laboratory Examination.—Urine, 2 plus albumin, with many granular casts, no sugar; red blood cells, 2,200,000; hemoglobin, 72 per cent; color index, 1.63; marked distortion of the red cells. The gastric contents were not examined.

No blood investigation was made at the time the patient was first seen. The rather universal opinion that solvents produce anemia was accepted by the family physician, and he assumed that the man had been affected by a solvent. Investigation of the cleaner showed that it did not contain tetrachloride. Its purpose was to polish, not to clean or act as a degreaser. The patient had pernicious anemia. Placed upon the proper therapy the man regained his health. The case was not compensable.

Case III.—Butch reports a case where a fifty-four-year-old man used a solution of 60 per cent carbon tetrachloride and 40 per cent gasoline to clean old telephones. Following a six months' exposure it was necessary to hospitalize him, at which time it was found that he had cirrhosis of the liver. After four months of treatment and absence from work he recovered.

From the history of this case, the illness was occupational in origin, and compensation should be allowed for a temporary disability of four months. The case illustrates the point that liver regeneration occurs in carbon tetrachloride intoxication and that permanent dysfunction of the liver is not to be expected.

Case IV.—Gonzales[4] reports the case of a housemaid, aged forty, who contrary to directions on the gallon container of "Quick as a Wink" cleaning-fluid, poured almost the entire contents in a basin and dipped the garments in the liquid. She immediately became dizzy and faint, staggered out of the small room in which the operations were performed, and collapsed. A physician after reviving her expressed the opinion that she had suffered a "heart attack." A few days thereafter, at the behest of her mistress, she proceeded to clean dresses in a similar manner and was found dead beside the basin containing the fluid.

Necropsy.—The necropsy was negative except for a moderate grade of chronic diffuse nephritis, pial edema, and injection of the laryngeal and bronchial mucosa.

A chemical examination of the organs revealed the presence of carbon tetrachloride and dichloropropane. The analysis showed 18 mg. in 500 gm. of lung and 14.5 gm. in 500 gm. of liver, of chlorinated hydrocarbons of low boiling point.

The amounts were too small to isolate the substances in pure form. These results were determined colorimetrically. Analysis of the color-

less fluid remaining in the container indicated approximately 60 per cent carbon tetrachloride and 40 per cent dichloropropane.

No comment is needed other than to state that this is an example of exposure occurring outside of industry where ignorance of the solvent, its dangers and the proper methods of usage exist.

Case V.—Hauseman and Helly[5] tell of a forty-six-year-old disinfector who inhaled the fumes of carbon tetrachloride. He died on the twenty-sixth day after his intoxication. Death was hastened by a preexisting nephritis.

It has been previously stated that this substance affects the kidneys. In this instance, the already insufficient kidneys were further damaged. Death was compensable since it is held that even though a workman has a disease existing at the time of injury, the employer has accepted him as he is; therefore, the aggravation of the disease resulting in death is compensable.

Case VI.—A thirty-eight-year-old Negro male was first seen by the writer on March 6, 1940. His usual occupation was that of a yardman engaged in handling scrap tin. On March 1 he was assigned the task of cleaning long felt bags which had contained lead dust. He did this by immersing these bags into a large vat which contained a cleaning solution. He could not estimate how many bags he had washed but states that he stood over this solution for seven and one-half hours.

Present Illness.—As he was finishing his day's work he felt sick to his stomach, and on his way home he vomited several times. This vomiting continued, and a severe headache ensued. Along with this headache the patient noted a bubbling sensation in his ears and a "waxy haze" over his eyes. Later that evening he noted several blebs or blisters on the dorsum of both hands and described these as "water blisters." The following day he reported to the office of the community physician, who opened these blisters and applied ointment. At this time the hands were quite swollen, nausea was still present but no vomiting, and headache was severe. The patient complained of an exceedingly dry throat, and the "waxy haze" over his eyes persisted.

Past Medical History.—Pneumonia at the age of seven; otherwise negative. Denies venereal disease.

Physical Examination.—The patient was a tall, well-built adult Negro. His pupils were small, and the reaction to light was questionable. The eyegrounds were normal in appearance. The teeth were stained, and there was some recession of the lower gums. The throat was injected and inflamed. The tonsils were large and cryptic.

(*a*) *Lungs.*—No adventitious sounds were noted. The heart rate was 80, regular; no murmurs were heard. Blood pressure was 140 90.

(*b*) *Abdomen.*—No organs or masses were palpated. There was alleged tenderness over the upper mid-quadrant.

(*c*) *Extremities.*—The skin over the dorsum of both hands was wrinkled, and there were numerous denuded areas. The surfaces of both hands including the fingers and palmar surfaces were greatly swollen. The denuded areas constituted second-degree burns.

Laboratory Investigation.

Tests on March 7, 1940

Urinalysis

Voided specimen:
Straw color
Clear appearance
Specific gravity, 1.022
Acid reaction
No sugar, albumin, or acetone
Microscopic: 3–4 pus cells.
Mucous threads present

Blood Count

Hemoglobin .....88 per cent
Erythrocytes 5,520,000 per cu. mm.
Leukocytes .....7550 per cu. mm.
Polynuclear
neutrophiles .....55 per cent
Lymphocytes .....39 per cent
Large mononuclears ....3 per cent
Eosinophiles .....3 per cent

Blood Tests

Wassermann .....negative
Kahn .....negative
Laughlen .....negative

Tests on March 11, 1940

Urinalysis

Voided specimen:
Straw color
Hazy appearance
Specific gravity, 1.018
Acid reaction
No sugar, albumin, or acetone
Microscopic: 0–1 pus cells

Blood Count

Hemoglobin .....92 per cent
Erythrocytes 5,300,000 per cu. mm.
Leukocytes .....8100 per cu. mm.
Polynuclear
neutrophiles .....45 per cent
Lymphocytes .....43 per cent
Large mononuclears ....6 per cent
Eosinophiles .....6 per cent

This patient improved slowly, and ten days after his initial exposure the headache had disappeared, there was no nausea or disturbance of hearing, and there was no complaint of disturbed vision. He was annoyed by an exceedingly dry throat. The burned hands improved gradually and were completely well twenty-six days after the initial burn.

Inquiry revealed the fact that the solution used contained 25 per cent carbon tetrachloride and 75 per cent ethylene dichloride. Among

other effects from the latter substance there has been reported a smoking of the cornea due to an infiltration by the lymphocytes and connective tissue cells. When such a disturbance occurs, it usually clears up within several months. This reaction was not actually noted in the case just reported, but from his complaints there must have existed a mild but unrecognized alteration. His general systemic complaints were compatible with carbon tetrachloride intoxication.

The reaction to the hands is not common. Ordinarily these solvents do not cause burns to the skin to this degree. It is thought, however, in this instance that the continual submergence of the hands in the solution for over seven and one-half hours did produce the burns noted. It is interesting to note that the patient was not warned against the character of the solution, and the employer did not know the consistency of the substance used. The case was compensable, the temporary disability being one month. There was no permanent disability.

Case VII.—On March 29, a night janitor employed by a movie theater was assigned to clean a carpet in a restroom, which had only a small opening in the ceiling for ventilation. He worked on his hands and knees over a bucket of cleaning fluid, constantly wringing out the cloth and re-dipping it in the cleaning solution. He worked at this for hours with one short intermission in the fresh air. He went home and slept from 6 A.M. until 1 P.M.

Present Illness.—Upon awakening he felt rather dizzy and had a severe headache. Thirty hours after the exposure he became nauseated and vomited several times. He felt feverish and chilly, and his eyelids, hands, and feet became swollen and felt stiff. Frontotemporal headache continued. Vomiting also continued at intervals for the next five days. He developed a cough, expectorated blood-streaked sputum, and the swelling of his hands and feet grew gradually worse.

On April 5, he voided practically no urine and had extreme burning on attempts at urination. He stated that he could void only a few drops of urine at a time. It was on April 5 that he was brought to the Golden State Hospital. The patient's complaints on admission were headache, dizziness, recurring nausea and vomiting, loss of appetite, burning on urination, oliguria, stiffness of the fingers, and swelling of the hands, feet and eyelids.

Past Medical History.—Entirely negative for any previous injury or serious illness. The patient is married; has no children. His habits are temperate. His only alcoholic indulgence is that of a light wine.

Physical Examination.—The patient was a robust, white male of about forty-five years, appearing to be acutely ill. Temperature,

97.4 F.; pulse, 64; respiration, 20; blood pressure: systolic, 156; diastolic, 90. The skin was free from eruption and presented slight generalized pitting edema.

(*a*) *Head.*—There was a diffuse subconjunctival hemorrhage of both eyes. The fundi appeared to be clear on the first ophthalmoscopic examination.

(*b*) *Lungs.*—A few large, rhonchus râles were heard. Otherwise normal.

(*c*) *Abdomen.*—Tenderness existed across the upper abdomen, particularly over the liver area. Upper border of liver dulness at sixth rib; lower border at the costal margin. Liver palpable and quite tender. There was no rigidity of the abdominal muscles.

Laboratory Examination.

Urinalysis	*Blood Count*
Straw color	Hemoglobin .....80 per cent
Clear appearance	Color index ..... .89
Acid reaction	Erythrocytes 4,570,000 per cu. mm.
Heavy albumin content	Leukocytes ....11,050 per cu. mm.
Numerous epithelial and pus cells	Small lymphocytes.....22 per cent
No blood, casts, sugar, or acetone present	Large lymphocytes.....6 per cent
	Large mononuclears ....1 per cent
	Polynuclear neutrophiles 71 per cent
	N.P.N. .....128 mg. per 100 cc.

The urinary output during the first twenty-four hours after admission was only 40 cc. On the second day it was 60 cc.; third, 33 cc.; fourth, 60 cc. The fifth day, or the eleventh day after exposure, it was 250 cc. From this period there was a gradual increase in the urinary output.

Blood chemistry on the second day after admission, or the eighth day of the disease, was as follows: N.P.N., 77.5 mg. per 100 cc.; urea nitrogen, 56.2 mg. per 100 cc.; creatinine, 5.5 mg. per 100 cc.; uric acid, 20 mg. per 100 cc.

Subsequent urine examination continued to show heavy amounts of albumin, and microscopic blood appeared. The blood chemistry repeated on April 11, or the thirteenth day, was as follows: N.P.N., 100 mg.; urea nitrogen, 68.5 mg.; creatinine, 4.5 mg.; uric acid, 8.4 mg.

Two days following admission, the blood-pressure reading was 170 100. This gradually receded until at the time of discharge it was 116 76.

On April 12, examination of the right and left eyes showed the media clear. Retina, disc, macula essentially normal.

On April 24, the blood count in contrast to the previous one revealed: hemoglobin, 110 per cent; color index, 1.1; erythrocytes, 5,010,000; leukocytes, 9100; polynuclear neutrophiles, 74 per cent.

Shortly after the patient entered the hospital in addition to the liver dulness and the tender liver, he developed an icteric tinge to the skin and sclera. This was very light. On April 24, approximately twenty-six days after the onset, the van den Bergh tests, both direct and indirect, were negative, and the icteric index was 2.5.

On May 15, a repeated Wassermann was negative. The renal function tests, including the phenolsulfonphthalein and Mosenthal, indicated satisfactory kidney function, and the N.P.N. on that date was 37.5 mg. per 100 cc.

The patient was discharged from the hospital on the third day of May. He was examined at periodic intervals; on June 15, for instance, he had gained weight; blood pressure was 110 66; pulse, 72. There was still slight residual puffiness of the eyelids. The urinalysis on that date was negative.

Concerning the injury to the eyes, the following report of the case by the ophthalmologist is of interest:

Examination of the eyes was first done about the second day of hospitalization. At that time the patient complained of "blurred vision." He stated that he could recognize people but that the image was "blurred." A detailed examination of the visual acuity was not done at this time since the patient was extremely ill and it was difficult to get his full cooperation. Examination showed the presence of subconjunctival hemorrhages involving the bulbar conjunctivae and external canthi of both eyes. The ocular tension as estimated by palpation was found to be within normal limits. The conjunctivae were pale. The corneas were clear and iris of each eye negative. After dilatation the pupils were found to be round. The media was clear. The fundi were clear. No variation from the normal was seen in the macula, nerve head, or retina of either eye.

After the patient was discharged from the hospital further examination of the eyes was done. The patient complained that he could not see as well as formerly for distance or near. Examination of bulbs showed them to be essentially negative. No changes were seen in the fundi. Uncorrected vision was found to be R.E., 20 30; L.E., 20 40; near, Jaeger 111, R.E.; Jaeger 111, L.E. After the use of homatropine, vision was found to be 20 50-2 in the right eye and corrected to 20 20-3 with a plus 75 sphere combined with a plus 25 cylinder axis 90 degrees. Left eye 20 70 corrected to 20 20-2 with a plus 1 sphere. Posteycloplegic examination of near vision showed correction to Jaeger 1 with a plus 75 sphere addition in each eye.

Perimetric examination of the visual fields showed a contraction for form and color in both eyes and within 30 degrees when using a

5 mm. diameter test object on a 30 cm. radius perimeter. The central and blind spots were explored with one degree test object at 1 meter distance. No change was found in these areas. The patient had difficulty distinguishing green and blue in all areas within the field of vision.

A further check-up examination on October 28, showed no essential changes in visual fields other than that noted in the previous examination. Visual acuity, however, had improved to R.E., 20 20-3 and L.E., 20 20-4 uncorrected; and corrected to R.E., 20 20-1; L.E., 20 20-2.

At the time this patient was admitted to the hospital the exposure was established by the testimony of a fellow-worker and by his employer who assigned him to this job and gave him the cleaning-fluid that was used. The manufacturer of this fluid first maintained that this solution contained no harmful substances and specifically claimed that it contained no carbon tetrachloride. However, analysis revealed it to be a very light naphtha containing 25 per cent carbon tetrachloride.

The findings of acute toxic nephritis, a hepatitis, bronchitis, and a disturbance of vision were in keeping with the known effects of undue exposure to this solvent. Temporary disability lasted from March until December of the same year, and during this time the insurance carrier assumed all liability. On December 11, the patient brought suit against the manufacturer and recovered his claim. He was then lost sight of, and no further statement can be made as to whether there remained any permanent disturbance of his visual field. Other than this possibility, there remained no permanent disability.

TREATMENT

The severe case of carbon tetrachloride poisoning will require a rigid regimen, the purpose of which is twofold: to *treat the liver and renal damage*, and to *restore a normal blood chemistry*. The patient should be removed from all contact with the substance. He should be supplied with an abundance of fresh air and with inhalations of from 5 to 7 per cent carbon dioxide in oxygen, if necessary, for respiratory stimulation.

Increasing Blood Calcium Level.—These patients have a low blood calcium level. Therefore, a high calcium intake should be provided to bring their blood calcium to the high or normal level, which it has been shown experimentally prevents liver and renal damage to some degree. This can be accomplished by giving 10 cc. of a 10 per cent solution of calcium gluconate intravenously two or three times daily, with 12 gm. calcium gluconate, or 8 gm. of calcium lactose by mouth.

Proteins in Diet.—The diet itself should contain a high proportion of carbohydrates, 200 to 250 gm. or more per day, together with a low fat and a low *meat* protein ratio. The *meat* protein is limited because of the evidence that increased retention of guanidine prolongs and increases the symptoms of carbon tetrachloride poisoning. However, in view of the demonstrated value of protein in liver regeneration, a high level of other forms of protein should be maintained. Actually, following the early acute symptoms, a still higher protein level would probably be of value. Bread, milk, sugar, and Karo syrup are obviously of much value in the diet, to which may also be added extra vitamins.

Dextrose Solutions.—The intravenous administration of from 2000 to 3000 cc. daily of 10 per cent dextrose in physiological saline is indicated. In *renal disease* or when *circulatory collapse* induces defective renal function, the injection of physiologic solution of sodium chloride may occasionally produce or aggravate acidosis. This effect is brought about chiefly by diluting the bicarbonate already in the body at a time when the kidneys are not excreting the excess of chloride. In some instances, then, the use of 10 per cent dextrose in distilled water, rather than in 0.9 per cent sodium chloride may be indicated. However, in most instances the 0.9 per cent sodium chloride solution may be used since even abnormal kidneys excrete salt solution well if serum proteins are not reduced or cardiac failure is not present. Dextrose solutions, by providing food and aiding the circulation, help the kidneys to adjust extracellular volume and concentration when sufficient sodium chloride is available.

The administration of from 50 to 200 cc. of 50 per cent dextrose intravenously per day is also of value, but where *cardiac involvement* is present, not more than 50 cc. should be given at one time. It has been suggested that some of the fluids may be given per rectum, rather than intravenously; 120 cc. of 10 per cent dextrose every four hours may be used rectally.

Insulin.—From five to ten units of insulin three times per day may aid in the oxidation of the intermediate products of protein, fat, and carbohydrate. There have been a few reports of the satisfactory use of Hartmann's buffer solution and of methylene blue, but neither of these agents has been used extensively in these cases.

Digitalis.—This has been advised routinely for the treatment of myocardial involvement but would seem to be of questionable value in most cases. When used, regular digitalizing dosages of $1\frac{1}{2}$ grains (0.1 gm.) per 4.5 kg. of body weight should be employed. *Epinephrine* should not be used, particularly if myocardial involvement is present.[6]

Diuretics.—Diuretics, other than the mercurial ones, are indicated. In treating oliguria or anuria the use of papaverine hydrochloride or sulfate, $\frac{1}{2}$ grain (0.0325 gm.) intravenously or by mouth, is said to be of value on the basis that these drugs relax the arteriolar spasm associated with the toxic nephritis. There are claims of success for the use of hexylresorcinol for treatment of kidney irritations.

Other Medication.—Free catharsis is of value, but oily preparations are contraindicated. Transfusions are sometimes necessary. If *anemia* of the secondary type is present, ferrous sulfate in enteric coated tablets, 5 grains (0.32 gm.) to two tablets, three times per day should be prescribed, and if a macrocytic type of anemia accompanies the liver damage, the use of liver extract is indicated. If any *bronchial conditions* develop, inhalations of compound tincture of benzoin and pineneedle oil, followed by a cough syrup with codeine phosphate or sulfate added should be used. Sedation may be necessary for the *restless* or for the excited. When the *eyes* are involved, their treatment should be consigned to the ophthalmologist.

Experimental work with a purified concentrated hog's liver extract[7] has demonstrated an accelerated healing of liver tissue in rats after carbon tetrachloride poisoning, in addition to protecting the liver from damage. The active ingredient appeared to be sodium xanthine. Its clinical use in carbon tetrachloride poisoning is not yet reported.

Prophylaxis.—From the standpoint of prophylaxis, proper ventilation is essential. In addition, closed systems should be used, or, if these are not possible, positive-pressure helmets may be necessary when the concentrations are very high. Exhaust systems should be provided with intakes at the floor level rather than near the ceiling. The possibility of skin contact with the substance should be eliminated. There should be frequent examination of workers, and careful selection of those who are to work with this hazard. Rotation of workers should be frequent. The diet of carbon tetrachloride workers should be high in calcium, including at least one quart of milk daily. Alcoholic beverages should be avoided.

REFERENCES

1. Poindexter, C. A., and Greene, C. H.: "Toxic Cirrhosis of the Liver. Report of a Case Due to Long Continued Exposure to Carbon Tetrachloride," *J.A.M.A.*, *101*: 2015–2017 (June 16), 1934.
2. Smythe, H. F., Smythe, H. F., Jr., and Carpenter, C. P.: "Chronic Toxicity of Carbon Tetrachloride; Animal Exposures and Field Studies," *J. Indust. Hyg. and Toxicol.*, *18*: 277–298 (May), 1936.
3. *Ibid.*

4. Gonzales, T. A.: "Needs for Improvement in Medico-Legal Investigation," *New England J. Med.*, *215*, 385–390 (Aug. 27), 1936.
5. McNally, Wm. D.: *Toxicology*, Industrial Medicine Publishing Co., Chicago, 1937.
6. Davis, P. A.: "Carbon Tetrachloride as an Industrial Hazard," *J.A.M.A.*, 962 (Sept. 29), 1934.
7. Barrett, H. M., MacLean, D. L., and McHenry, E. E.: *J. Pharmacol. and Exper. Therap.*, *64*: 131 (Oct.), 1938.

CHAPTER VII

CARBON DISULFIDE

Investigators in most every industrial country have called attention to the harmful effects of this gas, but in America it has received scant notice. As far back as 1865 Delpech, in France, recognizing its devastating effects upon human tissue, remarked, "He who works in sulfur (CS_2) is no longer a man."

Occupational Hazards.—Carbon disulfide once found widespread use as a solvent. Because of its deleterious effects it was later largely replaced by less noxious substances. Industry, however, still finds it indispensable in certain processes, the chief of which are these: as a solvent in the extraction of fats, oils, rubber, sulfur, iodine, the oils from seeds and bones; in the preparation of some lacquers and varnishes; as an insecticide and disinfectant; and finally and most especially in the manufacture of rayon (viscose rayon). It is mistakenly thought that this fast-growing industry is located only in the Pennsylvania-Delaware area. Actually there are fifteen or sixteen plants engaged in the manufacturing of artificial silk scattered elsewhere in the United States.

Survey of Literature.—The average industrial physician rarely comes in contact with illness resulting from chronic carbon disulfide poisoning; the medical literature is devoid of articles concerning it; textbooks of medicine ignore it, and even recent publications dealing with occupational diseases fail to describe it. Until Gordy and Trumper made their report[1] in 1938, no papers dealing with this subject had been published in America since 1905, although Dr. Alice Hamilton had urged an investigation of the rayon industry in 1937.

In 1938, with the cooperation of the United States Department of Labor,[2] the medical staff of the University of Pennsylvania, under Lewy, the American Chemical Society, and the Pennsylvania Department of Labor and Industry, the first coordinated group study of this gas ever to be conducted, either in America or in foreign countries, was made. Owing to the inexperience of the profession with this substance, and to the fact that a better understanding will become obligatory on all industrial physicians, a résumé of the aforementioned survey is presented.

Résumé of Group Study.—Carbon disulfide belongs to the anesthetic group of noxious gases. It is a colorless, transparent fluid which

is inflammable and highly explosive, an additional reason why it was largely discarded as a solvent. It enters the body through the lungs and is immediately carried by the bloodstream to all the organs, including the brain. It is eliminated through the urine, skin, and lungs. Lehmann[2] concluded, following his experiments, that from 80 to 85 per cent of the inhaled carbon disulfide is again exhaled, leaving from 10 to 15 per cent to be retained. If poor ventilation is present, exhaled fumes may be returned to the workman in the atmosphere to be again inhaled. Thus intoxication is dependent upon the relationship

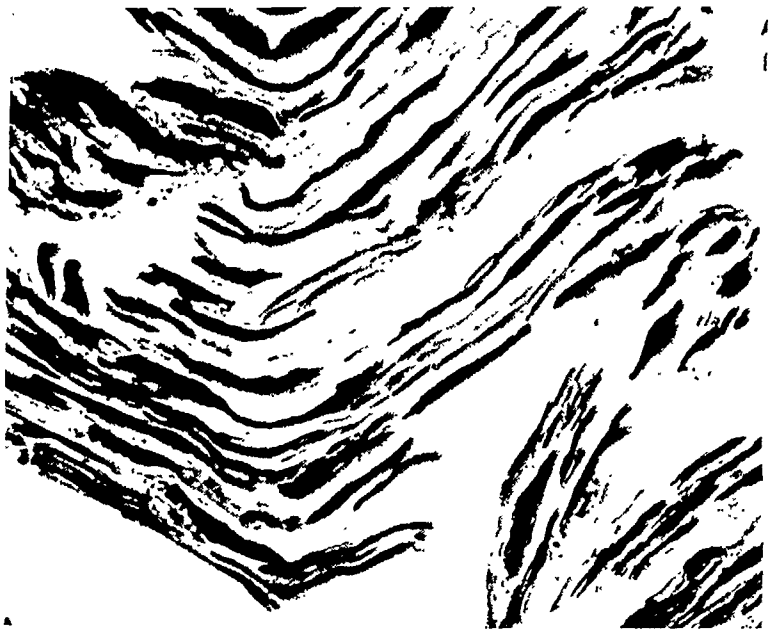


Fig. 28.—Longitudinal section through the sciatic nerve of a dog with experimental CS_2 poisoning. The axis cylinders are distorted, swollen, and fragmented while the myelin sheaths still stain fairly well. (Lewy.)

between the intake and the exhalation of CS_2. Dissipation of the fumes at the same pace as the intake will prevent the appearance of symptoms.

Artificial silk, or rayon, is made by a viscose process, which is the converting of cellulose into a gelatinous mass by the addition of sodium hydroxide and carbon disulfide. During this process, hydrogen sulfide is evolved. This by-product is less noxious and damaging than carbon disulfide. Carbon disulfide has a specific gravity of 2.62 as compared with air, and hydrogen sulfide 1.17 as compared with air. Being heavier than air, both of these substances hang in the

breathing zone of the workers. Following their investigation, the Pennsylvania Department of Labor and Industry[2] set the following standard of the permissible limits of CS_2 and H_2S in the breathing zone of the workers: 10 parts per million for CS_2, when existing alone, and 10 parts per million for H_2S, when alone. If combined, then 5 parts per million of each.

Pathology.—Primarily this gas produces severe *neural poisoning*. This can be understood when it is realized that CS_2 is a strong solvent for fat and that the neural tissue function is dependent upon the lipoid set-up in the nerve fibers, myelin sheaths, and cytoplasm (Figs.

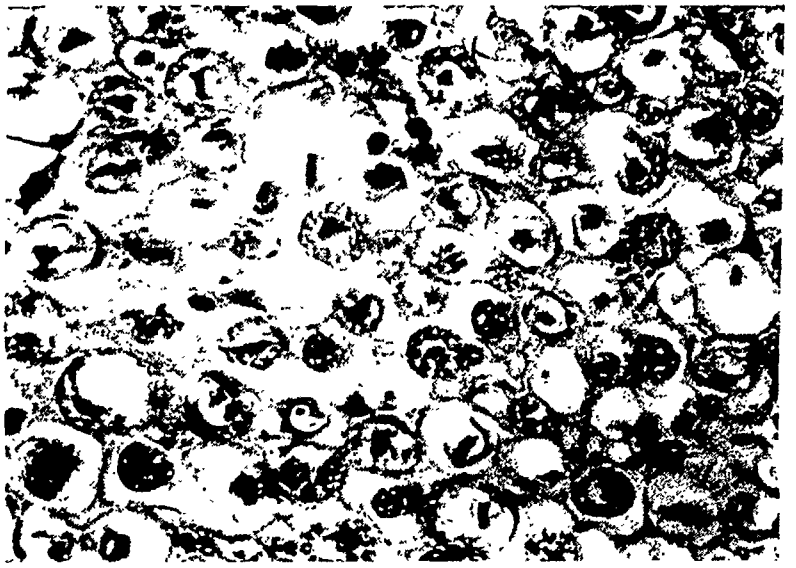


Fig. 29.—Cross-section of the sciatic nerve of a dog with experimental CS_2 poisoning. The myelin is swollen and fragmented. The axis cylinders are unequal in diameter, some swollen while others are extremely thin. (Lewy.)

28, 29). Any portion of the nervous system may be affected, thus accounting for involvement of the sensory (Figs. 30, 31), motor, mental, or psychical functions. *Blood changes* following disulfide exposure and intoxication are for the most part consistent with a secondary anemia, but no typical picture can be adhered to except for a more or less uniform abnormality of the monocytes. These show a relative and absolute increase in numbers, as well as characteristic alteration in the morphology and staining properties. They contain multiple minute neutrophilic granules and often numerous vacuoles. Creskoff[2] termed them "toxic monocytes" and felt that their presence in the blood picture was indicative of a pathological state.

Fig. 30.—Dog 165. After six-weeks exposure to 400 parts per million CS_2, eight hours daily, five days a week. The animal is unable to stand on its forelegs with the back legs supported. The dog is in partial flaccid paralysis. (Lewy.)

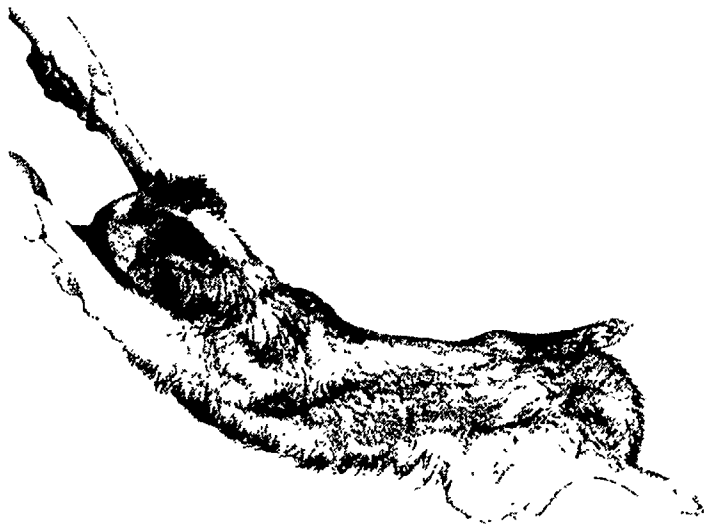


Fig. 31 —Same dog unable to stand on hind legs with the forelegs supported. (Lewy.)

DIAGNOSIS

Signs and Symptoms. *Acute intoxication* is rare in industry. In the mild to moderately severe cases the picture is that of alcoholic intoxication, with headache, nausea, reeling, giddiness, talkativeness, laughing, crying, or stupor. More severe cases may continue through this initial stage into coma and death.

Chronic Intoxication.—The following table is reproduced from the study of 159 viscose rayon workers and taken from the aforementioned Pennsylvania study.

TABLE 10 INDICATED SYMPTOMS OF CARBON DISULPHIDE AND SULPHURETTED HYDROGEN POISONING TOTALED FROM QUESTIONNAIRES OF 159 VISCOSE RAYON WORKERS [3]

I. *Brain:*	
Restlessness	115
Talkativeness	27
Easy excitability	100
Irritability	120
Change of character	66
Apathy	80
Withdrawal	78
Chronic fatigue	118
Depression	82
Listlessness	34
Difficulty of concentration	31
Forgetfulness	37
Partial loss of memory	29
Headache	99
Vertigo	97
Insomnia	109
Day drowsiness	99
Hallucinations: sight, hearing	49
Ideas of persecution	15
II. *Nerves:*	
Hands heavy	50
Hands numb and tingling	57
Weakness of muscles; legs, arms	91
Loss of sensation	18
Difficulty going upstairs	65
Muscular pain	98
III. *Disturbance of Vision:*	
Dimness of vision	65
Cloudiness of vision	64
Narrowing of field of vision	7
Change in color perception	7
Double vision	12
IV. *Digestive System:*	
Loss of appetite	68
Loss of weight	76
Indigestion	92
Pain in stomach	63
Nausea	59
Constipation	91
Diarrhea	12
Vomiting	16
Shortness of breath	34
Pain behind breast bone	31
Abdominal pain	21
Palpitation	23
Ulcer of stomach reported	6
V. *Characteristic Color of Complexion*	38
VI. *Reported Loss of Libido*	50
VII. *Significant Symptoms of Sulphuretted Hydrogen Poisoning:*	
Irritation of eyelids	56
Inflammations of eyelids	72
Pain in eyes	54
Feeling of sand in eyes	68
Photophobia	61
Tearing or burning of eyes	31
Haloes around lights	34

(*a*) *Disturbance of Vision.*—Of this vast and varied symptomatology just outlined, one of the most frequent and earliest disturbances noted is altered vision. Objects become indistinct and appear to be veiled in a color fog. Failure of vision is fairly rapid. In a report of his phase of the investigation Robb McDonald states[2]:

The failure of vision or amblyopia is known to be produced by several exogenous toxins, such as lead, carbon disulphide, tobacco, and alcohol. Fortunately, a differential diagnosis between these poisons by means of special examinations as the determination of the fields of vision, especially the central fields, is usually possible. Carbon disulphide causes a scotoma (a blind area) in the center of the visual field where the vision is most acute. This is called a central scotoma and it has several characteristics that distinguish it from the scotomas due to lead, nicotine, or alcohol. Some of these toxins also cause a contraction of the peripheral field of vision. The loss of vision comes on more rapidly in carbon disulphide intoxication than in any of the other intoxications and also shows more rapid improvement if the source of intoxication is removed.

These changes in the visual acuity are due to carbon disulphide affecting the optic nerve or the visual cells of the retina. Chronic mild inflammation of the optic nerve may cause an enlargement of the blind spot. (This is a small blind area in the field of vision denoting the area where the optic nerve enters the back of the eye. It is present in every eye and must not be confused with a central scotoma which was mentioned above.) In some affections of the optic nerve, an enlargement of the blind spot precedes the development of a central scotoma. In the observations during the viscose rayon survey, approximately one-half of the workers gave a history of some ocular trouble. However, no one was seen in whom diminution of vision could be objectively proven to be due to a central scotoma. Two gave a history very suggestive of having had a central scotoma at some previous time and one was found to have poor central color vision.

Enlargement of the blind spot was found in slightly less than one-quarter of those examined and was found with greater frequency in those employed in the churn room than elsewhere. This phenomenon has not previously been noted in the literature on carbon disulphide intoxication, and may be of significance, when present, in establishing an early diagnosis.

(*b*) *Disturbance of Hearing.*—Batson[2] concluded that the end-organ of hearing was not ordinarily disturbed, but the inability to fix attention by those intoxicated resulted in a pseudo loss of hearing.

(*c*) *Blood Chemistry.*—A study of the blood chemistry of these patients revealed an increased serum cholesterol above the average

and a decreased percentage of esterified cholesterol, no matter what the severity of their intoxication. For a variable period after exposure the blood and urine contained a significant and determinable amount of CS_2.

(*d*) *Composite Symptomatology.*—As this book goes to press, Gordy and Trumper,[3] who deserve much credit for their constant contributions to knowledge of this solvent, augment their previous reports with twenty-one additional cases. The symptomatology noted by them in these cases is as follows: 90 per cent showed encephalopathic features varying from lethargy, anxiety states, and irritability to vertigo. One of these was frankly psychotic; practically 75 per cent showed diminution or loss of libido; 70 per cent revealed varying degrees of neuropathy; and 67 per cent had subjective eye symptoms; more than two-thirds had gastro-intestinal disturbances; more than half, varying degrees of asthenia, headache, muscular stiffness, and dyspnea; and a few had a picture simulating angina pectoris; 55 per cent of these victims had symptoms which pointed to localized lesions of the central nervous system, including parkinsonism; 30 per cent had vasomotor symptoms, and a similar percentage showed blurring of the optic disc.

Differential Diagnosis.—One should be very reticent about accepting a case as one of carbon disulfide poisoning because of its rare use. Alleged cases demand immediate contact with the plant or industry to certify its presence or absence in the patient's working conditions.

Among the aids to differential diagnosis of CS_2 poisoning, the reaction of corneal and pupillary reflexes ranks first.

Corneal Reflex.—Marked decrease or loss of the corneal reflex, especially if bilateral and not accompanied by sensory disturbances in the distribution of the fifth nerve, is an extremely rare event in diseases other than CS_2 poisoning.

Pupillary Reflex.—The same consideration holds true in relation to the pupillary reflex in absence of syphilis, epilepsy, or an intracranial mass lesion.

Neuropathy.—Finally, in a polyneuropathy, especially with predilection for the peroneal nerve, CS_2 poisoning should be considered when other causes have failed to explain the disease. The combination of polyneuropathy with a Korsakoff psychosis gives a picture almost identical with CS_2 poisoning. Strangely enough, alcoholism is rare among viscose rayon workers.

Syphilis and Tabes.—Diminution or loss of patellar and pupillary reflexes, ataxia, shooting pain in the legs, and loss of libido may raise the question of differential diagnosis between cerebrospinal syphili-

or tabes and CS_2 poisoning. However, in uncomplicated CS_2 poisoning the Argyll Robertson type of pupillary reaction and the characteristic reactions in blood and spinal fluid are absent, and loss of vibratory or position sense has not been observed.

Multiple Sclerosis.—Differentiation between multiple sclerosis and CS_2 poisoning, though often impossible in the beginning, is not difficult in a fully developed case. So, also, the differential diagnosis against post-encephalitic parkinsonism and senile paralysis agitans is difficult only in the early stages.

Various Erroneous Diagnoses.—It may best serve the purpose of pointing out how to avoid a wrong diagnosis to enumerate some erroneous diagnoses recorded in various hospitals on histories of patients who were actually suffering from chronic CS_2 poisoning: *ovarian deficiency* because of loss of menstrual period and vicarious severe headache; *pituitary tumor* because of loss of vision, pallor of disks, loss of libido, temporal headache; *mastoid disease* or *tumor of the auditory nerve* because of ringing in the ears, subjective deafness, and vertigo; *thyroid gland disease* because of tremor, profuse sweating, and loss of weight; *tuberculosis* because of loss of appetite, cough, shortness of breath, and night sweats; *blood disease* because of increased number and pathological forms of monocytes.

Importance of Questioning Patients.—It may be difficult to diagnose chronic CS_2 intoxication if the physician *does not ask for* and the *patient does not mention* the hazards of his occupation. The occurrence of CS_2 poisoning and the opportunity for frequent contact with persons suffering from the effect of this poison are obviously linked with a comparatively small number of industrial centers. Many an experienced practitioner and specialist will never actually see a case of CS_2 intoxication. Nevertheless, carbon disulfide is now a recognized industrial poison in this country. It is, therefore, important that every physician question his patients about their past and present occupation and keep himself fully acquainted with industrial hazards and occupational diseases. This cannot be overemphasized.

MEDICOLEGAL ASPECTS

Temporary Disability.—Effects from mild exposure tend to disappear soon after removal from the exposure.

Permanent Disability.—This is not to be expected in the majority of cases. No conclusions have been drawn regarding the psychiatric stigmata of carbon disulfide intoxication. Concerning the peripheral neuropathies, weakness and paralysis of the muscles return to normal gradually when exposure is no longer encountered. Advanced paralysis may take from nine months to two years for recovery. The expectancy

for full recovery from most of the neurological disturbances is good, although from months to several years may be required. This may also be said for visual damage.

Illustrative Cases.—Several patients have presented themselves presumably suffering from exposure to carbon disulfide. In most instances this has been erroneous assumption, as illustrated by Case I.

Case I.—A fifty-six-year-old refrigerator repairman was suddenly seized with severe chest pain, became cyanotic and extremely short of breath. He was taken to a nearby physician by two fellow-workmen who informed the physician that the patient had been exposed to carbon disulfide. The physician administered artificial respiration and then transferred the patient to this clinic. While the patient was being examined, an associate immediately contacted the employer by phone to learn that carbon disulfide was never used in that plant, although sulfur dioxide was, but that this particular patient had not been exposed even to this latter gas. The clinical picture was typical of coronary thrombosis, and the electrocardiogram was confirmatory. Death occurred twenty-two hours later, and the autopsy revealed a fresh thrombosis.

According to the physician, either the fellow-workmen of the patient told him of exposure to carbon disulfide, or he misunderstood what the workmen said. Obviously his treatment was contraindicated in coronary thrombosis. A moment expended to call the plant to ascertain the type of exposure would have been expedient. The case was not compensable. It is used here to emphasize the need for immediate investigation of the hazard and thus avoid faulty assumption.

Case II.—A twenty-four-year-old woman was employed in the printing of "decap" paper. This paper is covered with a gelatinous coating which protects the printing underneath, but which upon wetting reveals the letters or configurations underneath, and the process enables the printing to be transferred to other substances, especially glass. They are designed to transfer printing or figures to windows, windshields, and the like. In the formation of the gelatinous mass, carbon disulfide was used. In order for the process to function properly, a dry room free from air currents was necessary. It appears that this company was newly formed and inexperienced in the process. The girl had been with the organization since its inception, which was five weeks.

Present Illness.—For several weeks she had noticed a loss of appetite, and on several occasions, particularly at the noon hour, she noticed when she went outside to eat her lunch that she seemed lightheaded, was inclined to be giddy, which was noticed by her fellow-

employees. One day at the noon hour she appeared to be unsteady on her feet and "felt drunk." Upon returning to her afternoon work she became slightly hysterical, first laughing and then crying. She was sent home, and upon arrival felt numb, and her limbs seemed heavy. Numerous aspirin tablets were taken to relieve a pronounced headache and pains over both kidney areas. On the following morning she felt greatly improved, the improvement continuing throughout the day. On the second day following the onset, she felt "practically normal," but reported to a physician because she had not urinated since the afternoon or her leaving work. Further inquiry brought out that she had dull pains in both flanks and was depressed mentally.

Past History.—Negative for illness except an appendectomy in 1924; divorced, no pregnancies.

Physical examination revealed a well-built, healthy appearing female. There were no objective findings in the physical examination.

Laboratory Examination.—Kidneys, ureter, and bladder, negative for pathology. A trace of albumin in the urine; no casts or red blood cells. Hemoglobin, 85 per cent; red blood cells, 4,500,000; white blood cells, 8600, with 63 per cent polymorphonuclears.

The author was too little versed in the knowledge of carbon disulfide at the time this patient was seen to subject her to certain other tests. A non-protein nitrogen test was not done, and the eyes were not examined by an ophthalmologist.

Exposure was verified, and the symptoms were classical enough as we then knew them, to accept this case as one due to occupation. The patient made an uneventful recovery within a week and returned to work, but not at the same place of employment.

Case III.—This case was reported by Gordy and Trumper.[1]

Present Illness.—A man, aged forty-nine, foreman in a viscose factory who worked principally in the reeling room, first became ill in April, 1935. Headache, vertigo, rapid loss of weight, and paresthesia in his hands and feet developed. With this there was also insomnia, causeless weeping, and night terrors. He had had aching in his bones for months. For some time he felt that his mind was not working properly. Both his wife and his son had noticed that his actions had not been normal and had told him about his crying and screaming while sleeping and that his body shook all over. His memory was unreliable. He described episodes resembling fugues in which he would find himself at some place and could not remember how he got there. He was unable to drive a car because he was uncertain of himself. He complained of dyspnea and a sense of oppression in the chest. He also had episodes of blindness lasting for a few minutes and also double vision. He was unable to retain a position because of his memory faults. He had a marked diminution of libido.

Examination.—The patient was fairly well built and gave evidence of having lost weight, his weight at that time being 156 pounds (71 kg.). The pupils were regular and reacted to light and to accommodation. The eyegrounds showed slight pallor of the temporal discs and slight atherosclerosis. The cranial nerves were normal except for mild tremor of the tongue. No ataxia or other evidence of cerebellar dysfunction was present. The biceps, triceps, knee, and ankle jerks were present and equal; there were no pathological reflexes and no disturbances in any of the modalities of sensation.

Blood pressure, 150 systolic; 100 diastolic.

Mental Status.—The patient tended to be morose, apathetic, somewhat indifferent, and his thought processes were retarded. He was oriented in all spheres, but his memory was definitely dulled. He found it difficult to place various occurrences in the past in any sort of proper sequence. He had formerly had the reputation of being a first-class worker, with an ability to handle other employees. At the time of examination he had a marked lack of initiative and a sort of child-like dependence on others.

A man who worked with viscose and was subject to chronic exposure to carbon disulfide fumes suffered headaches, vertigo, loss of weight, paresthesias, insomnia, causeless weeping, night terrors, fugues, petit mal episodes, depression, impairment of memory, loss of initiative, and impairment of libido. The neurological examination was negative.

The case was compensable.

TREATMENT

Prophylaxis.—The most important point is prevention of such poisoning. Preventive measures consist primarily of rigid examination of the plants, to insure modern, safe equipment and the presence of adequate ventilation. The storage and pipe conveyance of carbon disulfide must be satisfactory, and explosions should be guarded against. Medical examination of those exposed to this hazard should be at intervals not greater than one month. Men should be taught to report the first intimation of any unusual symptom; and if this solvent is the suspected cause of the complaints, the employee should be at once removed from the hazard. The drinking of alcoholic beverages by those apt to be exposed to carbon disulfide should be discouraged.

Diet.—To date no adequate or specific treatment for cases of chronic carbon disulfide poisoning is known. It would seem likely in view of the similarity of many of these cases to Korsakoff's syndrome, that a diet high in vitamin content with the vitamin B com-

plex as an adjunct would be of value. If vitamin B complex is to be used, the dosages should be relatively large, *e.g.*, from 50 to 60 mg. of thiamine chloride parenterally daily; 200 mg. of nicotinic acid daily; riboflavin, 1 mg. three times per day; vitamin B_6, 20 mg. twice daily parenterally, and so on. Liver extract in dosages similar to those advised in the chapter on "Manganese" might be tried.

Psychiatric and Ophthalmological Attention.—The aid of a psychiatrist will be needed for most of these patients, since a program of mental hygiene will need to be instituted. The ophthalmologist should be consulted on problems related to the eyes.

Exercises for existing muscular weakness, sedation and tonics may be of value. If parkinsonian-like symptoms are present, a trial of those drugs mentioned in the chapter on "Mercury" for control of these manifestations might be made.

The *acute case* will rarely be seen. When such a case does occur, artificial respiration, from 5 to 7 per cent carbon dioxide in oxygen inhalations, and the respiratory and circulatory stimulants mentioned in the chapter on "Petroleum Hydrocarbons" are used.

REFERENCES

1. Gordy, S. T., and Trumper, M.: "Carbon Disulphide Poisoning, with Report of Six Cases," *J.A.M.A.*, *110:* 1543–1549 (May 7), 1938.
2. Group Study: "Survey of the Carbon Disulphide and Hydrogen Sulphide Hazards in the Viscose Rayon Industry," Bull. 46, Department of Labor and Industry, Pennsylvania, 1938.
3. Gordy, S. T., and Trumper, M.: "Carbon Disulphide Poisoning, Report of Twenty-One Cases," *Indust. Med.*, *9:* (May), 1940.

CHAPTER VIII

THE CHLORINATED NAPHTHALENES AND DIPHENYLS

The chlorinated naphthalenes are naphthalenes in which one or more of the hydrogen atoms has been replaced by chlorine.

Occupational Hazards.—In industry they usually occur in mixtures in which more than one chlorinated product is present. In the manufacture of the chlorinated diphenyls, C_6H_6 is converted into $C_{12}H_{10}$, which in turn is chlorinated into $C_{12}Cl_{10}$. The higher the chlorination, the higher the toxicity. These substances are non-inflammable and are resistant to a high degree to heat, moisture, and electricity. Because of this they are used extensively in wire insulation and electrical condensers.

DIAGNOSIS

Signs and Symptoms.—A common reaction to exposure to these chlorinated naphthalenes and diphenyls is an *acneform skin eruption* which is rather characteristic. Systemic reactions are not frequently reported and are not well understood by the general profession. Animal experimentation would indicate damage to the liver, especially if there had been previous liver damage. Drinker, Warren, and Bennett[1] showed that their animals revealed no significant liver changes until extremely high concentrations were used. Prolonged exposure to high concentrations brought about death with severe jaundice in evidence. The liver at autopsy showed central fatty degeneration with necrosis of the liver cells. In these rats which showed the marked liver changes, no other organs were involved, apparently.

Exposure in Presence of Liver Dysfunction.—Greenburg[2] and his associates had the good fortune to study three fatal cases with acute, yellow atrophy in which this exposure existed and was considered the etiological factor. They emphasize the lack of knowledge concerning these substances and note the infrequency of case reports. They pointedly cite the case of a young woman who had been exposed to the chlorinated naphthalenes and had developed a jaundice. After a long, serious illness, the girl, according to her physician, was on her way to recovery and within a few weeks would be able to return to her former work. They intimate that the physician had never inquired into her employment and was unaware of the especial danger of exposure to the chlorinated naphthalenes in one who had had liver

dysfunction. In their three cases they were unable to locate any previous predisposing cause for the liver atrophy. In two of the three cases there had existed previous hepatitis from this exposure. They concluded their investigation with the following recommendations:

1. Persons suffering from typical acneform eruptions should be removed from further exposure.
2. Persons who have had, at any time in the past, jaundice of even a mild type should not work with these substances. They urge that workers who have had typhoid fever, malaria, gallstones, or other diseases known to affect the liver, be forbidden to work in this exposure.
3. Persons receiving arsphenamine or other drugs known to affect the liver should not be further allowed to work with potential liver poisons. (Certainly this is a worthwhile recommendation which might be applied to all workmen engaged in handling the solvents but one which has escaped any emphasis.)
4. Persons working with the chlorinated naphthalenes and diphenyls should not be subjected to chloroform or avertin anesthetics; or persons who have been subjected to such anesthetics should not be immediately returned to their exposure.
5. Pregnant women should be immediately removed from this type of work.

MEDICOLEGAL ASPECTS

Illustrative Case.—The following case[3] illustrates the toxicity of the chlorinated naphthalenes.

Case I.—A seventeen-year-old white girl was admitted to the hospital deeply jaundiced and in a semi-comatose condition. Other than a tonsillectomy, her past medical history was negative. Following one year of high school, she obtained a job, her only one, with a concern manufacturing electrical condensers for radios. Her work consisted in soldering condensers where she was exposed to fumes of trichloronaphthalene and tetrachloronaphthalene. She was also subjected to fumes of the higher chlorinated naphthalenes from the sealing operation conducted in close proximity to her bench.

Present Illness.—This began about five months before admission to the hospital, or approximately two months after beginning work in this plant, at which time she noticed several pigmented areas on her face. These continued to increase in severity and extent, and caused her to visit the Out-Patient Department of the Lincoln Hospital, where sugar was found in her urine. Upon being referred to the skin clinic, a diagnosis of acute catarrhal jaundice was made. Her rectal

temperature at that time was 100 F., and there was a diffuse papulopustular eruption on her face. From then on she suffered from increasing weakness and nausea; the appetite was poor, and the skin became more jaundiced. Headaches developed, but she continued working until seven days before admission to the hospital.

Physical Examination.—The patient gave the impression of being a colored girl. The temperature was 100 F.; pulse, 114; respirations, 26. Careful examination of the face and neck revealed a negroid type of pigmentation beginning in the hair line and extending down to and including the neck. The eyes revealed deep jaundice of the sclerae and the conjunctivae. The skin of the thorax and lower part of the body was a dark brown with a yellowish sub-tinge. Over the abdomen there was a girdlelike area of deeper pigmentation beginning at the level of the umbilicus and extending down to the pelvis. Other positive physical findings were a slight injection of the throat, apical systolic murmur, slight edema of the hands and feet, abdomen distended and tympanitic, liver on percussion small, with tympany extended to the very costal edge.

Laboratory Examination. (*a*) *Blood Count on Admission.*—White cells, 9100; red cells, 4,200,000; hemoglobin, 87 per cent; lymphocytes, 20 per cent; polymorphonuclears, 80 per cent; bleeding time, 4.5 minutes; clotting time, 2.5 minutes; platelets, 220,000.

(*b*) *Blood Chemistry.*—Non-protein nitrogen, 35 mg. (later 45 mg.) per 100 cc.; protein, 6.9 mg. per 100 cc.; urea nitrogen, 20.8 mg. per 100 cc.; creatinine, 2 mg. per 100 cc.; glucose, 88 mg. per 100 cc.; cholesterol, 145 mg. per 100 cc.; calcium, 8 mg. per 100 cc.; CO_2 combining power, 52 vol. per cent; icteric index, 230. Van den Bergh reaction, immediate direct reaction; Wassermann test, negative.

(*c*) *Stools.*—Urobilin present. (*d*) *Urine.*—Specific gravity, 1.008; alkaline; albumin, negative; sugar, a trace; acetone, 1 plus; bile pigment present; urobilin, a trace; a few red and white cells.

(*e*) *Spinal Fluid.*—Pressure normal; cells, 10; lymphocytes, clear; glucose present; globulin absent.

(*f*) *x-Ray examination* of the heart, lungs, and bony framework, negative.

(*g*) *Gastric Contents.*—Antemortem and postmortem examination revealed no arsenic, lead, tin, or antimony. A trace of phosphates was reported, probably from inorganic phosphates ingested before admission.

Clinical Course.—This was steadily downhill. On the evening following admission the patient went into a deep coma with Cheyne-Stokes breathing, pupils immobile to light, deep reflexes greatly depressed, and the liver dulness appeared diminished. The patient died two days after hospital admission.

Diagnosis.—The patient presented a picture which was distinctly puzzling to all who saw her. Consideration was given to:

1. Obstruction to the common bile duct
2. Toxic jaundice possibly due to some dye
3. Obstruction to the inferior vena cava
4. Blood dyscrasia
5. Addison's diabetes
6. Hemachromatosis, and finally
7. Acute yellow atrophy of liver and pancreas possibly due to an unknown industrial poisoning.

Autopsy Findings.—An autopsy was performed at the Medical Examiner's office by Dr. Charles H. Hochman, Assistant Medical Examiner. The report in full was as follows:

Body is that of a white, adult female, well developed and nourished. Cyanosis of lips, ears, and fingernails. Marked pigmentation about eyes, lower lips, neck, and abdomen. General icteric tinge to entire body. No evidence of violence. Some edema about the ankles. Hair is black. Eyebrows black. Brown iris. Conjunctivae and sclerae icteric. Rigor mortis present. Postmorten lividity of dependent parts. Scalp incised and reflected, calvarium removed. Brain found congested. Meninges bile-tinged. On section, no evidence of intracranial injury or hemorrhage. Dura stripped and reveals no evidence of fracture.

Body opened in usual midline incision. Sternum removed. Some remnants of thymus still present. Lungs are free. Bronchi contain a bloody mucus. Mucosa injected, has yellowish tinge. Hemorrhagic infarct about the size of a hen's egg in left lower lobe. This is airless and dark red in color, and firm. There are also similar smaller areas in other lobes. Heart is small and shows some sub-epicardial hemorrhages. Some hypertrophy of left ventricle. Valves thinned. Arteries natural. All are bile-stained. No significant findings in myocardium or coronary arteries.

Esophagus is natural. Stomach is distended, contains some dark material. The rugae are somewhat hypertrophied. The duodenum contains green chyme. The ampulla is patent. The jejunum, ileum, and large gut are natural. Many petechial hemorrhagic areas noted in the mesentery with some enlargement of mesenteric lymph nodes. Liver is small, weight 720 gm. Capsule, wrinkled. Right lobe on section shows areas of yellow surrounded by red areas. Left lobe much firmer although liver cuts firmly throughout. All ducts patent. Gallbladder small. Wall thickened throughout.

Pancreas is natural in size, somewhat congested. Spleen is natural in size. On section, firm, dark red in color. Follicles are somewhat indistinct. Kidneys show swollen cortex. Evidence of parenchymatous degeneration of epithelial cells. Capsule strips easily. No granulation. Degenerative changes in cells of glomeruli only. No glomerulitis. Uterus is small. Endometrium bile-stained. Ovaries are natural in

size; right contains corpus luteum cyst. Bladder wall is somewhat thickened. Mucosa is injected.

Microscopic Examination. (*a*) *Liver.*—From the size of a through-and-through transverse section one suspects that the entire liver was about one-half normal size. Liver segment is very firm and composed of two distinct and peculiar types of tissue: (*1*) that constituting major portion of parenchyma is red in color, firm and elastic in consistency; (*2*) other areas are light yellow and are scattered indiscriminately throughout reddish areas. Yellow areas from one-half inch to two inches in diameter. External surface of liver, judging from this portion, was smooth.

A typical picture of clear sub-acute, yellow atrophy is revealed. Slides show that red area is composed of so-called "red atrophy" in which all the liver cells have disappeared, their place being occupied by their normal supporting stroma, which seems to have been spared, great numbers of blood cells, early fibroblastic proliferation and older scar tissue. In these red areas, numerous bile ducts are seen. These also seem to have been spared destruction. The yellow areas are composed of actively regenerating islands of liver cells.

The toxin evidently has destroyed most of the liver cells, leaving intact their supporting stroma, blood vessels, and bile ducts. The latter seem to be regenerated. Those liver cells spared destruction have regenerated and have formed yellow islands seen in the gross picture. There evidently has been one or more attacks of hepatitis judging from the different ages of the pathological process in various parts of the liver.

(*b*) *Skin.*—Shows an increased density of corium which stains deeply with eosin. Some of the epidermal cells are slightly deeper in corium than normal.

(*c*) *Heart.*—Shows mild parenchymatous myocardial degeneration.

(*d*) *Kidneys.*—Show severe epithelial degeneration involving epithelium of tubules and glomeruli. No inflammatory reaction of glomeruli.

(*e*) *Adrenals.*—Show severe parenchymatous degeneration of cells.

(*f*) *Pancreas.*—Shows intense degeneration of cells of acini and of islets.

Summary.—An unknown toxin has evidently caused a severe diffuse cytolysis involving most of the organs, predominantly the liver. The latter has evidently undergone and recovered from previous attacks of a similar nature.

Too few cases of this type find their way into the literature in such a completely studied manner. It serves not only as a study of the effects of the chlorinated naphthalenes but to reiterate the effect the solvents have upon the body, especially the liver.

TREATMENT

Prophylaxis.—It should be constantly reiterated that the primary treatment is prevention. Ventilation should be proper. As indicated in the foregoing chapter, the recommendations of Greenburg and his associates concerning the type of person to be employed where this hazard exists should be followed. The knowledge of an existing hazard by both the employer and the employee, and the recognition of certain early symptoms by the workman, will greatly reduce actual intoxication. Obviously this is an educational program.

No specific therapy has been evolved for these cases. The acneform *skin lesions* and the *liver damage* are the two symptoms toward which treatment is chiefly directed.

Skin Lesions.—Prevention of the papulopustular skin eruption may frequently be accomplished by installation of showers, and by the formulating of rules requiring a change from street clothing to work clothing at beginning of the day's work, and, following a shower, a change back to street clothing at the end of the work period. The most satisfactory treatment of the skin eruption, once it has occurred, consists of quartz light and *x*-ray therapy, together with mechanical removal of the comedones. The worst cases are treated with quartz light each day for from fifteen to twenty minutes, the less serious ones three times per week, and the mild ones once per week. *x*-Ray therapy should be directed by the radiologist or dermatologist. The pustules should be opened, and these might respond well to the use of sulfathiazole powder or ointment locally. Extreme body cleanliness should be practiced. Cases of very resistant exfoliative dermatitis have been described in which the use of sodium thiosulfate, splenic extract, calcium gluconate, autohemotherapy, roentgen therapy, and numerous baths, powder, and ointments were of little avail.

Liver Disorder.—This may be treated much as described under "Tetrachlorethane," the chief points being a high carbohydrate, high protein diet with adequate fluid intake, and particularly intravenous infusions of 5 to 10 per cent glucose in saline in amounts of from 2000 to 3000 cc. or more daily. The use of calcium and of sodium xanthine experimentally has been of no value in prevention or treatment of the liver damage due to the chloronaphthalenes.[1] This differs, for example, from the treatment of carbon tetrachloride poisoning in which calcium products are of much value.

REFERENCES

1. Drinker, C. K., Warren, M. F., and Bennett, G. A.: "Problem of Possible Systemic Effects from Certain Chlorinated Hydrocarbons," *J. Indust. Hyg. and Toxicol.*, *19*: 283-311 (Sept.), 1937.

2. Greenburg, L., Mayers, M. R., and Smith, A. R.: "Systemic Effects Resulting from Exposure to Certain Chlorinated Hydrocarbons," *J. Indust. Hyg. and Toxicol.*, *21*: 29–38 (Feb.), 1939.
3. *Ibid.*: Taken by Greenburg from the services of Drs. Kenneth Taylor, Edwin Hauser, and Scott Johnson of the Lincoln Hospital, New York.
4. Drinker, Cecil K.: "Possible Systemic Toxicity of Certain of the Chlorinated Hydrocarbons with Suggestions for Permissible Concentration in Air of Workrooms," *J. Indust. Hyg. and Toxicol.*, *21*: 155 (May), 1939.

CHAPTER IX

TETRACHLORETHANE AND TRICHLORETHYLENE

THE vapor from tetrachlorethane ($C_2H_2Cl_4$) is six times heavier than air and is industrially prepared by the action of chlorine upon acetylene.

Occupational Hazards.—It is an excellent solvent for fats, gums, and especially for cellulose acetate. It is by far one of the most dangerous of the chlorinated hydrocarbons. It smells like chloroform, is absorbed easily and slowly, with prolonged after-effects.

Pathology.—In animal experimentation it produces fatty degeneration of the liver, kidney, and heart. In the human, its usual effect is the production of acute yellow atrophy. The outstanding lesions found at postmortem examination are those of acute cirrhosis and atrophy of the liver, fatty degeneration in the kidneys and heart, hemorrhage into the lungs and serous membranes, and edema of the brain.

Hamilton[1] reports a case of a girl making safety frames for goggles with celluloid dissolved in a mixture containing 30 per cent tetrachlorethane. Almost at once she felt nauseated and after four days was forced to stop work because of gastric distress. Following a few days' rest at home she returned to another job but in the same room. She became jaundiced, but continued to work for two weeks and then became rapidly worse, dying in ten days. The immediate cause of death was hemorrhage into the mucous membranes, especially the stomach, which led to the observation that benzene may have been present in the mixture. Autopsy, however, revealed a marked atrophy of the liver.

Diagnosis. *Signs and Symptoms.*—Excessive concentration gives an *initial warning* by lacrimation, salivation, and irritation of the nose. Continuation in the exposure will result in restlessness, general nervousness, dizziness, nausea, or vomiting and coma. In the acute stage of the poisoning, the diagnosis is greatly aided by the blood picture, which shows a progressive increase in the mononuclear cells, sometimes reaching 40 per cent, a slight elevation in the white cells, and a progressive anemia. Experimenters have stated that the narcotic action of tetrachlorethane is similar to chloroform but the hemolytic effect is seven times that of chloroform.

Medicolegal Aspects. *Temporary Disability.*—If the exposure has not been immediately fatal, the non-fatal and the eventually fatal

cases run a similar course, according to Zollinger.[2] This period may be from three days to three months. After this time, in the non-fatal cases, the symptoms gradually decrease, and recovery takes place. Fatal types enter into a second stage of exacerbation of symptoms and finally pass into a third stage of coma and death. Zollinger calls the first stage of from several days to several months the "early prejaundice." The second stage is jaundice without toxemia, whereas the third stage is jaundice with increasingly severe toxemia. He, as well as others, agrees that when jaundice is severe the patient will not recover.

Permanent Disability.—If the exposure is not immediately fatal and the victim survives the non-fatal period of several months, there is no permanent disability, for recovery appears to be complete.

Treatment.—The chief concern in formulating a therapeutic regimen is directed toward the *improvement of liver function.*

Diet.—The patient should be given a diet high in carbohydrate, since this food factor aids in the regenerative process of the injured parenchymatous cells. In addition, the inclusion in the diet of foods high in protein is indicated, since recent research[3-6] indicates a protective action to the liver of diets high in protein. The ability of the liver to deaminize and metabolize amino acids appears to be normal up to the very last stages of hepatic insufficiency. The fat content of the diet should be low; from 200 to 250 gm. of carbohydrate and from 1.5 to 2 gm. of protein per kilogram of body weight should thus be included in the diet.

Infusions of Dextrose.—It is of particular importance when severe or even moderate liver damage is present to administer by intravenous infusion from 5 to 10 per cent dextrose in physiological saline solution or in distilled water, preferably about 3000 cc. daily. It may, in very acute cases, be necessary to give such infusions by continuous intravenous drip.

Vitamin K and Bile Salts.—The use of 2-methyl-1,4-naphthoquinone (synthetic vitamin K) and of bile salts has been suggested. Vitamin K may be given orally or parenterally in doses of from 1 to 4 mg. daily, and the bile salts are administered orally in dosages of from 2 to 3 gm. of desiccated whole fresh bile. Prior to the administration of these substances the prothrombin level of the blood should be determined, and a subsequent check on this reading will indicate whether the vitamin K is of value in the individual case.

On the question of the value of vitamin K in such cases there is considerable disagreement. There is evidence to indicate that if hepatic injury is severe enough, vitamin K is not effective in correcting the prothrombin deficiency.[3] Since, however, little toxic reaction to vi-

tamin K or to whole fresh bile occurs, their trial use is suggested.[7, 8] In cases of hemorrhage, blood transfusion is indicated, and for secondary anemia, if present, enteric coated tablets of ferrous sulfate, 5 grains (0.32 gm.), two tablets three times daily should be used. The intravenous administration of 10 cc. of a 10 per cent solution of calcium gluconate twice daily will be of value. The oral use of 12 gm. calcium gluconate or 8 gm. calcium lactate will supply about 1 gm. of calcium daily. Under normal conditions the daily calcium requirement is from 0.5 to 1 gm.

REFERENCES

1. Hamilton, A.: *Industrial Toxicology*, Harper and Brothers, New York, 1934.
2. Browning, E.: *Toxicity of Industrial Solvents*, Medical Research Council of Industrial Health Research Board, Chemical Publishing Co., New York, 1938.
3. Bollman, J. L., Butt, H. R., and Snell, A. M.: "The Influence of the Liver on Utilization of Vitamin K," *J.A.M.A.*, *115*: 1087 (Sept. 28), 1940.
4. Goldschmidt, S., Vars, H. M., and Ravdin, I. S.: *J. Clin. Investigation*, *18*: 277 (May), 1939.
5. Miller, L. L., and Whipple, G. H.: "Chloroform Liver Injury," *Am. J. M. Sc.*, *199*: 204 (Feb.), 1940.
6. Messenger, W. J., and Hawkins, W. B.: "Arsphenamine Liver Injury Modified by Diet," *Am. J. M. Sc.*, *199*: 266 (Feb.), 1940.
7. Townsend, W. R., and Mills, F. S.: "Hemorrhagic Tendency Associated with Prothrombin Deficiency and Its Treatment with Vitamin K and Bile," *Canad. M. A. J.*, *42*: 541 (June), 1940.
8. Butt, H. R., and Snell, A. M.: *Vitamin K*, W. B. Saunders Co., Philadelphia, 1941.

TRICHLORETHYLENE

Trichlorethylene, belonging to the group of chlorinated hydrocarbons of the aliphatic series, of which chloroform is the best-known representative, is produced from ethylene by the replacement of three hydrogen atoms with chlorine. It is insoluble in water, but mixes freely with alcohol, benzene, acetone, and other organic solvents. It vaporizes easily at room temperatures, is relatively stable in air and non-inflammable, but under certain physical and chemical conditions it may decompose, with the formation of hydrochloric acid. In contact with naked flame, phosgene is formed, as will be illustrated later by a specific instance. Trichlorethylene is fast becoming a popular industrial solvent.

Occupational Hazards.—In drycleaning it is replacing gasoline, naphtha, or benzene since it is non-inflammable, is readily recovered, and no excessive loss occurs. It is also replacing carbon disulfide in the extraction of fats and oils. Some common uses are to obtain oil from olives, corn, cottonseed, and linseed; extracting certain properties from wool, silk textiles, and leather; as a solvent for tar, pitch, and resins; as an admixture with rubber cement for adhesive

purposes (crepe soles); as an insecticide; and on a large scale as a solvent for garbage, tankage, and bones.

Pathology.—Postmortem examination of a victim to sudden, *severe exposure* may reveal nothing of note. Several such negative autopsies have been so reported. Animal experimentation and the tissue of a number of humans reveal generalized congestion of all organs. Edema of the lungs and petechial hemorrhages occur. If death is delayed several days, fatty degeneration of the liver, kidneys, and heart is invariably present.

Chronic exposure results primarily in affecting the central nervous system. By searching the literature the writer found the following lesions reported by different observers: retrobulbar neuritis, neuromyelitic complex, paralysis of the hyoglossal nerve, polyneuritis of the limbs, amblyopia with central scotoma, paralysis of the sensory fibers of the fifth nerve, partial or complete destruction of vision, and paresthesia of different areas. Atrophy of the liver is not frequently reported.

DIAGNOSIS

Signs and Symptoms.—The initial effects from small, minute dosages are similar to those from chloroform, namely giddiness, excitement, or confusion, to be followed by narcosis or death if the exposure is intensified or prolonged. Constant exposure of the skin results in extraction of the fat from the skin and subsequent cracking, dry dermatitis. Stuber[1] collected 284 cases of poisoning by trichlorethylene, 202 being acute and 84 chronic. Of this group 25 were fatal. She lists the feeling of drunkenness, confusion, mental dulness, nausea, and vomiting among the more frequent symptoms. Of the fatal cases one patient supposedly died of phosgene poisoning. General fatigue, loss of appetite, dyspnea, and unexplainable pain in the chest are additional complaints. *Psychic* disturbances with epileptoid seizures have been reported. As mentioned under "Pathology," partial or total blindness is a common sequela.

MEDICOLEGAL ASPECTS

McCord[2] established 10,000 parts per million parts of air as definitely narcotic to man.

Temporary Disability.—From mild exposure, recovery may occur within a few hours or a few days. In severe cases, recovery from symptoms of headaches, dizziness, nausea, paresthesia, and the like, may be expected after a few weeks to several months.

Permanent Disability.—Permanent disability is usually the result of an involvement to varying degrees of the optic and trigeminal

nerves. Ratings should not be made until the condition shows evidence of being stationary. However, the reader is warned against hastily accepting an eye disability as one resulting from undue exposure to trichlorethylene until this source has been proved as the cause to the exclusion of all other factors. With the increasing use of this solvent it can be foreseen that many victims of optic atrophy will assume the cause as occupational and relate it to some previous exposure to trichlorethylene. An equitable decision on the part of the physician will require careful evaluation of the occupational factors as well as elimination of the common causes of optic atrophy, such as tabes, paresis, atrophy following skull fracture, focal infection, diabetes, hypertension, and other non-occupational conditions.

Illustrative Cases:

Case I.—A young man presented himself at the clinic with an extremely dry dermatitis involving the palmar and dorsal surfaces of both hands, as well as the flexor and extensor surfaces of the lower third of the arms. Numerous fine cracks were in the skin. He had been under treatment for epidermophytosis without success. Inquiry revealed that for the past several months the involved portion of his body was exposed to trichlorethylene.

The skin reaction in this case was typically one where the tissue had lost its normal oil content. It was known that the solvent he used could do this and also that no other cause for the condition was apparent. The case was, therefore, considered of occupational origin. The condition cleared after he changed his occupation.

Case II.—Two Negroes were assigned the task of cleaning machinery parts in a large tank containing trichlorethylene. One of these was but slightly exposed since he was required to carry and stack the parts after they had been cleaned. The other man worked over a tank of this solvent in the far corner of the plant where ventilation was poor. This was the first day this work was undertaken, and after about two and one-half hours, the Negro engaged in the actual cleaning fell over onto the floor unconscious. Rushed to the hospital, he arrived in a semi-comatose state. He could be aroused but could not answer questions. There was a two-inch gash over the right temporal area. The patient's partner described the work these two had been doing and volunteered the information that the patient complained of being dizzy and "acted drunklike." Further examination disclosed profuse perspiration; deep, slow respirations, which were increasing in frequency as the examination proceeded; heart rate of 86; blood pressure, 102 70. While the examination was being conducted, the plant foreman was reached by phone and after slight delay informed us that

the solvent was trichlorethylene. Oxygen was then administered. The patient became mentally clear within a half hour only to vomit frequently for the next several hours. During this time he complained of severe headache and epigastric pain. Subsequent laboratory investigation was not significant. He was discharged from the hospital in three days and returned to work at the end of ten days, free from symptoms.

Case III.—McNally[4] reports a case of narcosis in a man following five days' exposure to trichlorethylene. On the fifth day of exposure the patient "began to feel sick, went home and slept through the rest of it, all of the following day and until two o'clock in the morning of the third day." Among the various things noted in the physical examination were nystagmus, enlargement of the blind spot, injection of the throat, congestion of the lungs, and marked dyspnea. The patient made an uneventful recovery. McNally cites this case as one of phosgene poisoning resulting from the decomposition of trichlorethylene by the gas flames used to heat the solvent.

TREATMENT

Prophylaxis.—From the standpoint of prophylaxis, several points are important. Addiction to this solvent has been reported by numerous investigators, and it is known that men will at times voluntarily inhale it. For this reason, rotation of men should be practiced where this substance is used for long periods. In degreasing plants where heat is used, acid-proof flues are necessary to carry off the fumes to the outside. If such flues are not provided the gas heating process should be condemned. In the general use of trichlorethylene, proper ventilation should be strictly enforced at all times, and the skin should be protected by clothing and gloves as nearly as possible. It is well to apply ointments of oil or fat, for example a mixture of equal parts of vaseline and rose-water ointment, to areas such as the hands and arms, which are liable to be most exposed.

Dermatitis.—For patients who present a dry, fissured type of dermatitis, the chief therapy is similar to that suggested in those measures to be used in prophylaxis. The patient should be removed from exposure to the offending solvent until the dermatitis has cleared, and in quite a few cases it will be best to advise him to change his occupation, although careful protection of exposed parts of the body may prevent recurrence of the dermatitis. For the treatment of the acute dermatitis, simple ointments, such as rose water with 1 per cent phenol and 0.5 per cent menthol, may be of value.

The Acutely Ill Patient. *Respiratory Stimulants.*—These are needed. Here, the most valuable of all will be inhalations of a mixture of from 5 to 7 per cent carbon dioxide with oxygen. When such in-

halations are not available, artificial respiration by the Schaefer method must be used, and later if pulmonary edema is present, the administration of oxygen either by a tent or intranasal apparatus will be of value. It should, however, be remembered that oxygen in itself is not a respiratory stimulant, and when used without the addition of carbon dioxide may actually depress rather than stimulate respiration. The intravenous or intramuscular injections of coramine, 1.5 cc., or the intramuscular injection of metrazol, from 1½ to 4½ grains (0.1 to 0.3 gm.), are of questionable value in treatment of the respiratory failure, although their use has been advised.

Shock.—They may, however, be of definite value if shock is present. For treatment of this, from 1 to 2 cc. of camphor in sterile oil, or of caffeine sodium benzoate, 7½ grains (0.5 gm.), may be used. Of much greater value will be the use of transfusions of citrated serum and the intravenous infusion of 1000 cc. of 10 per cent dextrose in saline.

Diet.—Following the very acute period a soft diet and normal fluid intake may be resumed, but if symptoms of nausea and vomiting persist the use of from 2000 to 3000 cc. daily of 10 per cent dextrose in physiological saline is advisable. The administration of thiamine chloride (vitamin B_1) in dosages of from 30 to 60 mg. daily, at first parenterally, is suggested in treatment of the neurological manifestations. During the convalescence a high caloric diet with vitamin adjuncts should be given.

Exposure to Phosgene.—If the acutely ill patient has been exposed to phosgene resulting from the decomposition of trichlorethylene by gas flames, he may present a somewhat different picture, with pulmonary edema, dyspnea, and cyanosis playing prominent parts in the picture. Here again respiratory and circulatory stimulants are indicated. In addition, venesection of 500 cc. may be of value in treating the pulmonary edema. During the first World War intratracheal medication of from 1 to 3 drachms of the following mixture two or three times per day was found to be beneficial after the very acute stages had passed: 5 per cent each of guaiacol, camphor, and menthol in liquid petrolatum or olive oil. No instances of lipoid pneumonia from the use of this preparation are mentioned.

The Gray Stage.—During the acute phase a syndrome may occur, referred to in the findings of the chemical warfare service as the "gray stage," in which pronounced pulmonary edema, almost utter inability to breathe, and shock are seen. In such a stage the temperature falls, and there is a marked increase in blood concentration necessitating oral and intravenous administration of physiological saline solution. A fine degree of medical judgment is needed to balance the need for

venesection on the one hand and for decrease in excessive blood concentration by administration of fluids on the other.

REFERENCES

1. Stuber, K.: "Gesundheitschädigungen bei der gewerblichen Verwendung des Trichloräthylenes." *Arch. f. Gewerbepath. u. Gewerbehyg.*, *2*: 398, 1931.
2. McCord, C. P.: "Toxicity of Trichlorethylene." *J.A.M.A.*, *95*: 409, 1932.
3. McNally, Wm. D.: "A Case of Phosgene Poisoning." *Indust. Med.*, *6*: 539–544, 1937.

CHAPTER X

NITROBENZENE, DICHLORODIFLUOROMETHANE, AND ETHYLENE DICHLORIDE

NITROBENZENE

While nitrobenzene ($C_6H_5NO_2$) poisoning is not a frequent industrial condition, it occurs often enough to warrant a short discussion in a book of this type. In general, its action is similar to the other nitro and amido compounds. Early in the literature it was pointed out that the amido compounds affected the blood chiefly, but in addition the nitro compounds involved the central nervous system.

Occupational Hazards.—Nitrobenzene is used in the manufacturing of shoe dyes, floor polishes, perfumes, flavoring extracts, explosives, and the anilines.

Pathology.—Icteric pigmentation of the skin to deep jaundice, acute yellow atrophy of the liver and necrosis of liver cells, degenerative changes in the other organs, and ecchymosis of the membranes of the intestinal tract are common findings.

Blood Picture.—Blood studies reveal *anemia*, with alteration in the size, shape, and staining qualities of the cells, as well as paleness, fragmentation, and polychromatophilia. Early spectroscopic examination of the blood will reveal a methemoglobin or lines between a methemoglobin and oxyhemoglobin. Later, if cyanosis is deep, this cannot be demonstrated. In the acute exposure, if death has not occurred, the cells regenerate, and the blood picture is much like that of *pernicious anemia*. The white cells are usually increased. In chronic poisoning these white cells may decrease below the normal with a relative increase of the lymphocytes. Malden,[1] in his study of the workers in a factory where aniline and benzene were made, noted that in the slower forms of poisoning there was a stimulation of the red blood cells with a decrease in the hemoglobin. He commented on the change in the size of the cells and their stippling qualities and considered *stippling* as characteristic of aniline poisoning as in lead poisoning. If death is immediate, there are no characteristic pathological changes.

Signs and Symptoms.—In a *severe exposure* death may be preceded by fast, irregular breathing, weak, thready pulse, marked cyanosis, convulsions, and coma. In *mild poisoning* the face is flushed, and there may be severe headache, burning of the throat, tightness in the

chest, dizziness, disturbance of gait, and diminished or cloudy vision. *Chronic poisoning* is said to occur and is marked by languor, drowsiness, dyspnea, mild cyanosis, and sometimes diminished vision.

Medicolegal Aspects.—*Temporary disability* is usually short but, of course, is dependent upon the degree of intoxication and the complications which have arisen.

Permanent disability is rare. When present it is usually due to an altered vision. Bladder tumors may result from exposure to the amido compounds, but they are not attributed to the nitro group.

Illustrative Case:

Case I.—A chemist's assistant dropped a can of nitrobenzene, spilling it on his lower legs and saturating his trousers. He started to pick up the can, but soon fell forward onto the floor. He was rushed to the hospital without having his clothes removed. Upon arrival he was unconscious. Respirations, at first rapid, became slow and irregular. The pulse was difficult to feel, the heart tones distant and weak. Cyanosis was marked. A sample of blood was of a deep-brown color. Death occurred an hour and a half after the accident. Postmortem examination revealed nothing of note.

The man had been in good health and two weeks before had passed a preemployment examination. It was definitely established that the substance was nitrobenzene. The symptomatology, sudden death, and character of the demise were in keeping with the known reactions to acute exposure to this agent. The case was compensable.

It should be emphasized in cases such as this, or the one cited under "Phenol," or any other type of saturation from a noxious substance, that *all clothing be removed* from the victim immediately. Such a procedure obviates the possibility of further absorption.

Treatment. *Immediate Measures.*—In acute poisoning the contaminated *clothing should be at once removed*, and areas of the skin on which the substance has been spilled should be cleansed with alcohol. Gastric lavage with water containing epsom salt, which delays absorption, is performed, although as in phenol, mercury, and arsenic poisoning, the lavage is not of as much value in the industrial case as in that of poisoning by ingestion, which is more apt to be seen in private practice. *Oils*, *milk*, and *alcohol* should not be given by mouth since they tend to favor absorption.

Prevention of Respiratory and Circulatory Failure.—Inhalations of carbon dioxide, from 5 to 7 per cent, in oxygen are indicated for respiratory stimulation, and coramine, 1.5 cc., metrazol, from 1½ to 4½ grains (0.1 to 0.3 gm.), caffeine sodium benzoate, 7½ grains (0.5 gm.), or camphor in oil, from 1 to 2 cc., may be given intra-

muscularly for respiratory and circulatory stimulation. Intravenous infusions of from 5 to 10 per cent dextrose in physiological saline solution are indicated, and transfusion (350 to 500 cc.) may be necessary. If acidosis is present, it should be treated as described under "Methyl Alcohol." Epsom salts, from ½ to 1 ounce (15 to 30 gm.), is usually given orally.

Hepatic Inquiry.—Following the acute stage, treatment directed at correction of the liver damage may be necessary, and this should follow pretty closely that outlined under "Tetrachlorethane." If a macrocytic type of anemia develops, liver extract, from 15 to 30 units per day for the first three or four days and then at intervals of from 5 to 10 days, is given.

REFERENCES

1. Malden, W.: Some Observations on the Conditions of the Blood in Men Engaged in Aniline Dyeing and the Manufacture of Nitrobenzene and Its Compounds," *J. Hyg.*, 7: 672–685, 1907.

DICHLORODIFLUOROMETHANE

This substance is known to the trade as "freon" or "F-12." It is the result of experiments conducted by refrigeration engineers to find a less noxious refrigerant. It is a clear, water-white liquid, boiling at −21.6° F. Experiments conducted by the United States Bureau of Mines, with dogs, monkeys, and guinea pigs exposed to this gas, permitted this body to conclude that injury or ill effects from this refrigerant were remote possibilities. To our clinic has never come a patient alleging any ill effects from this gas, but there were reported to the writer two deaths supposedly resulting from a heavy exposure.

Two men entered a fairly small room, which had been kept closed, to test the presence of this gas. They were immediately overcome and died. I was unable to check the veracity of this report. The engineer who reported this instance claimed it was due to a dense, highly concentrated collection of this gas. Its presence is detected by burning a halide light against a copper plate. If freon is present, the blue light turns to green.

Brief mention is made of this gas, not because of the likelihood that the reader will see many cases like the above, but rather to inform him of the remote possibility of ill effects.

ETHYLENE DICHLORIDE

Occupational Hazards.—Ethylene dichloride ($C_2H_4Cl_2$) finds its usefulness chiefly as a solvent for fats, as a thinner for lacquers, as a degreaser in drycleaning, and as a fumigant for moths, weevils, and the like. It is frequently combined with carbon tetrachloride (see case

under "Carbon Tetrachloride"), and is of approximately the same order of toxicity as trichlorethylene.

Signs and Symptoms.—Following acute exposure, dizziness, nausea, vomiting, weakness, trembling, abdominal cramps, diarrhea, drowsiness, and slight breathlessness may be noted. In addition, there may be evidence of irritation of the nose and eyes, ataxia, degeneration of the liver, nephritis, and perivascular hemorrhages of the brain. It appears to have a greater selective action upon the kidney than most of the solvents. Opacities of the cornea have occurred due to the infiltration of lymphocytes and connective tissue cells. In animal experimentation these usually clear within several months.

Treatment.—Treatment consists of general supportive measures as indicated with the other solvents and of similar prophylaxis. The use of a high carbohydrate, high calcium diet has been of definite value. The intravenous administration of 10 cc. of 10 per cent calcium gluconate to the patient shortly after his admission to the hospital has relieved markedly the epigastric cramps and vomiting. This presumably is because of the relaxing effect on smooth muscle of the calcium carbonate. As in treatment with the other solvents, it will be valuable to use intravenous infusions of from 5 to 10 per cent dextrose, in physiological solution of saline if chloride loss following vomiting is present, or in distilled water if it is felt that the salt content will increase renal damage, in amounts varying from 2000 to 3000 cc.

CHAPTER XI

FLUORINE AND CHLORINE

FLUORINE

Fluorine is not a constituent of the human body at birth, but due to its inevitable ingestion and incomplete elimination, it does become present in later life. The substance is widely found in nature, being observed in excess quantities in water in certain portions of the country,[1] in ores and minerals, and in soil.[2] Its two chief components are sodium fluoride and hydrogen fluoride.

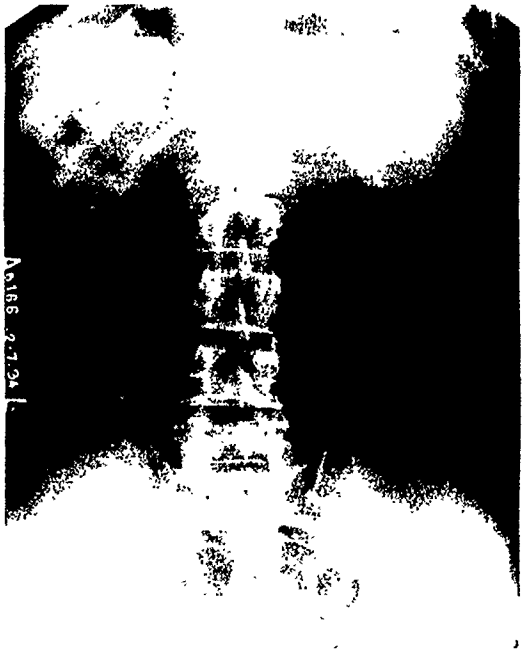


Fig. 32.—Normal spine of man. (Bishop: *Am. J. Roentgenol.*, May, 1936.)

Occupational Hazards.—Art-glass workers, brewers, bleachers, dyers, etchers, glass-finishers, aluminum-extractors, insecticide-manufacturers, and those engaged in the mining of various ores and minerals are liable to this hazard. Cryolite is a source of aluminum, alum, and caustic soda. It is a fluoride of aluminum and sodium, containing

as much as 50 per cent of fluorine. In the United States the phosphate rock from which superphosphate fertilizers are obtained contains as much as 4 per cent fluorine. Rock phosphate is a source of phosphate for baking-powder. Fluorine forms a part of numerous insecticides and fumigating preparations.

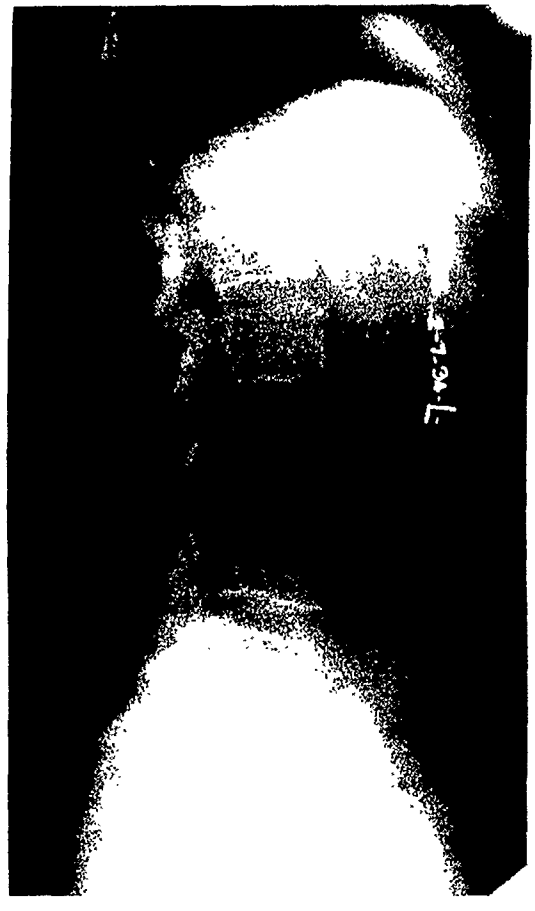


Fig. 33.—Normal spine of man. (Bishop: *Am. J. Roentgenol.*, May, 1936.)

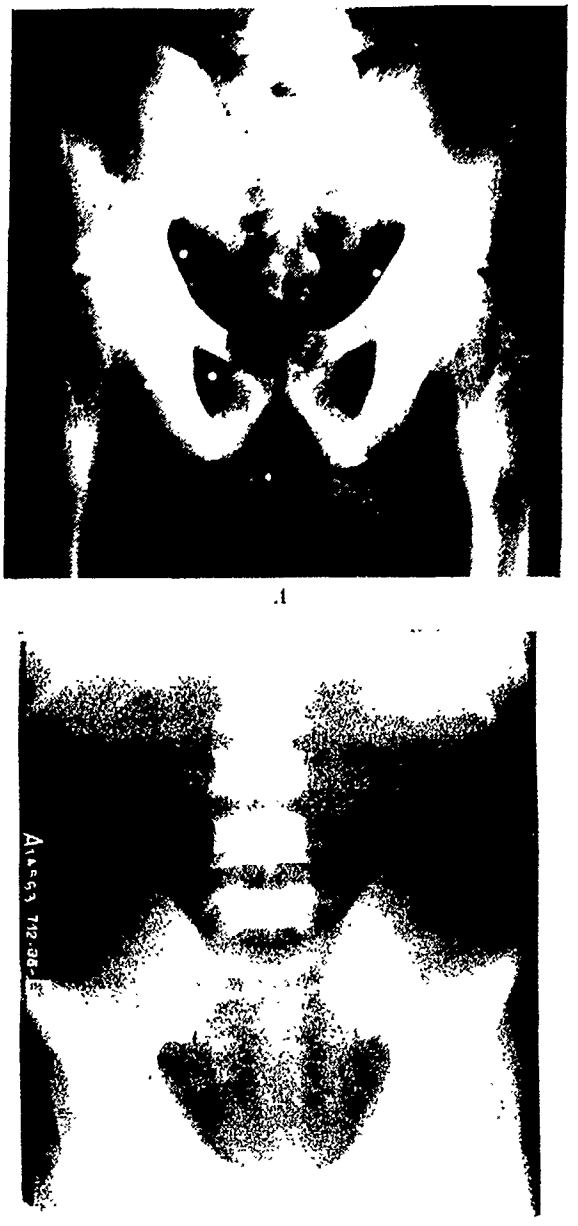
Signs and Symptoms.—In industry action is usually *local*, causing degrees of injury varying from slight burns to corrosive, destructive lesions. It may affect the eyes, the upper respiratory tract, the lungs proper, the intestinal tract, liver, spleen, and kidneys. Vomiting, diarrhea, shallow respiration, convulsions, and coma have been reported. *Fatal doses* diminish the blood calcium and damage the vasomotor and respiratory centers. *Chronic poisoning* causes mottling of the

teeth, gastric and nervous disturbances, and loss of weight. The *x*-rays reveal marked changes in the *bones*. There is a disappearance of normal bone structure with a replacement of an almost milky white opacity, the outlines of which lack sharpness.

Bone Changes.—Bishop[2] quite recently reviewed the available literature concerning the roentgenographic study and added a case of his own (Figs. 32–37). The first observations were made of a group of cryolite-miners who were being studied for silicosis. It was noted that these men had general ill health, although length of exposure was only

Fig. 34.—Bone changes of chronic fluorine poisoning. Note density of ribs and scapulae and the enlargement of the inferior ridges of the ribs, posteriorly. (Bishop: *Am. J. Roentgenol.*, May, 1936.)

ten years. In addition to thirty-nine of the group of seventy-eight showing first- or second-degree silicosis, over half of the group presented definite bone changes. The changes varied from a "fleecy thickening of the bone lamina and an increase in the whiteness of the bone shadows on the roentgenogram to an actual opacity of the bones and calcification of the ligamentous attachments." These bone changes have been mentioned here because they might, at some future time, enable the reader to recognize what otherwise might be passed over as an obscure condition and also to suggest that a roentgenogram of the bones might prove a valuable confirmatory factor in suspected cases.



A

B

Fig. 35.—*A*, Bone changes of chronic fluorine poisoning. Compare with *B*. Note increased bone density, normal bone trabeculation, and spicule formation along anterior iliac margins and external pubic borders. *B*, Osteoplastic metastasis of carcinoma of prostate. Note mottled appearance due to areas of bone absorption adjacent to areas of sclerosis. Normal bone architecture has been lost. (Bishop: *Am. J Roentgenol.*, May, 1936.)

Tests.—There is no simple test to determine the presence of this substance in the chronic case. If an *acute case* results from ingestion, the stomach contents can be examined for the presence of fluorine. Excessive amounts can be determined post mortem by comparing the fluoride present in the bones with the normal known range.

Fig. 36.—Osteoplastic metastasis of carcinoma of prostate, same case. Note mottled appearance of vertebral bodies, the crush fracture, and the absence of dense osteophytes. (Bishop: *Am. J. Roentgenol.*, May, 1936.)

Medicolegal Aspects.—There has been too little industrial experience with this substance to form a basis for an estimation of either the expected *temporary* or *permanent disability*. Aside from its reaction on the bones, nothing is known about its effect on other tissue. It appears that the fluorine salts are deposited in bones with other

calcium salts. In the body metabolism, that part of fluorine which is not combined with calcium is eliminated. An adequate calcium intake prevents toxic symptoms. Hence the reaction in one person may be entirely at variance with that in another. The majority of cases of fatal poisoning have been those of accidental ingestion, or occasionally where taken with suicidal intent. Industrially, there are numerous cases of local injury to the tissue resulting in temporary burns without permanent damage.

Fig. 37.—Osteoplastic metastasis of carcinoma of prostate, same case. Compare with Fig. 34. Note mottled appearance and lack of widening of ribs. (Bishop: *Am. J. Roentgenol.*, May, 1936.)

Illustrative Cases:

Case I.—We have not seen this case duplicated in the literature. The patient was admitted to the surgical service of Dr. Harry Alexander, with a history of having fallen while carrying a small bottle of hydrogen fluoride. The bottle broke upon the cement floor and splashed the victim on his face, both hands, left arm, and left thigh. One-half hour after the accident the patient was admitted to the hospital, and it was noted that all involved areas had burns of a second degree, but the fingers of the left hand were blanched. Within two hours, the distal portions of all fingers were dusky. The following

morning they were definitely gangrenous in appearance. In spite of treatment, subsequent amputation of all the distal phalanges of the left hand except the thumb was necessary. The lesions elsewhere healed without complication.

The caustic or corrosive action of fluorides upon tissue has been frequently mentioned but there exist no case histories of gangrene following contact with hydrogen fluoride. In this case a destructive endarteritis occurred. There was no question of the occupational origin. Permanent rating for the loss of a portion of the fingers was allowed.

Cases of Acute Poisoning.—Hickey[1] reports a case of a girl, ten years old, who was given by mistake a teaspoonful of sodium fluoride for potassium and sodium tartrate. She died soon afterwards. McNally[2] reports a woman who died soon after eating an omelet in which roach powder had been used instead of starch. This same author cites a case in which a woman took a white powder for a laxative and died four hours later. Her physician ascribed death to carcinoma. Six weeks later the body was exhumed, at which time gross pathological change in keeping with fluorine poisoning was noted and fluorine in excessive amounts was found in the bones.

Treatment. *Combating Calcium Deprivation.*—Since the mechanism of fluorine toxicity consists of a calcium deprivation, treatment should primarily be designed to correct this and thus render the fluorine inert. In the acute case of poisoning, gastric lavage with lime water or a weak solution of calcium chloride, together with from 10 to 20 cc. of 10 per cent calcium gluconate intravenously from once to three times a day is used.

Respiratory and Circulatory Complications.—If cyanosis is present the administration of oxygen by an intranasal tube or by a tent is indicated; and respiratory stimulants may be needed, such as camphor in oil, from 1 to 2 cc., intramuscularly; carbon dioxide, 7 per cent, in oxygen as inhalations; coramine, 1.5 cc., intramuscularly or intravenously; or metrazol, from 1½ to 4½ grains (0.1 to 0.3 gm.), intramuscularly or intravenously. The latter two or adrenalin, 1 cc. of a 1:1000 solution, intramuscularly are indicated for circulatory failure.

Shock.—The use of 1000 cc. of 10 per cent glucose in saline given slowly intravenously will be of value in combating shock, particularly if pulmonary edema is absent. The patient is, of course, kept warm with blankets and hot-water bottles.

Sedatives in Complications.—Codeine, from ½ to 1 grain (0.0325 to 0.065 gm.), repeated every four to six hours, is at times needed to allay violent coughing. The codeine may be given in any of a number

of soothing cough syrups. Dilaudid hydrochloride, from $^1/_{32}$ to $^1/_{16}$ grain (0.002 to 0.004 gm.), given hypodermically or by mouth may also help in this regard, as do morphine sulfate, from $^1/_6$ to $^1/_4$ grain (0.011 to 0.016 gm.), and pantopon, from $^1/_6$ to $^1/_3$ grain (0.011 to 0.022 gm.). These sedatives and one of the barbiturates may be needed to control convulsive seizures.

Bronchial Asthma.—If symptoms of bronchial asthma appear, the use of one of the following is indicated: ephedrine sulfate, $^3/_8$ grain (0.024 gm.), given every four to six hours; neosynephrin, from 0.25 to 0.5 per cent solution used in a nebulizer; aminophyllin, $1^1/_2$ grains (0.1 gm.), every four to six hours, or $7^1/_2$ grains (0.48 gm.) in 20 cc. of distilled water, given intravenously slowly over a five-minute period; or epinephrine, from $^1/_2$ to 1 cc. of 1:1000 solution hypodermically, or as inhalations of a 1:1000 solution from a nebulizer.

Obstipation and *constipation* may need attention; and if *vomiting* is severe, the further administration of from 5 to 10 per cent dextrose in physiological saline solution (1000 to 3000 cc. daily) is used. No established treatment exists for *stiffness of the body* due to bony and ligamentous changes, but it appears that on gradual elimination of the fluorine considerable reduction takes place in osseous overgrowth.

Treatment for Burns.—It is suggested that the burns be treated as described by Jones.[6] The burned area is thoroughly washed with or immersed in a warm saturated solution of sodium bicarbonate. Then an ointment prepared from thorough mixing of two parts medical paraffin or glycerin with one part of magnesium oxide is massaged into the burned area, and a dressing of the same ointment is applied; 2 cc. of sterile 10 per cent calcium gluconate solution is injected into and under all areas showing whitened skin. The magnesium oxide ointment dressing is renewed night and morning for five or six days. After this period boroseptic ointment is an efficient and soothing application, and healing can be completed by dressing with ichthyol ointment. The eyes, if involved, should be irrigated for at least an hour with normal saline solution, and then the ointment described above should be applied.

REFERENCES

1. Smith, M. C., Lantz, E., and Smith, H. V.: *Tech. Bulletin No. 32*, Arizona Agriculture Experimental Station, June 10, 1931.
2. Dean, H. T., Dixon, R. M., and Cohen, C.: "Mottled Enamel in Texas," *Pub. Health Rep.*, *50*: 424–442 (March 29), 1935.
3. Bishop, P. A.: "Bone Changes in Chronic Fluorine Intoxication; Roentgenographic Study," *Am. J. Roentgenol.*, *35*: 577–585 (May), 1936.
4. Hickey, M.: *Bull.*, Massachusetts Board of Health, 341, 1911.
5. McNally, Wm. D.: *Toxicology*, Industrial Medicine Publishing Co., Chicago, 1937.
6. Jones, T.: "Treatment of Hydrofluoric Acid Burns," *J. Indust. Hyg. and Toxicol.*, *21*: 205 (June), 1939.

CHLORINE

Chlorine is a greenish yellow gas, not combustible in oxygen, burns in hydrogen, and in the sunlight combines to form hydrochloric acid. It is produced largely from common salt by the electrolytic process and is sold in containers for commercial purposes.

Occupational Hazards.—Its greatest use is in those trades where a bleaching process is needed. It is also found in the manufacturing of nitric acid, in the dye industry, in paper (bleaching) and textile works, and is used widely in the making of disinfectants.

Signs and Symptoms.—Severe dosing causes suffocation, which may be short, or the gas may affect the nervous system producing unconsciousness or death. Following the first effects in the more severe exposures, the entire respiratory tract may develop edema with pneumonia developing within a few hours. While serving with an evacuation hospital during the first World War, the author noted congestion of the nose and throat among patients, with increased redness of the tissue. Scattered moist râles were frequently present. Pneumonia occasionally ensued, but most of these patients recovered. Sloughing of the tissue has been reported, but a recent communication with one of the officers in charge of our gas ward states that he never saw this occur.

Common Findings in Industrial Use.—In industry, the severe or tragic effects from this gas are rare. The average case is one where a whiff of the gas has produced coughing, and a sensation of an inflamed throat or burning "deep in the chest." These men usually recover within a few hours or a day. Instances are known where persons have been overcome and rendered unconscious only to recover within a short time without any ill effects. Chronic exposure to chlorine has been reported to have produced opacities of the cornea and perforations of the nasal septum. Like chlorine, bromine chiefly affects the respiratory system. It is rarely an industrial hazard and needs no further comment.

Medicolegal Aspects.—*Temporary disability* is usually short, a few days at the most. A complication, such as pneumonia, if it arises within a few days, is compensable, and the entire period of disability due to the complication is compensable.

Permanent disability is extremely rare.

Treatment. *War Use versus Industrial.*—The author has had experience with two sources of chlorine gassing: one while in the first World War and the other from industry. In the first instance, the victims were more severely affected because they could not usually withdraw from the hazard. In industry, there occur but few severe exposures. Men are warned of the presence of this gas and immediately

withdraw. If severe affection occurs, treatment is directed to the respiratory tract in order to relieve the irritation and the congestion.

Immediate Measures.—The patient severely gassed with chlorine should be removed from the toxic atmosphere promptly, and all constricting clothing about the neck should be loosened. He should be kept quiet in a recumbent position and should be wrapped with warm blankets while hot-water bottles are applied. Oxygen should be administered in all cases. It is considered best not to wait for cyanosis to develop, since the administration of oxygen will often prevent cyanosis and relieve the pain of deep inspiratory effort. Venesection of from 400 to 600 cc. is an extremely valuable measure and should be performed early on all patients who have been exposed to heavy concentrations; but, as already indicated, such exposures in industry will be very rare. The early use of such phlebotomy often prevents development of edema of the lungs, and if used later often relieves pulmonary edema and cardiac embarrassment. The early intravenous administration of 10 per cent dextrose in physiological solution of sodium chloride may be combined with venesection to combat shock and reduce viscosity of the blood. As with phosgene poisoning, venesection is contraindicated if the gray stage of anoxemia with pallor, collapse, and rapid, thready pulse is reached.

Relief of Pain and Excitement.—Morphine sulfate is not to be used if pulmonary edema and respiratory embarrassment have ensued. Some advise, however, its use for relief of pain and excitement soon after injury. Atropine, epinephrine, digitalis, and strychnine are not of much benefit. Caffeine sodium benzoate, 7½ grains (0.5 gm.), or from 1 to 2 cc. of camphor in oil given intramuscularly sometimes produces beneficial results.

Complications.—If pneumonia is a complication, oxygen therapy is continued. If a specific infection superimposed upon the injured lung is demonstrated, the use of the appropriate chemotherapeutic agents, e.g., sulfanilamide, sulfapyridine, or sulfathiazole, and serums is advised.

Olive oil in the eyes may be effective. Codeine phosphate or sulfate, ½ to 1 grain (0.0325 to 0.065 gm.), for cough, or spraying of the nose and throat with a soothing solution, is at times useful, but for this latter use products containing oil should be avoided because of the danger of developing a lipoid pneumonia. The patient should be placated regarding any permanent effects to the lungs. A neurosis may be avoided by the initial attitude of the physician and malingering obviated by the emphatic imparting of existing statistics and knowledge regarding these cases.

CHAPTER XII

HYDROGEN SULFIDE

Hydrogen sulfide in a concentrated form is deadly and, like hydrocyanic acid, exceedingly rapid in its action. Its characteristic odor, like that of rotten eggs, is familiar to all. It is a transparent gas, readily soluble in water, and is the natural product of putrefaction of animal or vegetable matter. Because of its intense odor it is easily recognized even in minute amounts.

Occupational Hazards.—This gas is never used directly in industry but is formed frequently as a by-product of certain processes. It is to be found in sulfur dye works, tanneries, in the production of carbon disulfide, in the heating of certain rubbers containing sulfur compounds, and may offer a hazard to those working in sewers, mines, wells, caissons, and tunnels. As reported under "Carbon Disulfide," it exists in the process used for the manufacturing of artificial silk or rayon. It is invariably present to some degree in the refining of oil.

Pathology.—This gas is both an asphyxiant and an irritant. When death results, it is from respiratory failure and occurs within a few minutes, although the heart may beat for a short period after respiration has ceased. It is important that those first on the scene of action remember this and institute artificial respiration. Persons seemingly dead have been resuscitated by such means. Locally it is an irritant; systemically it affects the nervous system. This may range from drowsiness or sleepiness to convulsions, delirium, deep coma, or death. Because of its action upon the vagus nerve, the heart action is slowed. Within the bloodstream hydrogen sulfide is rapidly oxidized without any toxic products resulting from this oxidation. The pathology resulting from poisoning by this gas is apparently transient, for if death does not occur because of asphyxiation, recovery is usually complete. It is significant that at postmortem examination in those cases where death has been instantaneous, the tissues reveal nothing characteristic.

Signs and Symptoms.—These can best be described by listing the reactions as they occur at certain concentrations. At a low concentration of between *100 and 200 parts per million* there exists a mild irritation, especially to the eyes. This usually results at the end of a day's exposure. Within a half hour to an hour *500 parts per million* will bring about excitement, headache, dizziness and staggering, diarrhea and dysuria, to be followed sometimes by bronchitis or bronchopneu-

monia. Short exposures of fifteen minutes to concentrations of from *800 to 1000 parts* intensify the previously mentioned symptoms or may result in death. Above this may mean instant death.

Chronic poisoning results in headache, inflammation of the eyes or eyelids, digestive disturbances, loss of weight, and general debility. This constant exposure is confined usually to chemists or testers in oil refineries.

MEDICOLEGAL ASPECTS

Temporary Disability.—This may be from a few minutes to a few hours or a few days. If a pulmonary complication arises, the disability is for the course of that affection.

Permanent Disability.—This is rare, if ever. As in cyanide poisoning, if the dose is not fatal complete recovery occurs.

Illustrative Cases:

Case I.—While working for an oil company in the Long Beach refineries, a Cracker Stillman's helper started out at 6.30 A.M. to make his rounds at gauging his tanks. Failing to return, he was found one-half hour later lying in six or seven inches of water, face down. Medical aid did not reach him until one-half hour later. The medical examination at that time revealed a male of apparently twenty-six or twenty-seven years. No cardiac sounds could be heard; the pupils were dilated and fixed. He was deeply cyanotic. The extremities were relaxed and moderately warm. There was a minor abrasion about ¾ inch in diameter over the left temple, with no swelling. The neck did not appear to be out of line, and no crepitation could be heard upon manipulation. He failed to respond to cardiac stimulants, and the pulmotor squad using carbon dioxide–oxygen inhalations obtained no results. The autopsy examination showed water in the lungs; otherwise it was entirely negative. No injury to the brain or body was found, and the heart was normal.

The top of the tank which this man was gauging was covered with a half foot of water to prevent the escape of gases. At three places around this tank there were escape valves or pipes, and at one place there was a covered gauging hole. This hole is covered by a flap top, which is opened by a mechanical foot pedal. It is so situated and arranged that usually the wind blows the escaping gas away from the workman. On this particular morning the wind was toward the position the workman would have to assume in operating this gauge. It was, therefore, felt that when the victim lifted the lid he received an unusually high concentration of hydrogen sulfide, was overcome, and fell forward into the water.

In this particular refining process, they were engaged in "cracking"

the oil into different fractions. This was being done by heating this oil to a temperature of 1000 C. At this point the hydrogen sulfide given off is abundant and highly concentrated. The author questioned several petroleum engineers and was told that this concentration is also increased under certain weather conditions.

In the absence of any other obvious cause of death or circumstances which could account for his death, it was held as compensable. The coroner stated that death had been too sudden for any changes to occur that would aid in the postmortem diagnosis.

Case II.—A workman engaged in repairing a tank from which hydrogen sulfide was escaping first became dizzy, noticed a pressure in his head, and then became unconscious. He returned to consciousness in eight or ten minutes and was removed to the hospital. There the examination revealed no cyanosis or dyspnea. The pulse was 60 per minute, and the blood pressure 124-78 70. A few scattered moist râles were heard throughout the lung fields. He recovered completely within thirty-six hours and returned to work at the end of four days without any further disability.

Case III.—Just prior to this material's going to press a second case of death in a Cracker Stillman came to the writer's attention. In a nearby oil refinery this man was found dead. His body was at the foot of the gauging hole. He was thirty-four years of age; his past medical history was of no significance; and autopsy revealed no organic lesions to account for his death. Death was held compensable.

TREATMENT

If this substance is apt to be present in an industrial process, *masks* should be worn which would not only prevent inhalation of the gas but also protect the eyes against irritation. *Ventilation* should be adequate.

Combating Respiratory Failure.—In severe poisoning, chief attention is directed to the respiratory failure, which appears to be due to depression of the respiratory center. The patient should at once be moved from the point of contact with the gas, and fresh air provided. Artificial respiration is started at once, and as soon as available inhalations of mixtures of from 5 to 7 per cent carbon dioxide, with oxygen, are commenced. The use of respiratory stimulants—such as caffeine sodium benzoate, 7½ grains (0.5 gm.); coramine, 0.5 cc., intramuscularly or preferably intravenously; camphor in oil, from 1 to 2 cc., intramuscularly; and metrazol, 1½ grains (0.1 gm.)—may be of value here because of the central character of the respiratory failure. Bronchitis and bronchopneumonia may appear as complications, and require the ordinary treatments for these conditions.

Care of Eyes.—The eyes may be severely irritated, and the condition may even go on to the development of corneal ulcers. As with other irritants to the eyes, *e.g.*, ammonia, they should at once be thoroughly washed with water. Following this the use of a saturated boric acid solution and of olive oil is advised. Local anesthetic solutions, such as 2 per cent butyn or 0.5 per cent pontocaine hydrochloride, may be used in relief of pain. Continuous warm boric compresses may be of value in preventing further complications. When available, the advice of an ophthalmologist should be sought.

CHAPTER XIII

CARBON DIOXIDE AND SULFUR DIOXIDE

CARBON DIOXIDE

Carbon dioxide is a colorless, odorless, stable heavy gas found even in the purest atmosphere. Considered as a waste product of cell metabolism, it is a vital factor in normal respiration. It is a product of respiration, combustion of coal and wood, decay and fermentation. It is frequently found in small, confined spaces such as caves, wells, cisterns, and crowded halls. It changes arterial blood into venous rapidly. Blood pressure increases following stimulation of the central nervous system. The pulse rate becomes slow, the respirations intensify, and sufficient quantities may produce paralysis.

Occupational Hazards.—Employees in blast-furnace shops, large refrigeration plants, boiler-rooms, charcoal burners, caisson workers, divers, coalminers, brewers, vatmen, and submarine workers are liable to this hazard.

Signs and Symptoms.—Headache, vertigo, dyspnea, drowsiness, weakness, and ringing of the ears are symptoms, and severe concentrations may produce giddiness, loss of muscular power, increased respiration, profuse perspiration, and mental excitement. Following the stimulating phenomena, high concentration may produce depression, coma, and death. *Dry ice* (solid carbon dioxide) produces only local but severe disturbances to the skin.

Medicolegal Aspects.—*Temporary disability* is very short, a few hours to a day or two.

Permanent disability does not occur. Deaths have occurred.

Illustrative Case:

Case I.—A longshoreman entered a hold of a ship just docked in order to unload fruit from South America. He was overcome in a short time. Two fellow-workers rescued him, but both of them were partially overcome. All three men reported back to work the following day. Examination of the men by a physician was not made until two hours after the incident. Aside from lassitude and headache, no other complaints existed.

The ship had been delayed by storms. The fruit was found to be in a marked state of decay. All of the men exposed reacted to the exposure. Subsequent physical examination was negative for other causes.

Treatment.—The chief objective is to restore normal respiration either by means of artificial respiration or oxygen therapy. Once sufficient aëration is obtained, the patient usually recovers without sequelae. The oxygen should be administered by a mask or intranasal apparatus. Artificial respiration should be without dead space or rebreathing. Treatment of the *acidosis* by buffer solutions may be of value, although these are seldom needed if the condition is recognized early. The solutions to be used are those mentioned in the discussion of the treatment of acidosis under "Methyl Alcohol"; that is, sodium bicarbonate, r-molar sodium lactate, lactate-Ringer's solution, interstitial salt solution, and physiological saline solution.

SULFUR DIOXIDE

Sulfur dioxide is an irritating gas arising from the combustion of sulfur. It is a common component of the atmosphere of industrial communities and can easily be detected in the air. Its concentration is higher in cities subjected to fog, such as Pittsburgh. According to Schade, its presence in the air in Pittsburgh was too slight to cause concern. In industry the maximum amount of concentration allowable for a short time (thirty minutes to an hour) is from 50 to 100 parts of SO_2 per million parts of air; for prolonged exposure, 10 parts per million parts of air.

Occupational Hazards.—Its most frequent use is in the operation of small, home refrigeration units. Exposure may also be found in the occupations of sulfuric acid makers, smelters, foundry workers, blast-furnace operators, bleachers, cellulose-workers, workers at coke ovens, disinfectant workers, dye-makers, petroleum-refiners, and vulcanizers.

Signs and Symptoms.—Irritation and inflammation to the eyes, nose, throat, and lungs, and occasionally digestive disturbances. It is so irritating to the eyes and throat that man usually withdraws from an exposed atmosphere before further damage is done. Instances where withdrawal was impossible are reported, showing edema of the lungs as well as paralysis of the respiratory center. Chronic exposure according to Kehoe produces a chronic catarrh, a nasopharyngitis and disturbance of the sense of smell or taste, increased lassitude, and fatigue. Other observers have noticed a lessened tendency to colds, bronchitis, or other respiratory infections. Direct contact of the eyeballs with this liquid has caused frozen eyeballs, according to Clark.

Medicolegal Aspects. *Temporary Disability.*—Usually there is none, but occasionally one or two days are required.

Permanent Disability.—There is no permanent disability. Exposure does not induce cardiac disease, tuberculosis, or chronic bronchial

disturbances. Removal from the exposure results in a disappearance of symptoms.

Illustrative Cases.—The cases seen at this clinic have invariably been very mild. We have experienced no fatalities. In most every instance it was not necessary for the patient to withdraw from his work. The following incident is an example of certain symptoms which may present themselves.

Case I.—A twenty-eight-year-old male was seen on April 9, 1940. He had previously been seen in February of the same year for a slight exposure to this gas. On the day of this most recent exposure, he and a helper had gone to a home to recharge an electric refrigerator and had taken with them a drum filled with sulfur dioxide. The patient stated that these drums are filled at the plant when cold, and occasionally more is put into them than they can stand. As they were about to recharge the refrigerator, the drum exploded, striking the helper on the lower leg and fracturing it. This occurred in a small, enclosed room, and both workers were almost overcome. The clothing of both was frozen stiff. The older mechanic managed to get his helper outside, where he removed most of the clothing and then proceeded to the hospital.

Examination of the helper revealed the fractured leg (*tibia*), and a few râles within the lung fields. His chief complaint was referable to his right eye, which had been frozen. He was placed under the care of the ophthalmologist for care of the eye and the orthopedic surgeon for the fractured leg. He required no particular medical treatment. The eye slowly returned to normal in four weeks, and the fracture healed without complications. There was a temporary disability of thirteen weeks. No permanent disability.

The mechanic complained of pain in the throat and chest and a feeling of constriction. His respirations were embarrassed. Examination showed the throat to be red and the eyes injected; the lungs were full of scattered musical râles, best heard at the beginning of expiration. His picture was typical of bronchial asthma. He was hospitalized for twenty-four hours, given two hypodermics of adrenalin, and thirty hours after exposure the lungs were clear. There were no abnormalities relative to the heart or blood pressure. His temperature was 99° F. on admission and returned to normal by the time of discharge. He remained away from work four days. He was reexamined two weeks later, at which time he was entirely normal. However, he decided to quit this type of work, owing to fear of a repetition of his recent experience. The temporary disability in this instance was four days, with no permanent disability.

Treatment. *Irritation of Eyes.*—Workers in industrial plants, firemen, or refrigerator repairmen, who are apt to encounter this gas,

should wear *gas-masks*. Treatment of an eye injured by this gas is similar to that of the eye injured by ammonia. Copious laving of the eye with water, followed by the introduction of saturated solution of boric acid and by the use of a local anesthetic, such as 0.5 per cent pontocaine hydrochloride, is indicated. These are followed by the application of olive oil or some similar oil. Corneal ulceration should be carefully watched for and treated appropriately if it occurs.

Respiratory Disturbances.—For those overcome by the gas, inhalations of from 5 to 7 per cent carbon dioxide in oxygen are used over a long or short period, depending on the severity of the case and the individual patient's reaction. If pulmonary edema is present, oxygen alone is used. In very severe cases artificial respiration is employed coincidentally with the inhalations. In some cases the use of respiratory and circulatory stimulants such as coramine, metrazol, and caffeine sodium benzoate may be of value. A few days of absolute bed rest should be advised for those who have a fairly severe exposure, and this should be continued until all evidence of respiratory embarrassment ceases. During this period codeine phosphate or sulfate, from $\frac{1}{2}$ to 1 grain (0.0325 gm. to 0.065 gm.), every four to six hours, will alleviate the cough and may frequently be combined to advantage with a soothing cough-syrup mixture. Bland oils should be applied to the exposed membranes. For those who display symptoms of acute asthma the subcutaneous administration of $\frac{1}{2}$ to 1 cc. of 1:1000 solution of adrenalin may relieve the symptoms. The use of a nebulizer for inhalation of 1:100 adrenaline might also be of value.

In very severe cases pulmonary edema requiring venesection could conceivably occur, although we have not seen such an instance. In such a case, acidosis may accompany the anoxemia and intense air hunger. Treatment of the acidosis, if present, should follow the suggested outline given under "Methyl Alcohol." Some authors feel that the severely ill patient should not even be asked to move in bed, since any slight exertion may cause the failure of an overworked and asphyxiated heart.[1]

REFERENCE

1. Henderson, Y., and Haggard, H. W.: *Noxious Gases and the Principles of Respiration Influencing Their Action*. The Chemical Catalog Company, New York, 1927, p. 120.

CHAPTER XIV

METHYL CHLORIDE AND METHYL ALCOHOL

METHYL CHLORIDE

Methyl chloride as one of the volatile hydrocarbons has the property of producing narcosis or anesthesia if inhaled in sufficiently high concentrations. The chloride, bromide, or iodide compounds of methyl, following absorption, break down into methanol (methyl alcohol) and the halogen acid; the latter combines with the sodium in the blood to form the sodium chloride, bromide, or iodide.

Methyl chloride occurs in both gaseous and liquid states. It has a low boiling point, is stable, non-corrosive to metals, non-explosive, and relatively non-inflammable. It is not injurious to foods or textiles and may be used in low-pressure systems of refrigeration. While it does have a sweet odor, it is not sufficiently irritating to serve as a warning.

Passing through the lungs with little or no irritation, it first acts upon the central nervous system as a mild to severe anesthetic. Entering the circulation, methyl chloride has an effect upon the various organs identical to that of the bad whisky of prohibition days, owing to the formation of methyl alcohol.

Occupational Hazards.—Methyl chloride is probably the most popular substance used in mechanical refrigeration. Except for chloroform-makers, dye- and color-makers, refrigeration-plant employees constitute the majority of workers exposed to this hazard.

Signs and Symptoms.—Progressive drowsiness, vertigo, disturbance of gait, mental confusion, nausea and vomiting, intestinal cramps, hiccoughs, visual disturbances, tremors, convulsions, coma, and death are the results of exposure to methyl chloride. *Prolonged inhalation* may injure the heart, liver, and especially the kidneys. Recovery from the initial symptoms may be complete, but a few days later death may result from the degenerative changes in the organs just mentioned.

Anuria may result with suppression for from thirty-six to forty-eight hours. Acetone and diacetic acid are common findings in the urine, along with casts and the presence of the formates. A blood count taken soon after exposure is apt to approach normal, but two or three days later there may be a moderate to profound anemia. Most

of these patients have leukocytosis. Spinal punctures may show an increased pressure, but this is not a constant finding. McNally[1] reports a drop in both the systolic and diastolic blood pressure. Weinstein[2] reports case histories of two patients in which there was no initial drop in blood pressure. He does not report the reading several days later. Tachycardia is common, but cyanosis is not.

Tests.—There are no specific tests. The finding of formates and acetone in the urine, and also the sweet odor of the breath or an "acetone" breath, are confirmatory aids.

Medicolegal Aspects. *Temporary Disability.*—Patients suffering minor exposures recover within a few hours to a day or two. Severe exposures may require several weeks to a month. These patients usually complain of residual headache and nervousness. Neurosis or malingering must be considered beyond that time.

Permanent Disability.—If death does not occur in the severely exposed, eventual return to normal may be expected.

Illustrative Cases:

Case I.—While repairing a refrigerator located in a small confined passageway of an apartment, a thirty-two-year-old repairman noted dizziness and headache. He stepped out on the balcony for fresh air and a rest. After ten minutes he returned and continued working for nearly two hours. The owner found him dazed and stuporous; when aroused, he answered questions incoherently. She thought that the man was drunk and telephoned his employer. Wisely sensing the possibility of exposure to a gas, the employer rushed him to a hospital. Upon arrival he responded intelligently to questioning. Vomitus was observed on his clothing, and he vomited several times following arrival. The odor to his breath was like that of chloroform. The temperature was normal; heart rate, 102; blood pressure, 124-80. The general physical examination was negative, and no abnormal neurological findings were present. The urine and blood count upon admission were normal. The following morning the urine was mildly positive for acetone and formic acid. He was discharged the third day after admission, at which time the red blood count was 3,400,000; hemoglobin, 60 per cent; white blood count, 12,350; polynuclear neutrophiles, 68 per cent; lymphocytes, 23 per cent; large mononuclears, 5 per cent; eosinophiles, 4 per cent. The urine was normal. Check-up examinations during the next three weeks were negative. On the twenty-first day following exposure his blood count was: red blood count, 4,700,000; hemoglobin, 80 per cent; white blood count, 8200. He had returned to work sixteen days after exposure.

A substitute repairman reported finding a leak in the system on which the patient had been working. No alcohol was detected on the

breath. No other cause for the illness was found. The physical examination and laboratory findings were in keeping with the history and conformed to the usual case of methyl chloride exposure. The case was considered compensable.

Case II.—A fifty-eight-year-old maintenance man, employed for six years by a refrigerator company, developed persistent pain in his chest, which was more severe at night. Some weeks later he developed a cough and noted dyspnea. Believing his lungs were affected by this gas, he consulted his family physician who informed him it was not his lungs but his heart that had been affected by this gas. He was ordered to stop working and rest in bed. A diagnosis of cardiac enlargement due to prolonged exposure to gas was sent to the insurance carrier. Twelve weeks later he was referred to our clinic for examination. A palpable pulsation was noted in the upper right sternum. At the second left interspace, both to the right and to the left of the sternum there was a loud, blowing diastolic murmur. A water-hammer pulse with high pulse pressure existed. Blood pressure was 190 100. The fluoroscopy revealed an aneurysm of the ascending aorta. The Wassermann was four plus.

Two major errors are apparent in the initial diagnosis. Search was not pushed to determine what kind of heart disease was present, and it was assumed that chronic exposure to methyl chloride could produce this heart disease. The case was not compensable upon the basis that the condition was due to syphilis, a disease not caused, irritated, exacerbated, or precipitated by this gas.

Treatment. *Inhalations and Artificial Respiration.*—Severely exposed workmen should at once be removed from contact with the substance. Inhalations of from 5 to 7 per cent carbon dioxide in oxygen are certainly indicated, and artificial respiration may be needed. In this phase of the treatment the object is to remove from the lungs all possible methyl chloride. It is felt by some that these inhalations should be continued every three hours over the first twenty-four to forty-eight hours. The respiratory and cardiac stimulants frequently mentioned in other sections, *e.g.*, the chapter on "Phenol," should be used when respiratory or circulatory failure appears.

Treatment as in Methyl Alcohol Poisoning.—As indicated above, the methyl chloride taken into the body is decomposed into methyl alcohol and hydrochloric acid. The latter forms relatively innocuous substances such as potassium and sodium chlorides in the blood. The treatment for the methyl chloride poisoning, then, becomes the same as for methyl alcohol poisoning. The patient is hospitalized immediately, and energetic treatment of his acidosis is begun. For details of this, "Methyl Alcohol" should be consulted.

Convulsions and Restlessness.—If convulsions occur, they require 4 gm. (one drachm) of potassium bromide in 4 ounces (120 cc.) of water as a retention enema, but *under no conditions should chloral or chloroform be given.* For the convulsions and restlessness the use of other sedatives, such as phenobarbital, seconal, nembutal, sodium amytal, and sodium luminal, as well as others of this type, may be of value.

Abdominal Pain.—Some cases in which paroxysms of severe abdominal pain have been a prominent part of the picture are reported. In these morphine sulfate, $\frac{1}{4}$ grain (0.016 gm.), was used, together with intravenous calcium. It would appear of value in cases presenting such severe cramps to use 10 cc. of 10 per cent calcium gluconate intravenously, repeated as needed. The use of 1 cc. of 1:2000 prostigmine methylsulfate with the morphine may enhance its effectiveness and allow reduction of the amount of morphine.[3]

Anemia.—If this develops, it is treated with ferrous sulfate, 5 grains (0.32 gm.), two or three times a day, if it is of a secondary type; if it appears to be a macrocytic type, with liver-extract preparations.

REFERENCES

1. McNally, Wm. D.: *Toxicology*, Industrial Medicine Publishing Co., Chicago, 1937.
2. Weinstein, A.: "Methyl Chloride (Refrigerator) Gas Poisoning: Industrial Hazard," *J.A.M.A.*, *108:* 1603–1605 (May 8), 1937.
3. Slaughter, D., Parsons, J. C., and Munal, H. D.: "New Clinical Aspects of the Analgesic Action of Morphine," *J.A.M.A.*, *115:* 2058–60 (Dec. 14), 1940.

METHYL ALCOHOL

The toxic effects of methyl alcohol would not have to be detailed if this substance were known only by this name. However, it is variously labeled as methanol, carbonol, wood naphtha, methyl hydroxide, Columbia, Colonial, or Manhattan spirits, standard wood spirits, green wood spirits, etc. It is obtained by the destructive distillation of wood, and its chemical formula is CH_3OH. It is used with varying degrees of purity.

Occupational Hazards.—Some form of methyl alcohol is used by woodworkers, painters, shellackers, linoleum-, leather-, and soapmakers, printers, photographers, dyers, drycleaners, rubber workers, cementers of rubber, felt-hat makers, aniline-dye workers, makers of antifreeze mixtures, in the collar-fusing process, and in many other trades.

Pathology.—The effect of methyl alcohol on the optic nerve is well known, having been dramatically presented to the profession and layman alike during the period of prohibition. In addition to the visual disturbances, it may affect the liver, kidneys, heart, and brain.

Certain investigators have reported anemia after chronic exposure. It is held that its toxic action is the same whether absorbed through the skin, by ingestion, or by inhalation.

Ruling Out Other Substances.—Before attributing deleterious effects to methyl alcohol when it is used in certain processes, it is well to consider the possibility of other substances being present and acting as the causative agent. Greenburg[1] and his associates studied the use of acetone methanol (methyl alcohol) in the collar-fusing department of a shirt factory. They found a disturbed blood picture in nine of the nineteen subjects studied; in others, an abnormal neurological change; and in some, symptoms of fatigue and drowsiness. A few gave a negative clinical picture. The exposure of these workmen was not only to acetone methanol, but to ethylene glycolmonomethyl ether. They then studied a control series of workers subjected to acetone methanol only and found no abnormalities in the blood picture or the physical examination. They concluded that the ethylene glycolmonomethyl ether was responsible for the altered picture in the first group.

Signs and Symptoms.—In contrast to the initial stimulation produced by grain alcohol, methyl alcohol causes a depression of the cerebral centers. Headache, temporal pain, nausea, drowsiness, which graduates into stupor, coma, and death; or in some instances, delirium, maniacal actions, then coma and death.

Medicolegal Aspects.—One part per million is considered a safe working atmosphere. There would rarely be any danger to the use of this substance if it were properly presented to the consumer. As McNally insists, every container should be labeled in large red letters "POISON—WOOD ALCOHOL."

Temporary Disability.—This is usually short, from a few days to a week or two at the most.

Permanent Disability.—This is invariably the result of impaired or destroyed vision.

Illustrative Cases:

Case I.—After working for three days cleaning a vat with a trade-name product, a workman became acutely ill, vomited, complained of gastric pains and blurred vision. He quickly recovered from all symptoms except his disturbed vision. He is now subtotally blind.

Case II.—Two women worked at pencil-varnishing. They suffered from headache, nausea, and blurred vision. Both claimed that in the open air the symptoms vanished. Constant exposure caused a gradual impairment of their vision.[2]

Treatment. *Special Measures in Industrial Case.*—The treatment of the case of methyl alcohol poisoning seen in industry will differ

to some extent from that seen in private practice. In industry the toxic action will have usually been produced by absorption through the skin or by inhalation, in contradistinction to the ingestion of the product more commonly seen in private practice. For this reason the use of emetics is not indicated, and the value of gastric lavage with 4 per cent solutions of sodium bicarbonate will not be so great, although this latter procedure may be used and will at least be of value in treating the resulting acidosis.

Correction of Acidosis.—The most important phase of the treatment is directed at correcting the acidosis which is produced in the body by the formation of formic acid from the methyl alcohol. It has been the writer's experience to find that medical literature frequently advises treatment of acidosis, but fails to give details of such treatment. For this reason, some detail in the treatment of acidosis is here included. In other sections where treatment of acidosis is suggested, reference to this description has been made. The use of sodium bicarbonate or sodium lactate intravenously is advisable. It is safest to calculate the dose necessary to restore the serum bicarbonate according to the method of Hartmann and Senn[a]:

$$\text{mM.} = \frac{(60 - \text{CO}_2)\ 0.7\text{W}}{2.24}$$

mM. = millimols of sodium bicarbonate or sodium lactate
CO_2 = serum carbon dioxide content in volumes per 100 cc.
W = body weight in kilograms

One mM. of sodium bicarbonate is 0.084 gm.; 1 mM. of sodium lactate is contained in 1 cc. of r-molar sodium lactate. In marked acidosis when serum carbon dioxide is not known, 5 mM. of sodium bicarbonate (1.4 gm.) per kilogram of body weight is a safe dose. Hartmann recommends the injection of one-half the calculated dose of sodium lactate intravenously and one-half subcutaneously as one-sixth molar sodium lactate. R-molar sodium lactate is obtainable in sterilized ampules, which most physicians will find more convenient than sodium bicarbonate. *Sodium bicarbonate cannot be boiled or autoclaved in an unsealed vessel without forming the highly toxic sodium carbonate.* Sodium bicarbonate may be weighed and added with aseptic precautions to sterile water or dextrose solution and injected intravenously in a 2 to 5 per cent solution. Both sodium bicarbonate and sodium lactate may be given by mouth when the patient is able to take them by this route.

(*a*) *Solutions for Use in Acidosis.*—Several very valuable solutions for use in treatment of acidosis are on the market. An interstitial salt solution (sodium chloride, 6.5 gm.; sodium bicarbonate, 2.5 gm.;

and potassium chloride, 0.18 gm. per liter) may be prepared by the addition of the contents of a 50 cc. ampule (Sharp and Dohme Co., Philadelphia) to 500 cc. of sterile water. It may be used in practically all cases of acidosis without additional sodium bicarbonate and may be given subcutaneously or intravenously. Lactate-Ringer's solution (sodium chloride, 6 gm.; sodium lactate, 2.7 gm.; potassium chloride, 0.4 gm.; and calcium chloride, 0.2 gm. per liter) is equally valuable and is prepared in concentrated form in ampules the contents of which are to be diluted with distilled water (Eli Lilly and Co., Indianapolis).

Other Measures.—In addition to treatment of the acidosis the patient should be kept warm. Intravenous infusion of 10 per cent dextrose in physiological saline may be of value in supporting him, and for this there has also been advised the intramuscular administration of atropine sulfate, $^1/_{40}$ grain (0.0015 gm.), not to be repeated; strychnine sulfate, $^1/_{30}$ grain (0.002 gm.); metrazol, $1^1/_2$ grain (0.1 gm.); camphor in oil, 1 to 2 cc.; coramine, 1.5 cc.; or caffeine sodium benzoate, $7^1/_2$ grains (0.5 gm.). Aromatic spirits of ammonia, from $^1/_2$ to 1 drachm (2 to 4 cc.) by mouth is said to be of value. If pain is present, the use of morphine sulfate, $^1/_4$ grain (0.015 gm.), despite its central depressant effect, or codeine phosphate, 1 grain (0.065 gm.), is frequently used; and if delirium is present from $^1/_{300}$ to $^1/_{200}$ grain (0.0002 to 0.0003 gm.) of scopolamine hydrobromide may be combined with the morphine.

Use of 50 per cent solutions of sucrose in treatment of cerebral edema if present has been suggested. The oral administration of a large dose of magnesium sulfate is usually part of the routine. With the exception of the eye, most abnormalities from chronic exposure clear up after removal from exposure.

REFERENCES

1. Greenburg, L., Mayers, M. R., and Smith, A. R.: Ind. Bull. Div. of Indust. Hyg., New York State Department of Labor, Vol. 18, No. 10, Oct., 1939.
2. Bull. No. 86, New York State Department of Labor.
3. Hartmann, A. F., and Senn, M. J. E.: "Studies in the Metabolism of Sodium r-Lactate: II. Response of Human Subject with Acidosis to the Intravenous Injection of Sodium r-Lactate," *J. Clin. Investigation, 11:* 337 (March), 1932.

CHAPTER XV

AMMONIA AND PHENOL

AMMONIA

Ammonia is an irritant causing inflammation of the eyes, nose, throat, and lungs. Like many other irritants, it is rarely absorbed into the bloodstream, affecting the surface tissues only, and especially those which are moist, such as the conjunctivae and mucous membranes. Ammonia in liquid form is furnished in steel cylinders, or supplied in water as a hydrate.

Liquid ammonia is most extensively used as a refrigerant, but there are at least thirty other occupations or processes which use ammonia. The aqueous solution is widely used in the various arts. Tannery and dye workers, employees working with explosives, fertilizers, sewers, varnish silk, nitric acid, etc., may be subjected to its effects.

Signs and Symptoms.—Conjunctivitis, swelling of the eyelids, burning of the throat, coughing, pulmonary edema, irritation of the skin, especially if it is moist, dyspnea, and respiratory failure are symptoms. Because the gas gives a warning signal, fatal dosing is not common. The maximum concentration allowable for prolonged exposure is *100 parts ammonia per million* parts of air. For a short exposure the maximum concentration allowable is from *300 to 500 parts per million* of air. Exposure to more than these amounts, or a very short exposure to from *5000 to 10,000 parts per million*, has been fatal.

Medicolegal Aspects.—This gas offers little in the way of controversy. The occupation, the facts surrounding the alleged accident, the characteristic odor, and the signs and symptoms presented easily establish the case.

Temporary Disability.—This is usually not more than seventy-two hours. If the eyes are severely burned, there may be a temporary disability of several weeks. Pneumonia may occur following pulmonary edema or severe inflammation of the lungs. To be considered compensable, the patient must present these symptoms within a few hours or days following exposure, and not several weeks or months later.

Permanent Disability.—This is exceedingly rare, and if it occurs is most apt to be due to involvement of one or both eyes. A concen-

trated amount of ammonia in contact with the cornea is apt to cause prolonged disturbance of vision or permanent damage. Once permanent loss of vision is established, the extent should be determined and so rated.

Treatment. *General Measures.*—If liquid ammonia is spilled upon the clothing, all *clothing should be immediately removed* and the body thoroughly drenched with water. The eye injured by ammonia should be immediately and copiously washed with water, and this may be followed by the introduction of a saturated solution of boric acid. If pain is severe, the use of a local anesthetic such as 0.5 per cent solution of pontocaine hydrochloride is indicated. Thereafter the application of olive oil or some similar oil is desirable. Continuous warm boric compresses to the eyes may be of value. The usual treatment for corneal ulcers should be instituted if this complication occurs, and an ophthalmologist should be consulted.

Respiratory and Circulatory Measures.—If the concentration of fumes has been severe and respiration affected, inhalations of from 5 to 7 per cent carbon dioxide in oxygen should be given, and if pulmonary edema ensues the use of oxygen by means of a tent or intranasal apparatus is advised. The administration of such respiratory and cardiac stimulants as the following may be of value: coramine, 1.5 cc.; metrazol, from 1½ to 4½ grains (0.1 to 0.3 gm.); and caffeine sodium benzoate, 7½ grains (0.5 gm.). Some of the respiratory and cardiac effects may be reflex from the pulmonary bed in origin, and because of this the intravenous administration of atropine sulfate, from 1/60 to 1/75 grain (0.0011 to 0.0009 gm.), and papaverine hydrochloride, ½ grain (0.032 gm.), might be of value. These should be freshly prepared from the powders just before use.

PHENOL

Phenol is used widely in industry, but in spite of its severe toxic properties and corrosive action rarely leads to fatalities. The occasional case, however, is of interest.

We are not concerned with the accidental or intentional drinking of carbolic acid, but rather with the effect upon the system by absorption. Phenol is readily absorbed through the unbroken skin and is inhaled in the form of vapor. Phenol and the two naphthols, alpha and beta, once absorbed are partly oxidized, partly excreted in the urine in a combination with sulfuric and glycuronic acid, or are excreted unchanged. The effect is primarily on the central nervous system.

Signs and Symptoms.—When phenol is absorbed through the skin, the symptoms develop rapidly (as will be seen in the illustration

below). Dizziness, tinnitus, dyspnea, confusion, muscular twitching, loss of consciousness and death may follow. In mild absorption, headache, dizziness, irregular rapid respirations, and a weak pulse may be present. When death occurs, it is usually within a few hours of exposure.

Illustrative Case:

Case I.—A twenty-two-year-old male, employed by a concern manufacturing ink, was crossing the plant when he dropped a bottle of carbolic acid. This saturated the right leg, right side of the abdomen, and the chest. Fellow-employees immediately removed his shirt and threw water over his upper body. The patient then walked across the street to a physician's office without any difficulty. However, within fifteen minutes after arriving at the doctor's office he collapsed and died.

Autopsy examination, at this clinic, revealed extensive first- and second-degree burns of the right side of the body, right arm, right leg, and scrotum. There was hyperemia and edema of the lower portions of the lower lobes of both lungs, marked hyperemia and edema of both kidneys, pancreas, and spleen. There was no change in the heart or liver. Analysis of portions of the pancreas, spleen, and kidney was positive for carbolic acid, and the chemist reported the quantity found was increased five times over that usually found in these tissues at autopsy.

As in any instance where a patient is saturated with a known noxious solution, all clothing should be removed in order to prevent further absorption. The above case illustrates the rapid absorption through the skin and by inhalation, producing death.

Treatment. *Principal Steps.*—As indicated above, the case of phenol poisoning encountered in industry will usually differ from that seen in private practice in that the mode of poisoning will be by absorption through the skin or by inhalation in the form of vapor rather than by drinking of the material. The most important steps, then, in the acute case of industrial phenol poisoning will be: (1) the *removal of all clothing* to prevent further absorption; (2) *washing of all involved areas* on the skin with a 25 per cent solution of either alcohol or glycerin; and (3) *treatment of shock.*

Respiratory and Circulatory Measures.—The patient should be kept as warm as possible, and symptoms of circulatory shock and respiratory depression should be treated by use of the various stimulants mentioned in previous sections. For the respiratory stimulation the most valuable are inhalations of from 5 to 7 per cent carbon dioxide, and solutions such as metrazol, $1\frac{1}{2}$ grains (0.1 gm.); camphor in oil, from 1 to 2 cc.; caffeine sodium benzoate, $7\frac{1}{2}$ grains (0.5 gm.),

intramuscularly; or coramine, 1.5 cc., intravenously or intramuscularly. For the circulatory shock intravenous infusion of 1000 cc. of 10 per cent dextrose in distilled water, and drugs such as the coramine, caffeine, or epinephrine hydrochloride, from ½ to 1 cc. of 1:1000 solution, intramuscularly, may be used. However, there is some question concerning the advisability of the use of epinephrine in cases of poisoning, for the reasons indicated in the section on treatment of electrical injuries. The use of digitalis preparations is frequently mentioned in treatment of circulatory shock, but seems of very questionable value in this situation.

First-Aid Measures.—As first-aid measures, a quantity of olive oil, cod-liver oil, cottonseed oil, castor oil, or any other available vegetable oil should be given by mouth, as well as egg-white. Mineral oil affords no protection against phenol, since the solubility of phenol in it is quite small, about 1 in 50 parts. *Alcohol, previously thought to be of value, should not be given* by mouth or used for gastric lavage, since alcohol, although a good solvent for phenol, seems to increase the rate of absorption of phenol from the stomach. Thorough gastric lavage with 10 per cent solution of glycerin, or, if this is not available, with sodium sulfate, 15 gm. (4 drachms), to the pint of water shall be carried out.

Nephritis, if it occurs as a complication, should be treated by the standard methods employed for this disease.

It has recently been shown by Meyer[1] that a high protein diet given to rats increased very definitely their resistance to phenol poisoning. This observation, however, probably has no great practical significance in treatment of the poisoning once it has occurred, other than to suggest the use of a high protein diet.

REFERENCE

1. Meyer, A. R.: *Proc. Soc. Exper. Biol. and Med.*, *41*: 402 (June), 1939.

CHAPTER XVI

CARBON MONOXIDE

UNLIKE most of the hazards discussed in this book, that created by carbon monoxide is not confined primarily to industry. Poisoning from this substance is apt to confront men in any walk of life. As a cause of death its incidence closely approaches that due to automobile accidents. The cardinal facts concerning an illness or death following sub-lethal or lethal exposure are well known to all who practice medicine. It is said to be the oldest of all poisons, dating back to the time man first made a fire and hovered over the flame. Much has been written about carbon monoxide, and investigation of its action has been profuse. Yet there still remains considerable confusion as to certain phases of CO poisoning, which needs to be clarified, especially as it concerns workmen's compensation. It is with that fact in mind that the following paragraphs are written.

Properties.—Carbon monoxide is a colorless, tasteless, almost odorless, insidious gas with a density of 0.967 as compared to air. It can be compressed into a liquid and a solid. It burns with a blue flame. When combined with one volume of oxygen, it forms two volumes of carbon dioxide. Carbon monoxide originates from incomplete combustion of carbonaceous material.

Sources of Incomplete Combustion.—There are several:

Illuminating Gas.—This in many parts of the country is referred to as "manufactured gas" in contradistinction to natural gas. Illuminating gas is obtained by forming an admixture of water gas with coal gas, or a petroleum product. It generally contains between 20 and 30 per cent of carbon monoxide. In most cities where this gas is manufactured, the average composition is as follows:

Substance	*Per Cent*
Carbon dioxide	3.5 to 4
Benzol	1.0
Olefins	5.0 to 6
Oxygen	3.0 to 5
Hydrogen	44.0
Methane	19.5
Nitrogen	4.0 to 5
Carbon monoxide	20.0 to 21

Water Gas.—This is made by passing steam over heated coke and contains about 40 per cent carbon monoxide.

Coal Gas.—This is formed from the distillation of coal and contains about 5 per cent carbon monoxide.

Natural Gas.—This contains varying proportions of methane, ethane, nitrogen, and carbon dioxide. It contains no carbon monoxide, but if combustion is faulty there may be carbon monoxide present.

Occupational Hazards.—Because it is practically ubiquitous, it is impossible to list every hazard. The more common sources are electric furnaces, blast furnaces, manufacturing of gas, distillation of oils, charcoal ovens, refuse plants, kilns, and coal mines. To a lesser extent for acute poisoning, but a common source for chronic exposure, is the occupation of presser in tailor shops, laundry workers, garage mechanics, furnace-tenders, firemen, cooks, and housewives. Broadly, anyone who works where power or heat is being generated by a combustion process of carbonaceous material may be exposed to carbon monoxide poisoning.

Pathology.—As a prelude to the discussion of the pathology of carbon monoxide it would seem appropriate to mention certain physiological phenomena of this gas.

Types of Asphyxia.—CO belongs to the group of gases classified as asphyxiants. Of these there are two division: (1) Those which result in asphyxia due to a cessation of breathing. This may be from a mechanical obstruction to normal respiration or to the action of drugs upon the respiratory center. Strangulation, drowning, or confinement in an oxygenless vault or cave-in of dirt are examples of mechanical asphyxia. Morphine and chloroform, acting upon the respiratory center, are examples of the action of drugs. (2) Asphyxia resulting from a deficiency of oxygen but where breathing is not interfered with.

Comparison of Cyanide and Carbon Monoxide Poisoning.—This second type of asphyxia is produced by two chemicals, carbon monoxide and cyanide, each of which acts differently within the body. As pointed out under "Cyanides," the cyanides poison the tissue cells in all parts of the body to prevent the entrance of oxygen into the cells. Carbon monoxide on the other hand reacts within the bloodstream. While carbon monoxide enters the body only through the lungs, it causes no immediate reaction within those organs. During acute carbon monoxide poisoning there exists no interference with oxygen entering the lungs in sufficient quantities. But after the initial reaction with the CO, the blood can no longer absorb what oxygen the lungs supply. In cyanide poisoning the oxygen enters the body and is transported by the bloodstream, but the gate is closed against its entering the various tissue cells of the body.

Supplanting of Oxygen in Bloodstream by Carbon Monoxide.—As carbon monoxide enters the bloodstream, it immediately combines with the hemoglobin and replaces the oxygen. The normal oxyhemoglobin is therefore converted into carbon monoxide hemoglobin, as illustrated by the following equation: $HbO_2 : CO :: HbCO : O_2$. This reaction is reversible, for as oxygen is supplied and carbon monoxide removed the oxygen supplants the carbon monoxide existing in the bloodstream in combination with Hb to again form oxyhemoglobin. It should be noted that the red cells which carry this carbon monoxide are not injured in the process. Finally, concerning this interchange of gases, it is important to know that carbon monoxide has a greater affinity for hemoglobin than has oxygen, this affinity being estimated at approximately 300 to 1.

General Changes.—The body at the time of death may show cherry-red blotching at various areas. Upon opening the body, one finds the blood bright red. It is usually coagulated but at times fluid. Hemorrhages may be present in the lungs, pleura, pericardium, intestinal tract, uterus, and under the mucous membranes of the body. Regarding the heart, there is a marked lack of sufficient autopsy reports to enable investigators to formulate a definite conclusion concerning the cardiac damage which may result from CO poisoning. To date the evidence would indicate that there is no marked damage to the heart or coronary vessels unless the exposure has been of the severest type. The *lungs* usually reveal no special change unless the period of unconsciousness has been prolonged.

Changes in Central Nervous System.—Primarily, the central nervous system sustains the greatest injury as a result of carbon monoxide poisoning, although the nerve tissue of any part of the body may be involved. Vascular lesions constitute a prominent part of the pathological alteration, the arteries as well as the veins being involved. In the brain the vascular stasis is pronounced, the vessels being dilated and packed with red blood cells. Petechial hemorrhages are often numerous and scattered. Following the vascular congestion edema ensues. The increased congestion and accumulative edema will cause a rise in the intracranial pressure. Owing to this widespread involvement of the brain, a victim who survives a sublethal but severe exposure may show evidence of disseminated encephalitis, bilateral softening of the lenticular nucleus, internal hydrocephalus, or a combination of these lesions.

DIAGNOSIS

Signs and Symptoms.—Following the work of Haldane, Henderson, and Haggard, and Sayers and Yant, of the Public Health Service of

the United States and the Bureau of Mines, Hamilton[1] formulated the following table:

Percentage of CO in Air	*Effects*
0.02	Possible headache, mild frontal in two to three hours.
0.04	Headache, frontal, and nausea after one to two hours. Occipital after two and one-half to three and one-half hours.
0.08	Headache, dizziness and nausea in three-quarters of an hour; collapse and possible unconsciousness in two hours.
0.16	Headache, dizziness, and nausea in twenty minutes; collapse, unconsciousness, and possibly death in two hours.
0.32	Headache and dizziness in five to ten minutes; unconsciousness and danger of death in thirty minutes.
0.64	Headache and dizziness in one to two minutes; unconsciousness and danger of death in ten to fifteen minutes.
1.28	Immediate effect; unconsciousness and danger of death in one to three minutes.

The signs and symptoms of carbon monoxide poisoning fall into one of three groups: (1) acute asphyxiation, (2) acute asphyxiation with delayed symptoms, and (3) chronic poisoning.

Acute Asphyxiation.—As seen by the above table, this may vary in its extent, being dependent upon the concentration of the gas, the activity or inactivity of the patient, and the individual susceptibility. Headache and dizziness may be noticed with possible nausea if the concentration does not immediately overcome the patient. Survivors have related a throbbing type of headache with roaring in the ears, confusion, and general weakness preceding their unconsciousness. Unconsciousness occurs when nearly half the hemoglobin is bound to the CO. If bodily effort is made during the exposure, unconsciousness comes on more quickly. It is thought that CO is eliminated in from twelve to twenty-four hours. Patients usually recover consciousness at a 50 per cent CO-hemoglobin level if the poisoning occurred suddenly and the patient is not long in the contaminated atmosphere. The duration of exposure is more important than the percentage of CO in the determination of severity and in the production of permanent sequelae.

Acute Asphyxiation with Delayed Symptoms.—Symptoms and neurological changes may be noticed immediately, or there may be a delay of from a few days to several weeks. Based upon the recognized pathology, most authorities contend that signs or symptoms must appear within the first three or four weeks. After that time they are to be considered as coincidental and unrelated. This contention is frequently questioned in medicolegal cases, as seen in Case III. In the unquestioned case, the picture is varied. Headaches and dizziness may ensue and persist. Visual deficiency or blindness may be present. Twitching, choreiform movements, or convulsive seizures occur. The emotional life and willpower may be profoundly altered. Apathy, disinterest, dulled memory, lack of judgment, and in fact the whole gamut of mental changes even to idiocy or imbecilia have been noted. There may be confusion, hallucinations, states of excitement and symptoms suggesting general paresis. Parkinsonism is not unusual.

In addition to the effect upon the central nervous system, *pneumonia* may set in within a few days following the acute exposure. Deglutition pneumonia following vomiting of food has been reported. There are also references in the literature to complications following hemorrhage into the kidneys, spleen or liver. Permanent damage to the heart is highly controversial.

Chronic Poisoning.—Carbon monoxide is not cumulative. It is replaced by oxygen. The regulation of the number of working hours is such that man lives in an oxygen-pure air at least twice as long as in a contaminated air every twenty-four hours. Therefore, chronic exposure is not likely to produce *pathological* changes. But constant assault to normal function may result in dysfunction, such as loss of appetite, digestive disturbances, headaches, diminished vigor, poor color, and apathy. These disappear upon removal of the patient from the exposure.

(*a*) *Claims of Disability.*—Claims of disability due to chronic carbon monoxide poisoning are difficult to evaluate. There are usually no objective findings, and the subjective symptoms may well be the result of non-industrial disease and usually are. The basis for acceptance or denial of the case of alleged chronic poisoning is less stable than in the acute cases.

(*b*) *Unreliability of Tests.*—The blood cannot be depended upon for a diagnosis. While it is sometimes possible to demonstrate carbon monoxide in the blood of the chronically exposed, many factors tend to discredit the test. Testing the air after duplicating the exact conditions under which the patient works would be a better method when possible. While it has been generally contended that chronic exposure leads to an increased red count and hemoglobin, this cannot be ac-

cepted as a criterion for a positive diagnosis. Tests showing a polycythemia may have caught the patient at a time when the cells were responding to the tissue's demand for more oxygen. Later there may be a decline from this primary response to the point where the picture may be that of a moderate secondary anemia or even of pernicious anemia. In most instances a rigid investigation will reveal other causes for the symptoms allegedly due to chronic carbon monoxide poisoning.

Exposure on Highway.—Truck-drivers who make long hauls are subjected to an exposure which might be termed the "subacute" carbon monoxide poisoning. While that term has never been applied to this hazard by other writers, it is meant to designate those cases which fall short of exposure to a sublethal dose but exceed the minute chronic assault. The following investigation illustrates this contention and is an important contribution to the present status of carbon monoxide poisoning.

Survey in California.—In a survey[2] conducted in 1938 by the Industrial Hygiene Service in conjunction with the California Highway Patrol, 1105 commercial motor vehicles of various types were tested during five-minute runs on the highway, under varying weather and road conditions, to determine the amount of carbon monoxide in the air breathed by the driver; 2.9 per cent of these vehicles were found to be in a potentially dangerous condition, due to concentration in the driver's compartment of over 0.01 per cent of carbon monoxide, which may cause headache, sleepiness, weakness, faulty judgment, and impaired driving ability, if inhaled continuously over a period of six hours or longer.

The four-mile section of U. S. Highway No. 70, between Blythe, California, and the plant quarantine station at the state line, was selected as the location for the survey. The majority of automobiles entering Blythe from either direction have been run continuously for several hours. It is under such conditions of prolonged exposure that the driver and passengers are most likely to experience the toxic effects of relatively low concentrations of carbon monoxide in the car. Several instances have been reported in which the occupants of an automobile reaching Blythe, after a continuous three- or four-hour run, have gotten out of the car, walked a short distance, and collapsed, due to partial carbon monoxide asphyxia.

Automobiles to be tested were taken at random. The tester entered the car with the carbon monoxide indicator, and rode to the other end of a four-mile test run, making a continuous reading of the carbon monoxide concentration at the driver's breathing level, while the car was driven at a normal speed. Comparative readings were taken with

windows open and closed. Cordial cooperation was obtained from drivers and passengers of all cars included in the survey.

When the concentration of carbon monoxide in the car was found to be higher than 0.01 per cent, the source of the trouble was located by checking along the exhaust system with the car at a standstill and the engine idling. Large defects, such as broken mufflers and missing tail pipes, were obvious on visual inspection. Small leaks in the exhaust line, unnoticeable on casual inspection, were immediately apparent when the flexible hose attached to the carbon monoxide indicator was held at these points. Defects were pointed out to the driver, with recommendations for repairs.

Summary of Findings.—Of the 1005 automobiles in which determinations of carbon monoxide concentration were made, 30 cars, or 2.9 per cent, contained concentrations of the gas in excess of the safe limit; 50 per cent of these potentially dangerous cars were ten or more years old.

In a majority of cars in which comparative readings could be made with windows both open and closed, the concentration of carbon monoxide was higher when one or more windows were open than when all windows were tightly closed. This was particularly noticeable in cars with tight floor coverings, and with no large openings in the dash. This illustrates the fallacy of the widespread belief that keeping windows open while driving is in itself a guarantee of protection against gas. On the contrary, if gases are escaping from the exhaust system, the suction created by open windows may draw into the car larger quantities of gas than would enter with windows closed.

In each of the 30 automobiles in which a concentration of carbon monoxide in excess of 0.01 per cent was found, the trouble was traced to defects in the exhaust system. In the absence of exhaust-system leaks, the amount of gas reaching the breathing level of driver and passengers was greater in cars equipped with short exhaust pipes, than in cars with long pipes.

Illustrative Accident Due to Poisoning.—During the survey an accident occurred which is an illustration of the accident hazard due to exhaust leaks. A sedan being driven at moderate speed, on a straight, level highway, ran into the ditch and turned over. A sample of the driver's blood, taken one hour after the accident, showed that 18 per cent of the blood hemoglobin was combined with carbon monoxide. About one half of the carbon monoxide in the blood washes out in the first hour after removal from the exposure. At the time of the accident the driver of the car probably had a carbon monoxide–hemoglobin saturation of about 35 per cent. This is sufficient to cause head-

ache, weakness, nausea, dizziness, sleepiness, faulty judgment, impaired driving ability, and in some cases unconsciousness. The driver had noticed a hissing sound in the engine, but had continued driving. She noticed a gradually increasing headache and sleepiness before losing consciousness. Inspection of the wrecked car showed a blown-out exhaust gasket and several small holes in the dash. Undoubtedly, engine gases caused partial asphyxiation of the driver.

Conclusions.—The following conclusions were reached:

1. *Carbon monoxide* asphyxiation is responsible for many otherwise inexplicable highway accidents.
2. The underlying cause of this hazard is the escape of exhaust gases from leaks in the exhaust system and their entrance into the car in dangerous quantities.

Tests.—The hygienist, not the industrial physician, should concern himself with the estimation of the carbon monoxide present in air where man works. References to such tests are of no value, therefore, in this discussion, but the industrial physician should on occasion request that such estimates be made in order to establish the authenticity of the claim. On the other hand, every industrial physician should know and be prepared to test the blood of any patient supposedly overcome by carbon monoxide. Spectroscopic and microspectroscopic methods are designed for the investigator or one who has immediate access to a laboratory equipped for such tests. But the following test is simple.

Tannic Acid Test of Blood.—Sayers and Yant's tannic acid method gives an approximate quantitative estimate of the CO present. In a small test-tube dilute 0.1 cc. of suspected blood with 0.4 cc. of dilute ammonia (1 cc. of strong ammonia per liter). Mix and add 1.5 cc. of freshly prepared 1 per cent tannic acid solution. Let stand overnight and compare the color either with permanent standards or with standards prepared simultaneously as follows: Bubble CO or illuminating gas through 1 cc. of oxalated blood until saturated. Dilute this and also 1 cc. of normal blood with 4 cc. of dilute ammonia. Make up a series of mixtures of these laked bloods (in 0.5 cc. volume) corresponding to 10, 20, 30, 40, 50, and 60 per cent saturation. Add 1.5 cc. of tannic acid solution to each.

In none of these tests are the color differences well marked if the CO saturation is under 20 per cent. In acute poisoning the saturation usually ranges from 25 to 60 per cent or more, but it falls quickly after the patient is removed from the source of poisoning (30 to 50 per cent per hour in air, about four times as fast in 95 per cent O and 5 per cent CO_2).

Another simple test is merely that of boiling some withdrawn blood. Ordinary blood becomes brown-black, while blood rich in CO becomes a brick-red mass. While easy to do, this test is devoid of any estimate of percentages.

A handy, quick determination is that of taking 2 cc. of carbon monoxide blood with an equal quantity of water and adding three drops of one-third saturated solution of copper sulfate. This will give a brick-red precipitate, while normal blood will yield a greenish brown one (Zaleski).

Differential Diagnosis.—An authentic history of exposure, the absence of the odor of alcohol on the breath, and the presence of the symptoms previously outlined will indicate a CO poisoning. Difficulty lies in the variation of degree of intoxication resulting in a simulation of other diseases. Drug poisoning, alcoholism, cerebral and cardiac accident, and coma from diabetes or uremia must be considered. Suicidal intent must be differentiated from industrial exposure. Finally, *a diagnosis of acute carbon monoxide poisoning is never positive unless determined by an examination of the blood.* In instances where carbon monoxide is suspected but where it has disappeared from the bloodstream it may be found hours later in the spinal fluid.

Sequelae of Acute Carbon Monoxide Poisoning: 1. When the dose has been sublethal and the exposure short, recovery rapidly takes place within a few hours or a day or two at the most without residual symptoms or signs.

2. If the dose has been sublethal but severe, the patient may: (*a*) recover within a few days to continue his previous state of health without ever showing any subsequent evidence of carbon monoxide poisoning; or (*b*) signs or symptoms may be noted upon the patient's emerging from the initial coma; these may persist for several days, several months, or for the duration of the victim's life; or (*c*) following the unconscious period, the victim may be apparently normal for a few days to several weeks, only subsequently to reveal nervous or mental impairment. Here again the length of this impairment is indefinite. It may be temporary, lasting for several weeks to several months, or be a permanent change.

Effect on Liver, Spleen, Kidneys, and Lungs.—There is no volume of evidence that severe acute exposure affects these organs in any permanent manner. The lungs will invariably reveal moisture immediately following the gassing. This will immediately disappear, or pneumonia may be a complication, although this is not common. However, pneumonia, to be attributed to exposure to this gas, should ensue within a few days to a week following the exposure. Tuberculosis is not a sequela. Glycosuria is frequently present in acute poisoning

from this gas. It usually disappears within a few days, and normal liver function is resumed. It is held that the mobilization of sugar in such instances results from central nervous system impairment during the phase of asphyxia. Some differ from this view, placing the responsibility upon the adrenals or the thyroid, or both. While the urine also reveals albumin, this likewise soon clears up and rarely is there permanent damage to the kidneys.

The previous paragraph indicates that the majority of the systems of the body escape damage to any appreciable extent from an assault of carbon monoxide. Two systems, not mentioned in that paragraph, deserve more detailed consideration; namely, the nervous and circulatory systems.

Effect on Nervous System.—From what has been written, it is obvious that the central nervous system exceeds all the others in its frequency of injury. Yet the frequency of permanent damage does not parallel to any extent the incidence of injury. The profession and laity have an erroneous belief that patients who show early mental or nervous changes continue to do so for the rest of their lives. Probably the best concrete rebuttal to such a belief is to be found in the report of Schillito.[3]

In the New York City area from 1925 to 1935, there were more than 21,000 cases of acute intoxication from carbon monoxide. Schillito reviewed these records, as well as the records of seven state institutions that receive the mental and neurological patients from that city. From his investigation he estimated that only one in every 500 patients with acute carbon monoxide poisoning later showed mental or nervous symptoms. Regarding subsequent psychoses, he found only one case of carbon monoxide psychosis to 2000 other types of psychoses, or a percentage of 0.05.

Effect on Heart.—Does acute carbon monoxide poisoning produce cardiac changes of a permanent nature? Concerning the occurrence of nervous system lesions due to CO intoxication there exists no lack of evidence, but regarding permanent heart lesions, dogmatic opinion should be withheld until reasonable scientific deductions can be made from a large series of autopsied cases and also from a series of cases in which the state of health was known before exposure and in which these patients have been followed for a long period of time after exposure. The consensus in the literature is that, aside from the expected imposition placed upon the heart at the time of gassing, there persists no cardiac damage. Invariably, autopsy reports describe profusely the pathological changes in the central nervous system, but only occasionally refer to findings in the heart, which would suggest a coinci-

dental relationship rather than a consistent sequela. Paul White,[4] in discussing remote causes of heart changes says:

Some changes in the heart have been reported, as for example, subendocardial hemorrhage after CO poisoning, but such changes have been neither extensive nor consistent. Chronic symptoms and signs, such as the inverted T waves in the electrocardiogram resembling coronary T waves, should be ascribed to pre-existing heart disease, whether or not accentuated or revealed after CO poisoning, about which especially there has been so much uncertainty.

(*a*) *Electrocardiographic Changes.*—Stearns, Drinker, and Shaughnessy[5] conducted an electrocardiographic study of a series of cases of carbon monoxide asphyxia to find that the most common deviation is an abnormality in the T waves or in the level of the ST segment. They feel that the changes, having been *transitory*, may be ascribed to carbon monoxide asphyxia, except when coronary disease may have been a contributing cause in some cases.

(*b*) *Basis for Diagnosis of Cardiac Injury.*—There is no intention in the previous statements to imply that permanent cardiac damage cannot occur following a severe, prolonged exposure. In such instances signs and symptoms referable to the heart should be in evidence immediately or soon after the exposure. The condition should be followed by periodic examination; and if it is apparent that the impairment is of a permanent nature, the workman should be accorded compensation. But the onset of a heart lesion months or years after an exposure to carbon monoxide poisoning deserves no consideration as to their relationship. More will be said upon this point after several recent articles have been reviewed.

(*c*) *Opinions on Cardiac Involvement.*—That carbon monoxide rarely produces any cardiac damage finds adverse opinion in a report of Beck and Suter[6] in 1938, and reiterated more recently by Beck, Schulze, and Suter.[7] Their observations include both the acute and chronic types of exposure. The first article deals with the rôle of carbon monoxide in the causation of myocardial disease, while the second one considers nervous system lesions as well as the cardiac. They state that carbon monoxide anoxemia does not differ essentially from anoxemia associated with severe forms of anemia, and point out that certain investigators have called attention to the association between angina pectoris and pernicious anemia. The factor responsible in both instances is the diminished oxygen-carrying capacity of the hemoglobin. They vest authority for this belief in the papers of Keefer and Resnik, Elliott, Cabot, and Allbutt, wherein it is expressed that the

anginal syndrome, as well as coronary disease, may result from anoxemia of the myocardium. But Beck and Suter do not emphasize the fact that in severe anemias the anoxemia is a constant factor. It would seem that they refute themselves when they go on to quote Kroetz as stating that:

> The classic anoxemic lesions in the heart of victims of carbon monoxide are hemorrhage, necrosis and processes of granulation and regeneration, and these lesions coexistent with similar lesions in the brain add to their significance. The lesions undergo rapid regenerative changes, and the processes of repair may be fully developed by the ninth day.

This observation of Kroetz is in keeping with the observations of White and also those of Stearns, Drinker, and Shaughnessy, concerning electrocardiographic changes previously referred to, and in which there is evidence that the changes are transitory.

Beck and his workers state that carbon monoxide anoxemia has a predilection in the heart for the left ventricle and the papillary muscle of the mitral valve. These hemorrhagic lesions, they avow, are *frequently* found at autopsy. Such a contention is to be challenged. While it cannot be disputed that such hemorrhagic lesions may be found at postmortem examinations, they have not been observed or reported in sufficient frequency to classify them as regular, consistent sequelae.

It would appear that Beck and Suter, and Beck, Schulze, and Suter have had opportunity to study a series of cases which presented multiple symptoms of ill health. Among these were patients who also had symptoms referable to the circulatory system. In the first of the two papers presented by the above men they considered a series of 136 cases of supposed carbon monoxide anoxemia of a chronic type extending over a period of fifteen years. The chief complaints of many of these patients, they state, were referable to the cardiovascular system. They do not state how many of the 136 patients revealed cardiac lesions undoubtedly due to chronic exposure to this gas. Nor do they indicate that the percentage of patients having cardiovascular lesions was high enough to exclude any doubt as to the causal relationship. Only five histories are detailed; and, of these, two patients had coronary thrombosis; two had angina pectoris; and one myocardial failure. The first patient cited was thirty-six years of age; the remaining were seventy-four, fifty-nine, sixty, and fifty-five years respectively, a period when heart disease may be expected irrespective of other causes. The following is a case they report to illustrate their point:

B. C. K., a lawyer, aged fifty-nine, a resident of Oklahoma, referred by Dr. M. L. Lewis, had suffered for two years prior to admission from frequent attacks of precordial distress, clinically diagnosed as angina pectoris.

Although for several years the patient had had only occasional attacks of angina pectoris, in the late fall of 1934 they increased in frequency, averaging one daily until Jan. 10, 1935, when he went to Dallas for consultation. From there he came to Baltimore for further study and observation. During this period he did not have a single attack, nor did he have any while under our observation, a period of two weeks. On examination Feb. 6, 1935, he was unable to recollect having had a single attack during the summer months when not exposed to carbon monoxide. This fact led to the assumption that carbon monoxide was a factor in the production of his anginal attacks. Further inquiry revealed a history of exposure to carbon monoxide over a period of twenty-five years. His house was heated by natural gas in open heaters, inadequately ventilated. For the previous five years he had been exposed also in the gas and oil fields; besides, he frequently detected escaping gas in his automobile while driving with the windows closed.

In addition to anginal attacks, he complained of symptoms of anoxemia: dyspnea, palpitation, weakness in the legs, unsteady gait, cramps in the calf muscles, headache, vertigo, cardiospasm and paresthesia. He used alcohol and tobacco moderately, drank coffee to excess and overworked.

His blood pressure was 102 systolic and 80 diastolic, his pulse rate 66 and his temperature subnormal. The heart was slightly enlarged, with an occasional dropped beat and reduplication of the second sound. No murmurs were heard. The blood vessels appeared normal for a man of his age.

Laboratory studies showed mild glycosuria, a blood sugar content of 174 mg. per hundred cubic centimeters, a basal metabolic rate of minus 21 per cent and a negative Wassermann reaction. A blood count showed hemoglobin content 81 per cent, red cells 4,780,000, and white cells 8,250.

Electrocardiographic study by Dr. King revealed an abnormal tracing suggestive of myocardial degeneration, possibly of coronary origin. The diagnosis of angina pectoris was confirmed by Dr. L. F. Barker.

By improving the heating conditions and adopting a more hygienic mode of living, the patient improved and remained free from anginal attacks for some months. He later had symptoms of myocardial degeneration with congestive heart failure.

In the above history, the exposure to carbon monoxide allegedly resulted from living in a house heated by natural gas, from being ex-

posed in the gas and oil fields, and from his automobile. The case history does not reveal the extent of the exposure in any of these three phases. Just why these exposures must be accepted as the cause of the man's precordial distress is difficult to understand. Possible adverse criticism of such reasoning would not be entertained if the extent of exposure had been reported. But upon a similar basis of reasoning, the alleged cause cannot be accepted until the actual extent is known. As a counter argument the author's uncle could be used as an illustration. This member of the Johnstone family lived his entire life in homes heated by natural and manufactured gas, worked for over thirty years in the gas and oil fields, and had the usual exposure to automobile exhaust fumes. Yet he died at the age of eighty, free from any cardiac involvement. His case can be reduplicated by millions of others.

In the case reprinted from the article of Beck and Suter it is intimated that the freedom of attacks during the summer was due to the absence of exposure to carbon monoxide from heating sources. Elsewhere in their article they refer to the seasonal incidence of heart attacks and suppose that this increase in the winter is the result of gas stoves. They refer to reports by Wood and Hedley[8] and by Master, Dack and Jaffe,[9] wherein the seasonal incidence of heart attacks is studied. But after reading the two papers they refer to, one can find no inference that carbon monoxide is a factor in the production of heart attacks. These two sources are concerned with the factor of cold as a precipitant of heart attacks.

In contrast to these contentions of Beck and Suter, McNally,[10] who has had a wide postmortem and pathological experience with carbon monoxide, makes no observation that chronic exposure produces cardiac lesions or that cardiac lesions are apt to appear months or years after an acute exposure. Sandall[11] found no abnormality of the heart in 85 per cent of cases in which tachycardia had been a definite symptom. Loewy[1] reported a case of chronic carbon monoxide poisoning in a furnace-tender after an exposure of ten years in whom the symptoms were referable to irritation of the labyrinth. No cardiac lesion existed. Baader[1] reports a case of labyrinthine disturbance in a man who had charge of a gas-generating department of a manufacturing plant in which there was a four-year exposure. He observed no cardiac condition. Hamilton[1] states:

> The subjective symptoms described by men and women who work continually in air vitiated by carbon monoxide cannot be distinguished from the symptoms of ill health which come from factory work itself, from long hours indoors, making the same movements over and over, plagued by noise and vibration, by excessive heat and humidity, the

effects of all of which may be enhanced by poor food, crowded sleeping quarters, advanced age, or some chronic disease. It may be that the symptoms will disappear if the patient changes to another kind of work; but even that is not evidence of the action of carbon monoxide, for the new factory may be comfortable, the man may be better adapted to his new job, and his wages may be higher.

In her monograph she makes no mention that heart lesions follow chronic exposure or are to be expected; nor does she intimate that, once the patient has recovered from his acute exposure without immediate evident sequelae, a cardiac disturbance may appear sometime later.

(*d*) *Cardiac Resistance to Carbon Monoxide.*—An experimental example of the resistance of the normal heart to carbon monoxide is to be found in the experiment of Haggard[12] in which he subjected dogs to carbon monoxide, producing death in from twenty-five to forty minutes, during which time he took electrocardiograms. He noted no toxic action of this gas upon the heart, the reaction being due to the anoxemia alone. If the anoxemia was relieved, the effects upon the heart disappeared. Zondek made observations on three young adults who were rendered unconscious by carbon monoxide. In addition to a fall in blood pressure, irregularity of pulse and tachycardia which subsequently became slow, the *x*-ray study of the heart of these three youths showed dilatation. All three patients made a complete recovery. To these cases of Zondek's[13] may be added an interesting case of Colvin's.[14] This patient was rendered unconscious from the fumes of an automobile exhaust. Electrocardiograms taken from twenty-four to seventy-two hours after the poisoning revealed evidence of intraventricular block. Subsequent electrocardiograms revealed improvement, and a similar study sixteen days later was normal. He was discharged as normal.

The papers of Beck and his associates have been criticized at some length because of the dangerous implications they make. They would have us draw conclusions upon a basis of reasoning not ordinarily acceptable in medicine, certainly not in medicolegal medicine. In industrial medicine, to assume without accurate knowledge involves an economic injustice. To give credence to Beck's contentions would lead to many false claims and, therefore, to legal entanglements that would parallel the former status of silicosis. To reiterate, the conclusion he and his workers reached is this: In a group of 136 patients who had been *intermittently* exposed to carbon monoxide over *varying* periods, they noted that myocardial symptoms were frequently manifested. They propose that the carbon monoxide was the etiology of the car-

diac disturbance, yet it is not shown that there existed any constant relationship between the anoxemia of CO and heart disability. They do not reveal any figures of the amount of CO in the blood of these patients, or of the amount of CO in the atmosphere. A great deal of their study was with the natural gas of West Virginia, which contains no carbon monoxide; and unless all the allegedly affected persons were using defective appliances, they would not be exposed to carbon monoxide. It is apparent that every here and there in their series there was a patient who had heart symptoms and who had been exposed to carbon monoxide at intervals to no known amount.

Master[15] has estimated that from 500,000 to 1,000,000 Americans sustain a coronary occlusion annually and that this occurs chiefly between the ages of thirty-five and sixty. This estimation does not include heart lesions due to infection or toxic states. There thus exists a large group of cardiac cases in which carbon monoxide is not entertained as the etiology. On the other hand, millions of Americans who have had a chronic exposure to carbon monoxide live and die without any evidence of heart lesions. Finally, since this gas is probably the oldest known poison and since its properties are well known, it is most likely that investigators, toxicologists, and pathologists would have long ago recognized a relationship between chronic exposure and heart lesions had one existed.

MEDICOLEGAL ASPECTS

Criteria for Establishing Compensability of Heart Lesions.—In light of the existing knowledge of the effects of carbon monoxide upon the heart, the following are suggested criteria upon which the industrial physician could estimate the justice of a claim:

Immediately after Exposure.—If immediately following, or within from a few days to a week after a severe exposure to carbon monoxide fumes, a patient develops a cardiac lesion which was not known to be present prior to the exposure, he is to be accorded the benefit of the doubt and be duly compensated. A non-disabling valvular lesion recognized as being of long standing and due, for instance, to a luetic or rheumatic cause should receive no consideration.

Several Months after Exposure.—If a cardiac lesion is alleged to have had its onset a number of months following an acute exposure of varying intensity, the cause is to be seriously questioned and all factors carefully evaluated before a decision is reached.

Several Years after Exposure.—If a cardiac lesion is alleged to have had its onset several years following an acute exposure of varying intensity, the claim is to be denied. I know of no way in which one could reasonably establish the authenticity of the claim.

After Prolonged Exposure to Small Amounts.—If a cardiac lesion has its onset after a prolonged exposure to minimal doses of carbon monoxide, the case must be most carefully investigated. This includes search for evidence of the existence of a lesion in previous medical examinations; estimation or measurement of the gas in the alleged hazard; examination of the blood of the patient if he is still working in the hazard at the time he makes his claim; and consideration of his past medical history concerning rheumatic infections, repeated tonsillitis, quinsy, lues, the thyroid state, evidences of long-standing dyspnea, orthopnea, and the like. Certainly the burden of proof in such an instance is upon the claimant. For instance, it would be unreasonable to assume that a minimal chronic exposure was responsible for arteriosclerosis with hypertension and decompensation in one in his fifties or sixties.

Temporary Disability.—This ceases with the termination of the symptoms. It may be from a few days to several months. Prolongation demands consideration of the factors of the original exposure as to degree, length, and severity of the initial symptoms.

Permanent Disability.—Almost without exception this occurs when there has been damage to the central nervous system. Such changes should be manifested within from several days to three weeks following exposure. If immediate recovery takes place, a symptom arising months or years later should not be attributed to carbon monoxide without adequate foundation. Search for unrelated pathology should be made.

Illustrative Cases:

Case I.—A night watchman was found unconscious in "his office," which was only large enough for a bunk, stove, chair, and reading-lamp. The light was burning, a detective magazine was by his side, and the gas was escaping from the rubber hose, which had split at the wall connection. Carried outside, he was given artificial respiration. Upon his admission to the hospital, the physical examination was in keeping with the history of exposure and the blood positive for CO poisoning. He remained in a deep coma for ten hours. Following his exit from coma, a complete right-sided paralysis was present with inability to speak. He eventually recovered his speech, the complete use of the right leg and partial use of the arm, but he remained childish, had a dulled memory, failed to differentiate his wife from the rest of the family or friends. He answered all questions by yes or no, but never spoke a completed sentence. Little or no improvement occurred in the following three years.

All of the requisites for accepting this case as compensable are satisfied. History, positive evidence in the blood test, and sequelae

are in keeping with experience. In this instance, complications were present from the beginning, there being no delay in the onset of symptoms.

Case II.—Two truck-drivers, making a long haul, pulled into a road-side eating-place. While the helper entered to procure sandwiches, the driver stretched out in a partially closed cab with the engine running. Interest in a broadcast of the war news in Europe delayed the helper. Upon returning to the truck, he found the driver unconscious. Rushed to a nearby physician, the patient regained consciousness while in the doctor's office and before resuscitation was attempted. No blood examination was made. Headache was complained of for about ten days. He returned to work fifteen days after the incident. The physician's diagnosis was carbon monoxide poisoning.

Because the patient was in a cab of a truck, the motor of which was running, indicates that he might have been in a coma from carbon monoxide poisoning, but such an assumption was not established. The proximity of the exhaust to the cab was not stated, no mention of a leak in the exhaust system was inquired into, the patient's blood was not examined for CO, nor was there any attempt to find any other cause for the coma.

Compensation for temporary disability was paid by the insurance company. The circumstances and the "doctor's diagnosis" precluded any other course. If subsequent mental sequelae of an indefinite nature had arisen, the questionable rôle played by carbon monoxide could never have been established equitably.

Case III.—A fifty-nine-year-old night mechanic was found unconscious in a garage nearby the running motor of a truck. He was apparently normal two hours previously when he dispatched a truck and driver from the place. After preliminary attempt at resuscitation he was taken to a Long Beach hospital, where a diagnosis of carbon monoxide poisoning was made. After remaining in the hospital for one week, he went home, but continued to report thrice weekly to his physician for treatment to a leg burn suffered at the time of his attack. After seven weeks he returned to work and continued at that for two months at which time he was discharged by his employer.

Following his discharge, his wife filed claim for permanent total disability, stating that her husband had undergone a change in personality as a result of the carbon monoxide poisoning. This change, she alleged, consisted of loss of memory, apathy, and disinterest, inability to concentrate sufficiently to finish any project he attempted, and other conditions. Six months after the alleged poisoning and just prior to the trial, the patient was referred to the writer.

History.—The past medical history was irrelevant. With the exception of poor oral hygiene and slight bilateral edema of the ankles, the general examination was negative. The heart action and cardiac history did not account for the edema. The neurological examination was entirely negative. The patient had a stolid, quiet, reticent manner, but his expression did not impress the examiner as being typical of the mask-like countenance of parkinsonism. He readily answered all questions, was well oriented as to time, place, and events and smiled when humorous remarks were made. The laboratory investigation was entirely normal.

Testimony.—The following facts were brought out at the trial. Sometime before he lost consciousness (the patient does not know how long), he had a severe pain around his heart while outside the garage. When he was taken to the hospital *no blood test was made* for the presence of CO. The wife stated that she noticed definite mental changes in her husband two or three days after his accident. The physician who first cared for him had recorded no such changes in the progress notes or in his office record. The employer testified that he had noted changes in the character of the patient's work prior to his accident. The psychiatrist who testified for the claimant believed the man had parkinsonism as a result of carbon monoxide exposure. A second psychiatrist who examined the patient, but did not testify, reported by letter that he believed the man did not have parkinsonism and was able to work. The author testified that while the circumstantial evidence strongly indicated a carbon monoxide poisoning, this diagnosis could not be established in the absence of any blood test, and such a diagnosis, while likely, was clouded by the patient's statement that some hours previously he had had a severe heart pain. Furthermore, the author felt that it was impossible for him to evaluate any alleged change in this man since he did not observe him prior to this accident, and other than the wife's statement there was no evidence that such a change had occurred. Certainly to him the case was not a definite parkinsonism.

The commission found that no evidence had been produced to show that the claimant was either mentally or physically unfit to work as a mechanic. The case is presented not because it adds anything to the knowledge of carbon monoxide poisoning, but for certain lessons which can be drawn from it. These lessons are to be found in errors of omission committed by the physician in attendance. Had the patient been overcome in his own home, these omissions would not have been so significant. First, while the circumstances strongly indicated a carbon monoxide poisoning, the diagnosis was never established owing to the failure to examine the blood. It subsequently developed that a possibility of a cardiac episode clouded the issue.

Second, the wife's contention that mental changes set in shortly after the accident could not be denied or substantiated by the physician's hospital record or office file. He failed to appreciate the possibility of the development of latent effects. It should be apparent to the reader, then, that a case of carbon monoxide poisoning arising out of occupation should first be established by blood investigation for CO and then observations for sequelae recorded over a period of time.

Case IV.—A presser in a tailoring establishment claimed loss of weight, fatigue, and two attacks of coma due to chronic exposure to gas fumes. His physician, upon the basis of history, diagnosed the case as chronic carbon monoxide poisoning. He was given from three to six months' temporary disability and advised not to return to work. The insurance carrier desiring to determine the authenticity of the claim was unable to locate him for three months, but at the end of this time found him visiting relatives in Arizona. An investigation revealed that he was a Russian Jew; his mother had diabetes; he had been refused insurance because of sugar in the urine eight years previously; he had polyuria, polydipsia, excessive appetite, glycosuria, and a blood sugar of 280 mg. Investigation of his working conditions revealed that he pressed but rarely, usually being engaged in repairing and mending. The machine he did operate when pressing was located by an open window.

The case was not compensable. Preponderant evidence pointed to diabetes mellitus, and the occupational conditions indicated the improbability of exposure.

Case V.—The patient, aged thirty-nine, was brought to the clinic by a female physician, who believed that her patient had acute CO poisoning. The history was that the patient was found semi-conscious near a large coffee urn in the hotel where he was a cook. Upon admission he was cold and clammy but conscious. He complained of pain in the left chest; lips, ear lobes, and fingertips were cyanotic. Heart tones were distant, the pulse weak, and fibrillation was noted. Blood pressure was 90 systolic, with the sounds immediately disappearing. Electrocardiogram was not taken. The patient died in three hours. The blood was negative for evidence of CO. Autopsy revealed no evidence of CO in the blood or cavity fluids, but did show a fresh coronary thrombosis.

The case presents no difficulty in diagnosis. Had the patient died at home some days or weeks following exposure without blood determinations, there might have been legal controversy.

Case VI.—An unmarried professional man, aged twenty-seven, was found unconscious with a companion in a parked automobile on

the morning of February 18, 1934. The estimated duration of exposure to carbon monoxide was about six hours, and another three hours elapsed before he was brought to the hospital. The companion was dead when found, which indicated the severity of the exposure. The windows of the car were closed, the engine had been left running, and later investigation revealed a leak in the gas heater with which the car was equipped.

On his admission to the hospital, physical examination showed the patient to be in profound coma; he was cold and clammy; there was extreme cyanosis of the face and extremities; and the lips were cherry-red. The heart tones were of good quality. The lungs showed moist râles, and breathing was greatly labored. The abdomen was distended and tympanitic. There had been a profuse spontaneous diuresis before the patient was moved from the car. The pupils were widely dilated and reacted only slightly to light; corneal reflexes were absent. The only other neurological feature of import was a bilateral Babinski sign.

For the first thirty-six hours after his admission to the hospital the patient remained in a profound coma. For the next thirty-six hours the coma was apparently less profound, and the patient began to show some involuntary movements, moving his head and hands aimlessly. At this time he was also able to take fluids orally. On the following day a more noticeable restlessness developed; the patient opened and closed his eyes, snapped his fingers, and thrashed about in his bed. This behavior continued for nearly two weeks with but little change. Sixteen days after admission the patient rather abruptly regained consciousness and recovered his power of speech. Neuropsychiatric examination at this time showed a marked clouding of the sensorium.

Attempts to converse with him elicited only brief, but not entirely pointless remarks. Asked, "What is your name?" he replied, "Puttentain," a response suggestive of a childhood game which the patient had known. To other questions relative to his background he gave abusive retorts, as, "Who the hell wants to know?" He showed no evidence of recognizing his mother or sister with whom he had been living at the time of the accident, or other close relatives or friends. Questions relative to his physical condition brought a more adequate response, and he complained of pains in his legs and feet.

During the days immediately following he was overtalkative and showed a marked flight of ideas. He was irritable, profane, obscene, combative, argumentative, and extremely uncooperative. He showed an almost complete loss of memory for recent and remote events. He demonstrated some of the components of the parkinsonian syndrome in a marked degree. These consisted of pill-rolling, drooling from his mouth, and, to a lesser extent, the masklike facies. This condition was evident on the sixteenth day after exposure to carbon monoxide and persisted for over a month, disappearing gradually.

After he had regained consciousness the patient suffered severe pains in the arms and legs and was very voluble in his complaints about this condition. The toe drop on the right was noticed on examination of the limbs. The peripheral neuritis responded rather slowly to baking and massage, and except for mild paralysis of the muscles of the right foot, which was aided by a support, the patient made a complete recovery. He became oriented in all spheres, regained his memory for remote and recent past events, and returned to his normal mental level by the sixtieth day after admission. He was discharged seventy-seven days after admission, but he was unaware of the nature of his illness before this time. On being informed of the circumstances of the accident resulting in his illness, he showed a normal concern for his dead companion and for the possible consequences to himself and his relatives.

About a year after the accident the patient was able to resume his professional activities in a limited way, and a year after this (two years after the accident) he had so far recovered that he discharged his full duties without any noticeable impairment.[16]

This case illustrates the severity of exposure, the onset of neurological signs and symptoms within the expected two- or three-week period, and the eventual recovery after a temporary disability of two years.

Case VII.—A forty-two-year-old auto mechanic was found unconscious in a private garage where he had been dispatched to repair a car. From a subsequent history it was believed the door had been closed by the wind, without the victim's knowledge. The lips, ear lobes, nails, and face were cyanotic, and the body was cold. The respiration was very shallow. Artificial respiration was given until a doctor from a nearby emergency hospital arrived with an oxygen–carbon dioxide apparatus. After forty-five minutes of this treatment, the patient was removed to the hospital. Immediate examination of his blood showed a 35 per cent saturation of carbon monoxide. The blood pressure was 130, systolic; 85, diastolic. Pulse upon arrival was 130 and regular; temperature, 97.2° F. The heart sounds were distant and feeble; the lungs were normal. The oxygen–carbon dioxide administration was continued for two hours after admission. About one-half hour after admission the electrocardiogram showed the S-T segment depressed in Leads I, II, and III, and elevated in IV. The T wave in Lead IV was upright and abnormally high. The patient thrashed about the bed for nearly six hours. The following day he complained of headache, was not clear about the events of the preceding day, and acted indifferently to relatives. At this time there was moisture in both bases of the lungs, the pulse was regular with a rate of 90. The blood pressure was 118, systolic; 80, diastolic. There were no signs of central nervous system in-

volvement at this time. Against his wishes he was retained in the hospital for five days. Upon discharge his general physical condition was normal, as was the electrocardiogram. Two years later this patient was seen because of a fractured rib; and a re-check examination proved him to be normal in all other respects.

The factors surrounding the onset of coma indicated carbon monoxide poisoning. This was soon established by the finding of an abnormal amount of carbon monoxide in the blood. He returned to his work sixteen days after exposure. The case was compensable. It is used here to illustrate the effect upon the heart of CO poisoning, with the subsequent and rapid clearing of these signs.

TREATMENT

Prophylaxis.—The prevention of carbon monoxide poisoning in industry is not ordinarily a difficult problem. In the larger industries where combustion of various types is a part of the process, the hazard is well recognized, and, therefore, precautions are taken. Small plants and garages constitute a more likely source of this poisoning. Adequate ventilation is all that is necessary to insure against poisoning from this gas, but men need to be warned about closing windows and doors, which they are prone to do in the winter months.

Acute Anoxemia.—When this occurs, *the victim should be immediately removed from his exposure and artificial respiration begun.* There is no point in delaying this in order to obtain a pulmotor, as is so frequently done. This machine has not sufficient advantage over human manipulation to warrant the delay. It is extremely important, however, that inhalations of from 5 to 7 per cent carbon dioxide in oxygen be started as soon as possible. The inhalations should be increased to the point where full, deep inspirations are obtained from the patient. All emergency squads which are frequently called to attend such cases should be equipped with this carbon dioxide and oxygen mixture with inhalation apparatus for its administration. If such apparatus is immediately available, the patient should be kept warm with blankets and hot-water bottles and treated at the spot, rather than be moved to the hospital with attendant delay in the administration of the inhalations. It should be remembered that probably half of the carbon monoxide is eliminated from the body during the first hour after removal from the exposure, and consequently long-continued administration of the carbon dioxide and oxygen may not be required. If such inhalations are not available, the patient should be transported to a hospital where they can be obtained, and en route it is necessary to continue the artificial respiration.

Koch[17] recommended blood transfusions preceded by venesection in severely acute cases. He pointed out that the chief effect of carbon monoxide poisoning was the anoxemia and, therefore, the hemoglobin introduced by means of the blood transfusion will act at once as an oxygen vehicle.

Methylene blue, once thought to be an antidote, is not of value and is now thought to be harmful. With the possible exception of caffeine, it is felt that the *use of drugs is ineffectual* if not actually harmful. Strychnine, camphorated oil, digitalis, pituitary extract, adrenalin, alpha-lobeline, and morphine fall into this category.

The use of 12 ounces (350 cc.) of physiological solution of sodium chloride given by rectum every four hours during the first few days has been suggested, as has the rectal administration of a 4 per cent sodium bicarbonate solution.

REFERENCES

1. Hamilton, A.: *Industrial Toxicology*, Harper and Brothers, New York, 1934, pp. 122, 141.
2. Russell, J. P.: "Carbon Monoxide on Highways." *California Safety News*, 23 (June), 1939.
3. Schillito, F. H., Drinker, C. K., and Shaughnessy, T. J.: *J.A.M.A.*, *106:* 669, 1936.
4. Brahdy, L., and Kahn, S.: *Trauma and Disease*, Lea and Febiger, Philadelphia, 1937, p. 26.
5. Stearns, Wm. H., Drinker, C. K., and Shaughnessy, T. J.: "The Electrocardiograph Changes in 22 Cases of Carbon Monoxide Poisoning," *Am. J. Heart Disease* (April), 1938.
6. Beck, H. G., and Suter, G. M.: "Role of Carbon Monoxide in the Causation of Myocardial Disease," *J.A.M.A.*, *110* (June 11), 1938.
7. Beck, H. J., Schulze, W. H., and Suter, G. M.: "Carbon Monoxide, A Domestic Hazard," *J.A.M.A.*, *115* (June 6), 1940.
8. Wood, F. C., Hedley, O. F.: "The Seasonal Incidence of Acute Coronary Occlusion in Philadelphia," *M. Clin. North America*, *19:* 151 (July), 1935.
9. Master, A. M., Dack, S., and Jaffe, H. L.: "Facts and Events Associated with Coronary Artery Thrombosis," *J.A.M.A.*, *109:* 541–549 (August 21), 1937.
10. McNally, Wm. D.: *Toxicology*, Industrial Medicine Publishing Co., Chicago, 1937.
11. Sandall, T. E.: "Later Effects of Gas Poisoning," *Lancet*, *2:* 857–859 (Oct. 21), 1922.
12. Haggard, H. W.: "Studies in Carbon Monoxide Asphyxia, I. The Behavior of the Heart," *Am. J. Physiol.*, *56:* 390, 1921.
13. Drinker, C. K.: *Carbon Monoxide Asphyxia*, Oxford University Press, New York, 1938.
14. Colvin, L. T.: "Electrocardiographic Changes in a Case of Severe Carbon Monoxide Poisoning," *Am. Heart J.*, *3:* 484, 1927–28.
15. Master, A. M., Dock, S., and Jaffe, H. L.: "The Relation of Effort and Trauma," *Indust. Med.*, *9:* 399 (July), 1940.
16. Sanger, E. B., and Gilliland, Wm. L.: "Severe Carbon Monoxide Poisoning with Prolonged Coma Followed by Transitory Psychosis, Peripheral Polyneuritis and Recovery," *J.A.M.A.*, *114:* 324 (Jan. 27), 1940.
17. Koch, K. G.: "Blood Transfusion in Treatment of Acute Severe Carbon Monoxide Poisoning," *München. med. Wchnschr.*, *86:* 126 (Jan. 27), 1939.

CHAPTER XVII

THE CYANIDES AND NITROUS FUMES

THE CYANIDES

The cyanides are dramatized as suicide agents, and the wide use of these compounds in industry is not appreciated. Like carbon monoxide, they act as an asphyxiant, but instead of combining with hemoglobin within the bloodstream the cyanides attack the tissue cells, retarding or stopping oxidation. This protoplasmic action results in the arrest of internal respiration with paralysis of the respiratory center. Since this action within the tissue cells takes place even in an atmosphere of pure oxygen, oxygen therapy is of little use in cyanide poisoning.

The chief cyanogen compounds are *hydrocyanic acid*, cyanogen, acetonitrile, propionitrile, methyl isonitrile, ethyl isonitrile, benzonitrile, and cyanogen chloride. Of these the most toxic is hydrocyanic acid. It is used chiefly for the effect the gas has upon vermin. Sodium cyanide has a wide use in industry as a bath in dipping- or treating-processes. Henderson and Haggard cite the following psysiological responses to various concentrations of hydrocyanic acid as reported by Kobert. Slight symptoms appear after several hours' exposure with from 20 to 40 parts of HCN per million parts of air. The maximum amount for one hour without serious disturbance is from 50 to 60 parts; dangerous after thirty minutes, 120 to 150 parts; rapidly fatal, 3000 parts. The rule of the Public Health Service is that an experienced workman may enter a room after fumigation if the air contains no more than 0.1 part per 1000 parts of air.

Occupational Hazards.—Fumigation of ships, dwellings, hotels, and apartment houses; the hardening of steel, iron, cutlery, and machine parts; cleaning or coating silver; electroplating; metallurgy; tanning; dehairing and dyeing; and in use as a fertilizer (calcium cyanamide).

DIAGNOSIS

Signs and Symptoms. *Acute Poisoning.*—In fatal cases there are rarely any witnesses to the demise. Certain patients who narrowly escaped death report their preliminary symptoms as constriction of the throat, dizziness, fulness of the head, nausea, and vomiting. Some of these patients have then gone into convulsions. Examination of these patients has revealed the eyes to be glassy, staring, and the

pupils dilated. The body is covered with a cold sweat, and frothing at the mouth occurs, with the frothing often blood-stained. Respiration is slow and shallow. Involuntary defecation or urination is common. For a time there may be complete paralysis. If recovery occurs there is usually headache, drowsiness, and difficulty in speaking. As with all asphyxiants, a transient glycosuria is not uncommon. A rosy red color of the skin is usually present, although varying degrees of cyanosis have been observed. To all these cited findings, an *almond odor* to the breath is confirmatory.

Cyanide asphyxia has been considered the quickest acting mode of death, yet McNally cites several interesting instances where the suicide performed an act before death ensued. For example, a man took a drink of cyanide in a hotel, then walked to the window and tossed the bottle out.

Chronic Poisoning.—Cases of chronic poisoning are extremely rare. Gastrointestinal symptoms predominate, characterized by flatulence, cramps, vomiting, and obstipation. The patient becomes increasingly nervous, and headache and dizziness exist. Irritation of the skin, with conjunctivitis, edema of the eyelids, and lacrimation, frequently appears in those continuously exposed, especially if exposed to cyanogen chloride. A *cyanogen rash* is characterized by itching, papules, and vesicles, with frequent secondary infection. Certain observers think the reaction is more the result of lime and other caustic agents than cyanogen. The writer has observed this rash in men doing electroplating, but was unable to ascribe it to the cyanogen primarily. Polycythemia is the occasional blood picture of the chronically exposed.

Tests.—Reagents are: (1) Ferrous sulfate, 1.5 per cent; ferric chloride, hydrochloric acid, and sodium hydroxide, each 10 per cent. To about 10 cc. of the suspected fluid (blood or gastric contents, etc.) two drops of the sodium hydroxide are added. Then, following the addition of 2 cc. of ferrous sulfate and 1 cc. of ferric chloride, the mixture is warmed, and hydrochloric acid is cautiously added, avoiding any excess. A blue precipitate shows the presence of hydrocyanic acid.

(2) Dissolve 10 mg. of ferrous ammonia sulfate and 10 cc. of ammonium acetate (5 to 10 per cent) in 50 cc. of water. If 1 cc. of this solution is added to a small amount of blood containing cyanide, a purplish precipitate occurs.

MEDICOLEGAL ASPECTS

The author has seen but one industrial death due to cyanide. Of the cases reported in literature, they have occurred most often among fumigators.

Temporary Disability.—In the acute surviving cases, disability ceases with the disappearance of the symptoms which ordinarily do not last longer than one week and possibly two. In less severe cases, symptoms cease within a few days. In chronic cases, recovery occurs usually within a week or so following the removal of the patient from the hazard. However, cachexia, gastrointestinal disturbances, or psychoses may result in a longer disability.

Permanent Disability.—There are no cases following survival reported.

Illustrative Cases:

Case I.—A fifty-eight-year-old citrus-grove sprayer fell from a ladder as he was ascending it to spray fruit. He was rushed to a small community hospital, where the attending resident physician was incited into immediate specific treatment for cyanide poisoning because the employer stated that the man was possibly overcome by the spray, which contained cyanide. Failing to react to the first injection of sodium nitrate and sodium thiosulfate, a second one was administered. The patient remained in a deep coma for a number of hours, with a blood pressure of 240 130. Upon regaining consciousness, he was unable to talk; had a complete paralysis of the right side; albumin, casts, and red blood cells in the urine; and N.P.N. of 75 mg. Six weeks later the patient died, and suit was filed by a dependent, claiming death was due to apoplexy precipitated by exposure to cyanide.

It was brought out at the trial that the deceased had been refused insurance five years previously because examination revealed hypertension. Two weeks prior to the accident he had complained to a fellow-employee of weakness of the right arm, which lasted for two days. For several months he had frequented a local drugstore for headache medicine. His attack occurred immediately after lunch. He had not sprayed for one hour and was just about to resume spraying when he fell unconscious. No test of the blood was made for the presence or absence of cyanide. The evidence pointed to an ordinary cerebral hemorrhage, preexisting causes for which were known. The effects of cyanide do not occur one hour after exposure as alleged in this case. There is no evidence that small doses would increase the blood pressure. The claim was denied.

Case II.—A male, aged thirty-seven, worked for twelve years for a magazine publisher as an "art painter." During that time his work exposed him to cyanide baths. The methods had been changed at intervals, but the cyanide element was constantly present. He first noticed a loss of weight, then marked constipation. Four or five months after the onset of the original symptoms, he had flatulence accom-

panied by abdominal cramps. One week prior to being examined at the clinic, vomiting had set in.

Physical examination was essentially negative. Apparent focal infection was absent. Heart and lungs were normal. The abdomen disclosed no palpable organs or masses and no specific point of tenderness. Neurological examination was negative. Laboratory investigation revealed the urine and stools normal; Wassermann, negative; gastrointestinal series, negative; red blood cells, 6,500,000; hemoglobin, 110 per cent; white blood cells, 9200. The patient was removed from his occupation. Within three days vomiting ceased; within two weeks his appetite improved, and a gain in weight was registered; abdominal cramps were less severe and disappeared entirely within a month.

Specific laboratory work was not possible, but the mild polycythemia was suggestive. In view of the case history that an exposure existed, that the length of exposure was adequate, and that no other cause for the man's condition could be found, it was considered compensable. There was no permanent disability.

Case III.—Three men were fumigating grapevines to kill "leafhopper," a vine parasite. The material used was calcium cyanide powder, applied by dusting machines mounted on a truck chassis. The machines throw the cyanide powder through the air to a height of about six feet and a distance of about twenty-four feet on each side of the truck, dusting two rows of vines at each trip. When calcium cyanide comes in contact with moisture, it is decomposed, giving off hydrocyanic acid gas, which is extremely poisonous. Dusting is usually done at night, when air conditions are quieter than in the daytime. The men work in pairs and are instructed to work upwind from the section already dusted.

Details of this accident are not known, but a crew of three men was missed one night at about midnight. When found, one of the men was dead, and the other two were unconscious. First aid was given to all the men until they were removed to the hospital. The two who were still alive later developed pneumonia but recovered, with no disability.

Gas-masks have now been ordered for all men on this ranch who handle, or are exposed to, cyanide, but it is a question how satisfactory the use of such masks will be. Gas-masks are essential equipment for use in emergencies. Special canisters, designed to give protection against hydrocyanic acid gas, are good for concentrations of the gas up to 2 per cent in air, but are rapidly exhausted at such a concentration. In addition, hydrocyanic acid gas is readily absorbed through the skin, so a gas-mask does not give full protection. A man working

in a concentration of the gas as low as 0.5 per cent may absorb enough of the poison through his skin to produce symptoms after a half hour's exposure. In any case, if gas-masks are used, the canisters should be replaced at least every shift, and oftener if the exposure is at all severe; and men should keep out of the gas as much as possible.

In this accident, it seems probable that sudden air currents carried the gas back into the men's faces and they were overcome before they could escape. Gas-masks would probably have enabled them to get away. The death of the one workman was compensable. The other two had a short temporary disability.

Treatment

Death is frequently so immediate that treatment is futile. Oxygen has no value, as mentioned in the opening paragraph. Many patients, however, survive for three or four hours, and hence adequate time for treatment is available.

Methylene-Blue Treatment.—Hanzlik and Richardson[1] outlined the most effective measures for treatment in cases of acute cyanide poisoning. Fifty cc. of 1 per cent methylene-blue solution (containing 1.8 per cent sodium sulfate) is injected intravenously, and is repeated if necessary until 200 cc. are injected. Frequently, consciousness and reflexes are restored before the first 50 cc. are completely injected; but if the patient lapses into unconsciousness, or manifests respiratory depression, resume the methylene-blue treatment. As quickly as possible, proceed with gastric lavage, using 5 per cent sodium thiosulfate; this oxidizes any unabsorbed poison. For lavage 3 per cent hydrogen peroxide or 0.2 per cent potassium permanganate solution may also be used if sodium thiosulfate is not at hand. Artificial respiration or carbon dioxide, from 5 to 7 per cent, with oxygen may be needed.

Circulatory and Respiratory Stimulants.—Caffeine sodium benzoate, 7½ grains (0.5 gm.), coramine, 1.5 cc., or metrazol, from 1½ to 4½ grains (0.1 to 0.3 gm.), intramuscularly may be needed for circulatory and respiratory stimulation.

Sodium Nitrite Treatment.—An alternative treatment is as follows: Give at once a slow and careful intravenous injection of 1 per cent sodium nitrite solution, in five divided injections, until 50 cc. are injected in about one hour. If improvement is manifested, but prognosis is still unfavorable, the injection may be cautiously continued, but is to be stopped at once in case of sudden collapse. Epinephrine, 1 cc. of 1:1000 solution, should be ready at hand to combat nitrite shock, if necessary. Fortify the nitrite treatment at once with the intravenous injection of 20 cc. of a freshly prepared 5 per cent

aqueous solution of sodium thiosulfate (filtered), and if necessary continue the injection up to a total of 500 cc., if possible. Treatment directed at further circulatory and respiratory stimulation is used as indicated in the first outline. The solutions used in these treatments can be readily sterilized by boiling for fifteen minutes. The methylene blue should not be dissolved in physiological solution of sodium chloride since precipitation occurs owing to a common ion (chloride) action.

Both the methylene blue and the sodium nitrite appear to act by producing methemoglobin, which in turn combines with the cyanides to decrease the poisonous effects of the latter. Actually, sodium nitrite forms more methemoglobin, but this probably does not contribute to physiological recovery from poisoning, and there is danger of sustained circulatory collapse to add to the already precarious state of the patient.

Sodium Nitrite and Sodium Thiosulfate Treatment.—Ingegno and Franco[2] treated two patients with 0.3 gm. sodium nitrite in 10 cc. given intravenously at the rate of 2.5 to 5 cc. per minute, and followed this by 25 to 50 cc. of a 50 per cent solution of sodium thiosulfate given through the same needle and at the same rate. They warned that the sodium nitrite and thiosulfate must not be mixed before administration. If signs reappeared or persisted, one-half the dose of antidotes was repeated one hour later. They also used amyl nitrite inhalations for from fifteen to thirty seconds every two or three minutes.

Diet.—A high fat diet has experimentally been shown to reduce the mortality from sodium cyanide poisoning.[3] In the rare case of chronic cyanide poisoning such a diet possibly would be of use in addition to symptomatic treatment. The use of sodium thiosulfate, 10 cc. of a 10 per cent solution intravenously, two or three times per week might be of value. Removal from exposure is, of course, at once necessary.

REFERENCES

1. Hanzlik, P. J., and Richardson, A. P.: "Cyanide Antidotes," *J.A.M.A.*, *102*: 1740–45 (May 26), 1934.
2. Ingegno, A. P., and Franco, S.: "Cyanide Poisoning," *Indust. Med.*, *6* (Nov.), 1937.
3. Meyer, A. R.: *Proc. Soc. Exper. Biol. and Med.*, *41*: 402 (June), 1939. Cited in editorial, *J.A.M.A.*, *114*: 49 (Feb. 10), 1940.

NITROUS FUMES

The exposure of nitric acid to air liberates its oxides, the chief ones being nitrous oxide, nitric oxide, and nitrogen dioxide. The profession is well acquainted with the use of nitrous oxide as an anesthetic. This oxide as well as nitric oxide needs no consideration at this time.

Nitrogen dioxide (peroxide, NO_2) is evolved when nitric acid contacts organic matter. It is an essential part of many industrial processes.

Occupational Hazards.—Nitrous fumes are present in the manufacturing of certain explosives (not gunpowder), certain lacquers, photographic films, metal etching and photo-engraving, metal dipping, such as the "bright dipping" of brass and copper, or wherever nitric acid or materials composed of nitrocellulose are handled. Carbon-arc booths are a hazard but are usually well protected by design and ventilation. These fumes may also offer a hazard to tunnel workers or miners.

Pathology.—As a result of exposure to nitrous fumes, arterial blood is rapidly turned into venous and becomes thick, tarry, and coagulates quickly. Hemorrhages into the various organs occur, especially into the gastro-intestinal tract. Marked edema of the lungs is found. The kidneys and liver have revealed lesions in those exposed. The most severe reactions, however, occur with the arterial blood and in the lungs. In the rapidly fatal case there may be no pathology other than congestion of the lungs.

Signs and Symptoms.—It should be emphasized that the *symptoms are usually delayed*. This latent period may be from a few hours to twenty-four or thirty hours. Unquestionably this offers difficulty in some instances in diagnosis, and unless the facts are known the etiology may be overlooked. No doubt, many cases of nitrogen dioxide poisoning have gone uncompensated. A physician friend of the author's, who was exposed to the fumes resulting from the Cleveland Clinic disaster, drove six miles to his home, felt exhausted, sat upon his front steps for nearly an hour, and then died.

Acute Exposure.—In the acute non-fatal cases, headache, dizziness, cough, palpitation of the heart, sometimes cyanosis, restlessness, and inability to sleep, are common. In certain concentrations, the workman may feel dopey. This narcotic or depressive action sometimes retards the cough reflex, and the patient is not warned of his danger. In contrast to the narcosis that may develop, Ebright[1] reports a case where the man became wild, irrational, and ran around butting against walls and trees. Pneumonia is a common sequela.

Chronic Exposure.—This leads to headache, sleeplessness, loss of appetite and weight, dyspepsia, constipation, and ulcers of the mucous membranes; Laws[2] reports a complete loss of vision in some. No proof exists that exposure may lead to chronic pulmonary disease, although certain clinicians have so stated.

Medicolegal Aspects.—It is felt that above 0.2 mg. per liter of air constitutes a dangerous atmosphere.

Temporary Disability.—This is indefinite and entirely dependent upon the severity of the dosing, as well as the presence of sequelae.

Permanent Disability.—This is not common, but may occur.

Illustrative Case:

Case I.—While working on a California aqueduct, a workman was caught in the smoke from a dynamite discharge. He continued working that day but that night was awakened by extreme dyspnea and coughing. Three days later he was transferred from the field first-aid hospital to Los Angeles. At this time he appeared to be in great distress and was apprehensive and restless. His temperature was 104.2 F.; pulse, 136; moist râles throughout both lung fields, with signs of bronchopneumonia in the right lung; and a continuous wracking cough, which was productive of a rather foul sputum. The urine was not abnormal, and the blood count was normal other than 22,600 white blood cells, with 95 per cent neutrophiles. Subsequently albumin and casts were present in the urine. During a period of three weeks, all signs and symptoms cleared, and the patient returned to work in one month after exposure.

The physician who first saw this patient out at the aqueduct was acquainted with the effects of nitrogen fumes. When he received a history of the exposure to the "smoke" from the dynamite, he was immediately suspicious of the cause of the man's illness. Temporary disability lasted one month. There was no permanent disability. A check-up two years later revealed that the patient was working and healthy.

Treatment. *Correction of Pulmonary Effects.*—Oxygen, which has been passed through a bottle containing 2 gm. ammonium carbonate to 1.5 ounces of water, should be administered to patients in acute cases by means of an intranasal tube. (McNally recommends a tube in each naris.) For relief of the pulmonary edema, intravenous 50 per cent glucose in amounts of from 50 to 100 cc., up to 200 cc., daily, is used; salyrgan, from 0.5 to 2 cc. intravenously, at intervals of three days, is also of much value in treating the pulmonary edema. Sedation for restlessness will be needed in many cases. If pneumonia appears as a complication, the use of specific chemotherapy, *i.e.*, sulfanilamide, sulfapyridine, or sulfathiazole, may be instituted if a predominant organism can be demonstrated in the sputum. In case the carbon dioxide combining power is low, the treatment should be that described for acidosis under "Methyl Alcohol."

Chronic Exposure.—Persons affected by chronic exposure may also be benefited by intravenous salyrgan. It is said that these patients often find sleeping in a rocking-chair easier than in bed. They will fre-

quently need acetylsalicylic acid or phenacetin, 10 grains (0.65 gm.), every four to six hours for headache, or if this symptom is more severe, codeine sulfate or phosphate, from $\frac{1}{2}$ to 1 grain (0.032 to 0.065 gm.), every four to six hours. Sedation with drugs such as sodium bromide, from 10 to 30 grains (0.65 to 1.3 gm.), phenobarbital, $1\frac{1}{2}$ grains (0.1 gm.), or sodium pentobarbital, $1\frac{1}{2}$ grains (0.1 gm.), three times a day, is needed at times for restlessness. Constipation may need attention. The mouth washes described under "Mercury" may be of benefit in ulcers of the mucous membranes. The addition of extra vitamins to the diet should be of value.

REFERENCES

1. Ebright, G. E.: "The Effects of Nitroglycerin on Those Engaged in Its Manufacture," *J.A.M.A.*, *62*: 201, 1914.
2. Laws, C. E.: "Nitroglycerin Head," *J.A.M.A.*, *54*: 793 (March 5), 1910.

CHAPTER XVIII

METAL-FUME FEVER, WELDING, AND GASES FROM CARBON ARCS

METAL-FUME FEVER

METAL-FUME fever is purely an *industrial disease*. It is characterized by a febrile reaction following the inhalation of finely divided particles of the metallic oxides. Among the working-men it is known as brass chills, brass-founder's ague, metal ague, zinc chills, spelter shakes, and by other names. It is an old disease referred to frequently in the literature and needs but brief description. It is most important that its *brief duration* and *absence of after-effects* be emphasized.

Pathology.—Actually there is no pathology resulting from this exposure, the reaction being physiological. It baffled the early experimenters as to just why the fumes produced an illness but the dry powder of these metals did not. Injecting soluble zinc oxide, Lehmann[1] was unable to produce noticeable symptoms. It was suggested that the inhalation of fumes killed bacteria in the respiratory tract which, when absorbed, caused symptoms. Lehmann thought it might be that the inhalation killed off the epithelial cells, the proteids of which when absorbed produced the symptoms. Drinker[2] held that it was a physical difference; that the particles of ordinary oxides are large, settle quickly, and rapidly collect in clumps to adhere to the sides of the trachea. The fumes on the other hand are so fine that they pass rapidly through the upper respiratory tract into the lungs.

Immunity.—Immunity to these fumes may be developed, but when it occurs there is leukocytosis, functioning as a defense mechanism. It is noticed that if a workman absents himself from his exposure for a few days, he will lose this immunity. Many workers report a state of well-being during the week, but after a week-end they will notice various grades of reaction upon a return to work.

Certain workers have reported disturbance of liver function, glycosuria, and increased blood pressure and pulse rate.

Signs and Symptoms.—These are closely comparable to influenza or la grippe. Chills come on usually after the patient has left work and last for one or two hours, rarely longer. The body aches all over; nausea and not infrequently vomiting may occur; fever sets in and may reach 102° F., seldom higher; the throat is dry, and there may

be a dry cough. Weakness, lassitude, or exhaustion are complained of. The leukocyte count may range from between 12,000 and 16,000 and may persist after all other symptoms disappear. A mild leukocytosis is present as previously noted in those without symptoms.

Chronic symptoms are rarely present other than in some instances when a complaint of generalized lassitude is made. Metal-fume fever is acute in onset and short in duration, the patient usually making a complete recovery within twenty-four or thirty-six hours. Pronounced chronic symptoms should arouse suspicion of lead's being the etiological factor.

Medicolegal Aspects. *Temporary Disability.*—This rarely lasts more than one or two weeks. In mild instances the patient loses no time from work, merely changing his job or receiving better protection. Patients in more acute cases usually return to work in several days.

Permanent disability as a result of metal-fume fever is unknown. Cases of death have been reported, but no instances of recovery with permanent sequelae are reported.

Illustrative Cases.—A Negro, employed by a metal company, noticed a back-pain late one afternoon. He went home, changed his clothes, and reported to the clinic alleging back-sprain. While in the clinic he developed marked chills, began to ache all over, vomited on four occasions, had a temperature of 102.2° F. and a white count of 13,200. His headache became pronounced. During the early hours of the next morning a second Negro who had been working with this first patient was admitted with comparable symptoms.

These two men had engaged for the first time to dump old batteries and other scraps of metal into a reclaiming furnace which had a defective door, permitting the escape of fumes. Both recovered within two days. Their physical examinations were negative for focal infection or systemic disease.

Treatment. *La Grippe Regimen.*—No specific therapy is indicated in these cases. Patients may be treated as though they had la grippe. The administration of acetylsalicylic acid, 10 grains (0.65 gm.), every four to six hours combined at times with codeine phosphate, from ½ to 1 grain (0.0325 to 0.065 gm.), will afford relief. The forcing of fluids (water and fruit juices), the use of hot drinks, and rest in bed are of value. These patients will become symptom-free without treatment after absence from exposure for a short time, but for their composure the above regimen is prescribed.

Prophylaxis.—The most valuable point, however, is to adopt an adequate medical and engineering program for prevention of the metal-fume fever. The toxic fumes should be controlled at their source

by having operations which give rise to metal fumes carried on, if possible, in a closed process. It will usually be more practical to employ exhaust ventilation with hoods over the process producing the fume. The ventilating system should be designed so that a current of clean air is drawn past the operator and away from him toward the work. Masks and respirators may be provided for protection under unusual circumstances.

REFERENCES

1. Hamilton, A.: *Industrial Toxicology*, Harper and Brothers, New York, 1934, p. 82.
2. Drinker, P., Thomson, R. M., and Finn, J. L.: "Metal Fume Fever; Effects of Inhaling Magnesium Oxide Fumes," *J. Indust. Hyg.*, 9: 187, 1927.

WELDING

The process of welding is an important, necessary step in the structural-steel business, in repair shops, construction plants, and wherever solid metals are in use. Such operations are not without a hazard. Too frequently men are employed in small, non-supervised shops or in confined areas such as ill-ventilated rooms, vaults, or tunnels. Forced ventilation is frequently absent and respirators ignored.

Occupational Hazards.—The hazards of welding are from: (1) fumes, smoke, and gases; (2) radiation from the arc; (3) fire; and (4) electric shock. It is the first of these exposures that concerns us chiefly. In passing, it should be mentioned that protection from radiation is essential. This radiation is often a mixture of ultraviolet light, infra-red rays, and visible light. Exposed parts of the body should be well protected, but the eyes especially should be covered with glasses fulfilling the requirements of the federal specifications. Occasionally, claim is made that sexual impotence has resulted from the exposure to radiation. These claims and their remote possibility induced investigation along these lines. Today there exists no proof that this impotence occurs.

Respiratory Hazards from Welding.—These may be divided into three general classes: (1) poisonous substances which may be generated by the arc; or by (2) faulty combustion; and (3) exhaustion of oxygen in air.

Lead Poisoning.—This is the principal and typical hazard in the class of poisonous substances volatilized by heat. It occurs very frequently when metals covered with lead paint are cut by either the electric arc or the oxy-acetylene flame. The heat volatilizes the lead, forming lead fume, which results in rapid and severe lead poisoning to the unprotected welder.

Metal-Fume Fever.—Welding or cutting brass or zinc (galvanized sheets) frequently results in metal-fume fever, commonly called "zinc chills" or "zinc ague" (discussed earlier in this chapter). Even though

this illness is temporary, it is far from pleasant. Other substances to be found as a coating on certain widely used rods include silicon dioxide, aluminum oxide, titanium dioxide, calcium, magnesium and sodium oxide, iron, manganese, calcium fluoride, and carbon dioxide. It has been suggested that these coatings are toxic when volatilized, but there is as yet little information on this point.

Gas Poisoning.—Poisonous gases, of which nitrogen peroxide (discussed elsewhere in this book) is probably the most important, are generated by an electric arc. Only recently the death of a welder was attributed to this gas. In this instance, the worker was electrically welding together two halves of a hot-water tank in a room of about 4000 cubic foot capacity. All welding was on the outside of the water tank. The man became unconscious while welding and died six days later in a hospital.

There are cases on record of fatal gassing due to nitrous fumes generated by oxy-acetylene torches. A. D. Brandt[1] tells of a case in which several men were working with large oxy-acetylene burners on a heavy steel crosshead in a ship compartment. The compartment had a capacity of approximately 5000 cubic feet and was being ventilated at the rate of about 240 cubic feet per minute by means of a fan. After about forty minutes' work, several men collapsed, and one died in a hospital thirty-two hours later. The symptoms were typical of poisoning by nitrous fumes, and tests of the air showed nitrous fumes to be present in large amounts. Mr. Brandt also states that if welding, particularly with oxy-acetylene torches, is done in closely confined spaces, the oxygen of the air may be reduced to a dangerous level. Fortunately, cases of illness or death due to this cause are rare.

Other Pulmonary Disturbances.—Cadmium fumes following the welding of cadmium plates may produce severe pulmonary congestion. Chrome plating often irritates the skin. Carbon monoxide may arise from welding.

The writer is frequently confronted by welders who claim that their exposure over a period of time has affected their lungs. They complain of an irritating cough and that at intervals their lungs "hurt." In no instance have we noted any characteristic lung changes, except in one man who not only was a welder but had been exposed over a considerable period of time to iron dust. Britton and Walsh[2] studied a group of 1000 welders and concluded that changes in the chest films may be present in those exposed for many years. However, these changes were not accompanied by any disability.

Treatment. *Prophylaxis.*—Preventive measures include protection of the eyes from flashes, glare, and radiant energy; respirators for protection against inhalation; and protective coverings of the exposed

parts of the body. Indoor welding should be done where the walls are painted black to reduce the reflection, or booths should be provided. Outdoor welding should be done in portable canvas booths.

The treatment of metal-fume fever, and nitrogen peroxide, lead, carbon monoxide, or cadmium poisoning, when they occur as a result of welding, is indicated under those subjects.

Electric Ophthalmia.—When this occurs, a superficial injury to the corneal epithelium with exposure of superficial nerve terminals will have occurred. The pain is frequently very severe and may require one of the following for relief: morphine sulfate, $^1/_4$ grain (0.016 gm.); dilaudid hydrochloride, $^1/_{16}$ grain (0.004 gm.); or pantopon, $^1/_2$ grain (0.022 gm.). Both eyes should be completely bandaged, and locally anesthetic ophthalmic ointments, *e.g.*, 2 per cent holocaine hydrochloride, 2 per cent butyn, or 4 per cent metycaine, are used.

REFERENCES

1. Brandt, A. D.: "Decreasing the Respiratory Hazard in Welding," *The Welding Engineer*, *21*: 21 (Aug.), 1936.
2. Britton, J. A., and Walsh, E. L.: "Health Hazards of Electric and Gas Welding," *J. Indust. Hyg. and Toxicol.*, *22* (April), 1940.

GASES FROM CARBON ARCS

Occupational Hazards.—The commercial use of the electric arc was formerly largely confined to outdoor illumination. Therefore, any product of combustion of the electrodes presented no problem or hazard to health. In the last few years, however, the carbon arc has been used for radiation or as a light source for projection purposes within rooms and small booths. Frequently the question arises as to the harmful effects, if any, from this source.

Types of Carbon Arcs.—Carbon arcs may be divided broadly into three types,[1] the plain or low-intensity carbon arc, the flame arc, and the high-intensity arc. In the flame arc the primary source of radiation is the arc stream or flame between the electrodes. The carbons from flame arcs have large cores containing a relatively high percentage of "flaming material." This material may be metal, a mixture of metals, metallic oxides, or other compounds, depending upon the specific type of radiation desired.

The high-intensity arc is always operated on direct current at high current densities, and the source of light, which has the highest intrinsic brilliancy of any commercial illuminant, is a cup-shaped crater in the positive carbon. The carbons for this type of arc have large cores containing relatively large quantities of compounds of the metal of the cerium group of so-called "rare earth." Carbons of these types of arcs are made in a variety of sizes according to the power input

and currents, and in certain cases it is customary to plate the carbons with copper.

Gaseous Products.—The burning of carbons in any type of lamp gives rise to a variety of products, part of which are solid, originating from the volatilization of the impurities or substances added to the carbon to impart specific properties to the arc, and part of which are gaseous.

Carbon Dioxide.—Of these gaseous products carbon dioxide is the chief substance, with a small amount of carbon monoxide, and a fixation of atmospheric nitrogen. These amounts are far below the hazard level. When high-intensity carbons contain relatively large amounts of rare-earth fluorides there is no evidence of the presence of gaseous fluorine compounds. Tests fail to show the presence of any gas reacting with silver nitrate such as hydrogen sulfide, phosphin, arsine, or halogens. Investigators have concluded that the only toxic gaseous products that might be present from this type of combustion are the oxides of nitrogen. These gases are considered in a separate chapter.

Ozone.—It has been questioned whether or not ozone is present in this type of combustion. This question is not definitely proved one way or the other, owing to the reactions common to it and to the oxides of nitrogen. It would seem from the work of various investigators that the presence of ozone is not probable. It would also seem that its intense odor in exceedingly small concentrations would overpower any faint odor of nitrogen compounds and thus betray its presence. Hill and Flack[2] state that there are no respiratory effects if the concentration is so low that it cannot be detected by the sense of smell.

Oxide of Nitrogen.—Of the oxides of nitrogen only nitrous oxide can be classed as a non-irritant to the upper respiratory tract. The others, nitrogen trioxide and nitrogen pentoxide, react with alkali and moisture in the respiratory tract, causing an irritation. When nitrate is formed, there is a definite systemic action, if absorbed. If nitric oxide (NO) is given off from the arc, it will react with atmospheric oxygen to form NO_2, and with water, then forming nitrous and nitric acids. These are more commonly known as "nitrous" fumes and are insidious, due to the fact that they cause little irritation and little discomfort when inhaled in small concentrations.

Moving-Picture Projection Booths.—With the advent of the moving-picture booth the question arose regarding the health hazard to the operators in these booths. The question was thoroughly investigated by MacQuiddy.[3] After laboratory tests on guinea-pigs exposed to the undiluted fumes arising from an electric arc there was revealed the spectrum of methemoglobin in their blood samples. It was felt

from this guinea-pig test that the arc fumes might be toxic. It was MacQuiddy's conclusion that undiluted gross fumes from the electric carbon arc are toxic to mice, rats, guinea-pigs, rabbits, and cats when inhaled. The pathological changes indicated that the oxides of nitrogen produce these changes. Tollman,[3] La Towsky, and Bayliss further investigated the action of these combustion products. They concluded that the concentrations of the oxides of nitrogen are high enough to be toxic if exposures to the undiluted gases occur, but such conditions cannot occur with properly installed arc lamps. They further concluded that any hazard from these gases is easily overcome by adequate ventilation.

Since the operation of a movie machine is an employment of a widespread nature and not confined to region or country, it was deemed necessary to discuss this misunderstood hazard briefly and to point out that actually there exists little or no hazard.

Treatment.—The chief hazard here, when it does occur, is from the oxides of nitrogen. The prevention and treatment of such poisoning have been discussed under "Nitrous Fumes."

Prophylaxis.—The important point here again is prevention. The major portion of such prevention consists of adequate ventilation. It has been suggested that each projector lamp should be connected to a flue in which fumes from arc combustion are exhausted by means of motor-driven fans to the out-of-doors. This fan should turn on automatically as the arc is struck, and the exhaust should run at a minimum of 12 cubic feet a minute, with a rate nearer 100 being preferable.

REFERENCES

1. Coltman, R. W.: "Gases from Carbon Arcs," *J. Indust. Hyg. and Tox.*, *20* (April), 1938.
2. Hill, L., and Flack, M.: "The Physiological Influence of Ozone," *Proc. Royal Soc.*, *84*: 404, 1911.
3. MacQuiddy, E. L., Tollman, J. P., La Towsky, L. W., and Bayliss, M.: "The Combustion Products of the Carbon Arc" *J. Indust. Hyg. and Toxicol.*, *20*: 312 (April), 1938.

PART III

THE METALS

Until recently, metal poisoning was considered as confined to lead, mercury, and arsenic, but with the development of specific alloys, the toxic properties of all metals demand inquiry. Frequently a new process is established without prior knowledge of its effects upon the body having been determined.

Facts about Metals.—The following fundamental facts are important in the evaluation of any one of the metals:

1. Certain pure metals are harmless, but a compound of them or their acid salts may be poisonous.

2. The hazard offered by industry is seldom that of a single metal but rather a combination of metals. Frequently fumes from gases or solvents are present with the metal fumes.

3. Deleterious effects usually result from accumulation of minimal amounts, rather than a single exposure to massive amounts.

4. While many metals are irritating to the skin and mucous membranes, bodily damage is by way of the respiratory tract.

5. It is well to remember that when a workman in a metal hazard becomes ill, the clinical picture may not conform to the known syndrome of a single metal, but may be altered by the toxic effect of several metals.

6. Injurious exposure is frequently limited to a specific occupation or process within an industry; and, therefore, simply because a person is employed where metal is processed, it does not follow that he is experiencing metal absorption.

7. The clinical picture produced by the metal poisonings may be simulated by disease of non-occupational origin. In industrial medicine it is obligatory that the examination be complete and a thorough laboratory investigation be instituted.

CHAPTER XIX

LEAD

Few, if any, of the occupational diseases are so consistently erroneously diagnosed as lead poisoning. It is a sad commentary upon medical education that a disease known to the ancient Greeks, Latins, Arabians, and studied by physicians throughout the subsequent centuries, inciting profuse material in medical literature, and having such well-defined laboratory findings, should remain an enigma to the profession at large.

At our clinic, fully 60 per cent of the referred cases have been previously diagnosed as "lead poisoning" upon a single symptom or a combination of symptoms, without sufficient laboratory study. Similar inadequate presentations are made frequently before the Industrial Accident Commission. By the same process of reasoning, one might well contend that a person with unequal pupils has syphilis. Like syphilis, this disease is protean in its manifestations. Concerning syphilis, one assumes that with positive serology and clinical symptoms, the diagnosis is correct even though no history of exposure or contact is obtained. However, in lead poisoning, the history of exposure is all-important. It, combined with the clinical picture and the laboratory findings, forms the triad for diagnosis.

Occupational Hazards.—Lead is ubiquitous in industry. The United States Department of Labor lists well over 150 occupations offering this exposure. Thus is its widespread prevalence indicated. Some of these, however, present no perceptible danger even over a period of many years, as, for instance and in spite of traditional belief, the printing trade. Smelting, refining, burning, and processes where scrap is melted offer the greatest hazard. Two local companies which melt old storage batteries had a high incidence of lead poisoning until better supervision was instituted. Other dangerous sources are in using litharge, in making glaze for pottery and tile, in the manufacturing of batteries, and in the making of varnish. Enameling of bathtubs, stoves, sinks, fixtures, and metalware should also be mentioned. The addition of lead to motor fuel (tetraethyl lead) received consideration under "The Petroleum Hydrocarbons."

Modes of Entrance.—Lead enters the body through the lungs, by ingestion, or through the broken skin. The last mode of entrance has

no significance industrially. The greater portion of ingested lead, if not all of it, passes through the intestinal tract without absorption and is evacuated. An equivalent amount of lead is 100 times as toxic when inhaled as when swallowed. These facts should readily discount claims made that one has contracted lead poisoning from contamination of the hands. However, while this chapter was being written, Raulston informed the author that he was investigating lead poisoning among roofers in Los Angeles County. Subsequently, Magnuson and Raulston[1] reported eight cases of lead poisoning among roofers who were in the habit of holding galvanized roofing nails in their mouths. The patients cited had been using fourteen-gauge "hot dip" galvanized nails of various sizes. Adherence to the requirements of the Federal Housing Administration had not been conformed to, and analysis revealed these nails to contain lead from 0.9 to 4.7 per cent in the zinc coating. Most of these patients presented symptoms which simulated appendicitis. At or about the same time the author received a letter from J. P. Russell, M.D., Chief of the State Industrial Hygiene Service of California, detailing an investigation among shinglers in the area around San Francisco. The urine and blood of these men were found to contain abnormal amounts of lead. The nails were examined and found to contain an abnormal or prohibited amount of lead.

Pathology. *Effects of Ingestion.*—In 1930, Kehoe[2] reported his experimental study on the effects of the ingestion of lead. Two human subjects were placed on a prolonged regimen of increased oral lead intake of soluble lead, one of them receiving 2 mg. daily for a period of six months, the other a daily dose of 1 mg. for thirty months. Complete clinical observations were recorded, as well as laboratory data of the microscopic blood picture, the excretory response, and the changes in the levels of lead concentration in the urine and blood. Most of the lead passed through the intestines unabsorbed. Kehoe stated that neither of his subjects had had any symptoms of plumbism, not even the vague type seen sometimes in borderline cases. Furthermore, the lead metabolism had not been disturbed in either one following acute upper-respiratory infections. This study of Kehoe is introduced, not to refute the work of Magnuson and Raulston, and Russell just previously mentioned, but rather to serve this warning: any case in which industrial plumbism from ingestion is alleged deserves a most thorough investigation from all angles before it can be accepted. In fact, any illness, occupational or non-occupational, attributed to small amounts of ingested lead should be seriously doubted until all other possible agents have been ruled out. Aub[3] emphasized this last point emphatically in a letter to the editor of the *Journal of the American Medical Association:*

Two months ago, in the January 20 issue of The Journal, you published an article by Drs. Rathmell and Smith to which I must take exception, particularly as it followed so soon after a paper by Dr. H. B. Williams in which also many experts doubt the diagnosis. In this article the child is supposed to have had acute lead poisoning due to only an infinitesimal quantity of lead obtained from orange juice put into an aluminum container. It seems to me unwise to give great publicity to a so-called established case of lead poisoning in a child who is said to have ingested less than 0.02 mg. of lead. Most persons ingest more than that every day in their drinking water, with absolutely no known deleterious effects. An analysis of the dishes was mentioned but no report on this was made, and it is obvious that no accusation can be made without that, particularly as aluminum dishes should contain practically no lead. Besides, lead does not usually give the acute symptoms ascribed to it in this article. One substance that I know of, namely antimony, may give gastrointestinal symptoms such as vomiting, but to incriminate an infinitesimal amount of lead without knowing whether antimony was present in the juice is distinctly unscientific.

I write this vigorous letter because too much publicity is being given to minor exposures to lead these days. This is particularly true with regard to lead taken by mouth, which is so much less toxic than inhaled lead. That lead may produce deleterious effects there can be no question, but there is no evidence that a small fraction of a milligram can produce the effects ascribed to it in this article.

Fate of Lead in Body.—As in the case of all dusts, inhaled lead dust is resisted in part by the expulsive action of the cilia. Once past this line of defense, it enters the bronchioles and alveoli to be eventually absorbed into the general circulation. Little or no reaction within the lung tissue occurs, although fine metallic lead has been thought to cause some proliferation of fibrous tissue in contrast to the nonirritating action of the more easily phagocytized lead compounds. The fate of lead in the blood is today a controversial point. For years many have held to the original opinion of Aub[4] and his co-workers that lead is transported as a finely divided colloidal lead phosphate to be deposited as a tertiary lead phosphate in the bones. Once deposited, it is held that lead metabolism closely parallels the action of calcium. Kehoe questions this action of lead.

Storage in Bone.—Following his original work, Aub, along with Robb and Rossmeisl,[5] found that most of the lead in bone was located in the trabeculae, not in the cortex. Since the trabeculae act as a storehouse for available calcium, it was assumed that lead stored here was available and more easily mobilized. As calcium is disturbed by

bodily functions, so is stored lead. Disturbance of this calcium repository by acute infections or toxic states also sets lead free into the circulating system. From this accepted principle has arisen the axiom that "once leaded, always leaded." While this may be true in principle, it is not always true in fact. The author had a patient who was treated two years previously for proved lead intoxication and who subsequently developed pneumonia with no apparent exacerbation from action of the stored lead. Nevertheless, it is thought that in the treatment of acute lead intoxication and in the treatment of leaded patients subsequently ill from some infectious process, lead metabolism parallels that of calcium. Opposed to this viewpoint is the opinion of Kehoe. In November, 1939, the author had a case in which the urinary findings and the blood findings were not according to the accepted ratio. By correspondence, the case was discussed with Dr. Kehoe. In no uncertain terms he disclaimed the views mentioned in the preceding paragraph as follows:

I would say in relation to this whole question of fixation of lead in the body, that our experience and observations will not permit us to accept the hypothesis that lead can be bound in stable form in any part of the body, with the possible exception of the nerve tissues. The idea that lead is fixed in stable form in the skeleton either normally or through any kind of therapy is, in our opinion, untenable. In fact, all of our evidence tends to show that if the lead content of the body is elevated above the normal, the excretion of lead in the urine will be more or less correspondingly elevated. The role of calcium and phosphorus in relation to lead metabolism has not been worked out. We have never found any evidence that such therapy produces more than minor changes in the distribution of lead in the body and in the rate of excretion from the body. We do not on this account, condemn the use of calcium or phosphorus, or both, in the treatment of lead poisoning, but we do, however, believe that whatever benefit is achieved is not the result of mobilization or demobilization of lead in relation to depots of deposit.

Opinions on Deleading.—In contrast to the opinion and investigation of Kehoe, Gray and Greenfield [6] cite two cases illustrating the dangers of deleading in acute lead poisoning and four cases illustrating the increased lead stream with a high calcium diet. In addition to calcium they urge an adequate phosphorus intake. They believe that deleading may be of aid in a differential diagnosis in isolated instances. The importance of deleading in the differential diagnosis is the finding of lead in biological materials below that which may be considered normal. Again they state: "Deleading may be of value occasionally in continued litigation to determine whether an abnormal storage

of lead exists and is responsible for the continued subjective symptoms in the absence of objective findings." Also, "if after deleading for a suitable period the amount of lead excreted in the biologic materials is within normal limits, the symptoms may be due to the toxic effects of lead absorbed during the previous exposure and not to the waves of liberation."

Symptoms Only in Presence of Circulating Lead.—These conclusions reached by Gray and Greenfield are difficult to understand. Regardless of the opinion on whether lead is stored or not and whether calcium or phosphorus aid in that housing, it is universally believed that symptoms occur only in the presence of circulating lead. Certainly there exists ample evidence of this, and if symptoms continue after lead is absent from the excreta, the patient must be considered a malingerer or a neurotic, with the exception, of course, of patients in whom the nervous system has been involved.

Effect on Red Blood Cells.—Abnormal or excessive absorption of lead disturbs the hematopoietic function. The resulting anemia is not because of diminished production of blood, but is due to the destructive changes to the red blood cells in circulation. The investigations of Aub, Reznikoff, and Smith[7] show that the surface of the red blood cells is affected by lead, causing them to shrink, lose their stickiness and agglutination reaction, and become increasingly brittle, hemolyzing on the slightest trauma. But while these surface changes occur, the interior of the cells is not affected, and the physiological properties of the hemoglobin remain normal. The important point, however, is that this loss in circulating red cells is compensated for by a regeneration of the erythrocytes. As these young cells enter the bloodstream they are affected by the action of the lead so that their basophilic substance is coagulated, and thus they are recognized microscopically as stippled cells. This finding of *stippling*, while it has its confirmatory value, is a prevalent stumbling-block in accepting or denying cases of lead intoxication. This point will be discussed under "Diagnosis."

Effect on White Blood Cells.—Regarding the effect of lead upon the white cells, there appears to be no definite uniformity of opinion. Some cases have been characterized by leukocytosis; others by leukopenia. An increase in the monocytes was reported by Brookfield, who thinks it is due to the stimulation of the reticulo-endothelium system by lead. But others, particularly Lenzi, look upon this monocytosis as being common to all forms of heavily cationed poisonings. The relation of large to small lymphocytes was considered an early diagnostic sign by Ferguson and Ferguson.[8] They believe that the ratio between the large mononuclear lymphoid cells to the small lymphocytes has a closer relationship to the clinical condition than

the stippled-cell count. Stippled-cell counts depend much upon the stain used, the technic of staining, and the visual acuity of the observer, whereas the determination of the ratio of the large to the small lymphocytes is much less dependent upon personal factors or technic. The Fergusons pointed out that the precise differentiation between the monocytes and the large lymphocytes may be difficult, and they, therefore, included both types in the classification of the large mononuclear lymphoid cells. Shiels,[9] in his investigation of this opinion, found that absorption of lead causes an increase in the ratio of large lymphocytes and monocytes to the small lymphocytes. A fall in this ratio below 2:1 while the patient is exposed to the hazard is associated with definite symptoms of lead poisoning. Generally speaking, the more severe the case, the lower the ratio. He concluded that the magnitude of this ratio is more closely associated with the clinical conditions than is the stippled-cell count, and it is a simple and useful indication by which to judge the imminence or otherwise of lead poisoning and is an aid in diagnosis.

Smith, Rathmell and Marcil[10] offered a study of shiftograms and hemograms for the early diagnosis of plumbism but concluded that the hemographic indication was not pathognomonic of lead poisoning. The action of lead upon blood platelets offers no diagnostic import.

Blackman[11] found intranuclear acidolphic inclusion bodies in the tubular epithelium of the kidneys and in liver cells in twenty-one cases of lead poisoning occurring in children, and similar inclusion was reproduced in the kidneys of guinea-pigs, mice, and rats by adding lead to the diets of these animals. Like the intranuclear inclusions of virus diseases, the majority are stained by dyes.

Effect on Nervous System.—In addition to the effect lead has upon the blood system, it also affects the nervous system in varying degrees. The first, and usually the most pronounced, effect is that it appears to have a selective action upon the smooth muscle of the intestine, producing increased tone with loss of motility. The colic of lead poisoning is probably due to the constriction of the intestines and the loss of mobility, similar to obstruction. Paroxysmal spasms with intense hyperacidity, similar to ulcer states, have been held as the cause by some. Many investigators emphasize the prevalence of gastric ulcers among victims of chronic plumbism. Lead encephalitis has been reported in older literature, although it is rare today. Lesions of the cranial nerves have also been reported, and paralysis of the muscles due to nerve involvement, of which the wrist drop is a well-known example, is known but rarely seen. It is apparent that when palsy occurs, it is in those muscles most in use or fatigued and, therefore, need not be confined to the wrist, as is so classically claimed.

DIAGNOSIS

Signs and Symptoms. *Acute.*—When it occurs, the acute phase is usually in the form of metal-fume fever as a result of inhalation of massive doses. There is general malaise, often fever, mild to intense abdominal pain, nausea, and possible vomiting, diarrhea (rarely, early constipation), headache, numbness of the limbs, occasional stupor, and in severe cases, vomiting. Hamilton states that myalgia belongs to chronic plumbism, but we have seen it in three acute cases within the past two years. The urine may be scanty with albumin and casts.

Chronic.—The symptoms found in acute poisoning are usually present in the chronic form but to a less degree. Abdominal pain, for instance, is almost constantly noted, but it is usually not of the severe, colicky type. Increasing fatigue, listlessness, and mild loss of weight characterize the chronic case. The appetite is poor and constipation common.

The value in Table 11 is that it makes a nice distinction between the very early and mild lead absorption and the more advanced but inactive stages, as well as the advanced active stage, with the proper allocation of the corresponding symptoms. From these multiple possible symptoms, it might be well to have fixed a typical average case.

The patient is usually pale, easily fatigued, lacks interest in extraneous activities, complains of more or less constant headache, has no appetite; frequently he complains of restlessness and insomnia, and constipation becomes pronounced. To this point he may still not seek medical advice unless he is employed in a hazard where supervision is adequate and men are informed. With the onset of intestinal pain, however, he presents himself for relief. Rarely does the workman continue in an exposure to the point of having the nervous system severely damaged, or rarely is he permitted to because of the hygienic principles ordinarily instituted in industrial plants.

General Principles of Diagnosis.—"Lead poisoning" is an inaccurate term which fails to distinguish the actual state of one presumed to be affected by lead. Lead is a natural constituent of the soil, water, vegetation, and air; and, because of this, human beings the world over have lead, to a slight extent, in the body tissues. If this were kept in mind, erroneous assumptions would not be so frequent. A patient should be classified as having:

1. Normal lead absorption
2. Abnormal lead absorption, or
3. Lead intoxication

For example, one may show lead absorption in the excreta or blood in excess of the recognized normal amounts, and yet have no

TABLE II. CLINICAL SIGNS AND SYMPTOMS OF PLUMBISM*

Group I	Group II	Group III
Suggestive Evidence of Lead Absorption	Suggestive Evidence of Incipient Intoxication and Inactive or Arrested Plumbism	Suggestive Evidence of Definite, Advanced, and Active Plumbism with Acute Manifestations
General Symptoms		
Patient becomes easily flustered, moody, restless and excitable	Pallor	Anemia
	Jaundice	Inanition
	Slight lead line	Lead line
	Arthralgia	Arthralgia
General feeling of malaise	Slight inanition	Jaundice
	Fatigued easily	General weakness
	Hypotension to normal	Hypertension
	Slight pyrexia	Pain in chest
		Wrist drop
		Foot drop
Digestive System		
Persistent metallic taste	Metallic taste	Metallic taste
Slight anorexia	Coated tongue	Coated tongue
Slight constipation	Anorexia	Anorexia
	Constipation	Marked constipation
	Slight abdominal colic	Paroxysmal colic
		Nausea and emesis
		Rigid abdomen
		Blood in stool
Nervous System		
Irritability	Slight frontal headache	Severe frontal headache
Uncooperativeness	Slight tremors to parkinsonian syndrome	Tremors
		Confusion
	Slight ataxia	Insomnia
	Insomnia	Convulsions
	Palpitation	Fibrillary twitchings
	Increased reflexes	Neuritis
	Increased irritability	Visual disturbances
	Eyegrounds may show choking of optic disks	Encephalitis
		Hallucinations
		Coma
		Paralysis
		Cerebral palsy
Renal Symptoms		
Lead which fluctuates between normal limits and a very slight rise	Trace of albumin and few granular casts in urine	Toxic nephrosis
	Lead which fluctuates between normal limits and a positive rise	Albuminuria
		Casts in urine
		Hematoporphyrinuria
		Hematuria
		Positive but fluctuating lead findings

*Smith, F. L., Rathmell, T. K., and Marcil, G. E.: "Early Diagnosis of Plumbism." *Am. J. Clin. Path.*, 8: 471, 1938.

toxic symptoms. If the meaning of the word is to be adhered to, then one has lead poisoning only when one is intoxicated from lead.

The Blue or So-Called "Lead" Line.—One is tempted to state that it would improve diagnosis if this phenomenon had never been described. Its color is not blue but rather a grayish black; it is not actually a line but a series of dots or markings along the free margin of the gums. These deposits may also be found on occasion on the anal margins. A hand lens may aid in bringing out the specks. Defective hygiene, pyorrhea, and staining of the teeth may all be mistaken

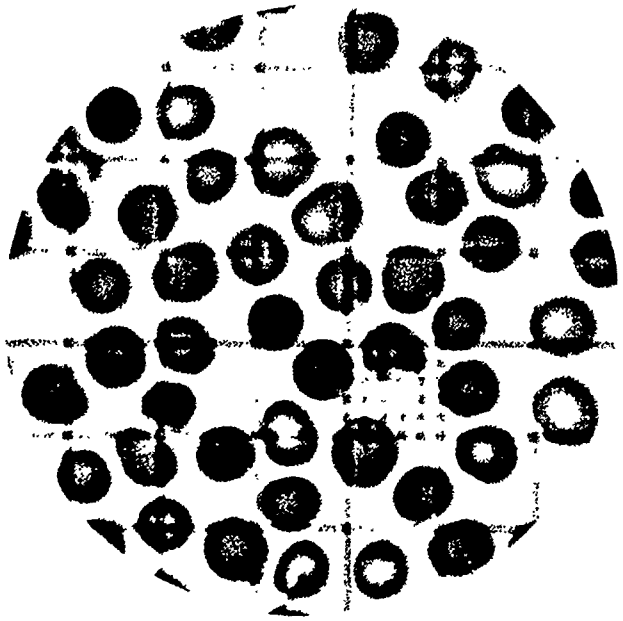


Fig. 38.—Section of fixed unlaked portion of blood smear used for the determination of total erythrocyte count and cellular abnormality. Photomicrograph with 10 · ocular and oil immersion objection. Straight lines shown are from Whipple grid used as a guide for quantitation. (Hyler and Bradley.)

for a lead line. When present this line is no indication of the amount of lead in the body.

Stippling or Basophilia.—It has previously been mentioned that when the red cells meet the destructive influence of lead in the circulating blood, young cells are called forth, and their basophilic substance becomes coagulated. When stained, this granulation has a stippled appearance. If found in a suspected case, the conclusion is often erroneously made that "lead poisoning" exists. Unfortunately, there exists a general impression that stippled cells are pathognomonic of plumbism. *This is not true.* These stippled cells may often appear

in pernicious anemia, secondary anemia, leukemia, hemolytic jaundice, neoplasms, poisoning from benzene, aniline, or any of the chlorinated hydrocarbons, and many other diseases. Furthermore, stippling is not present at all stages of lead poisoning. It is usually present during the acute stage or as held by some, during the stage of exacerbation, as in deleading. It is conspicuously absent in the chronic stage.

Tests for Lead Poisoning. *Stippled-Cell Count.*—The average physician resorts to a stippled-cell count for his laboratory investigation of a "lead" case. The finding of five or more stippled cells per

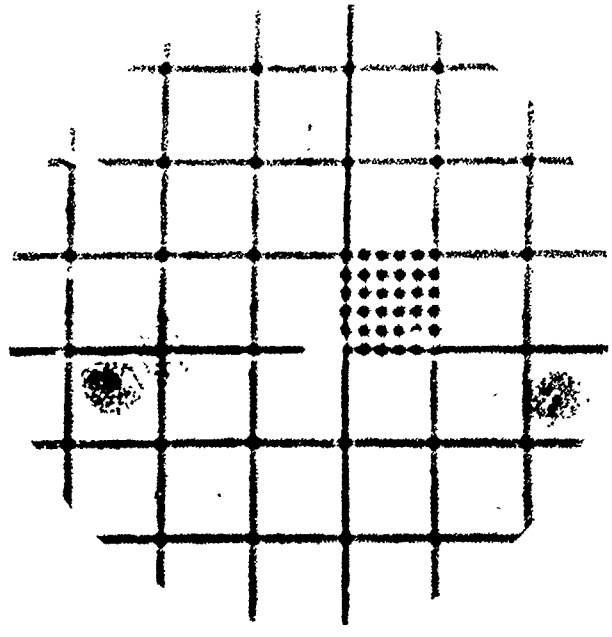


Fig. 39.—Laked portion of blood smear from the same slide as Fig. 38, showing four typical basophilic aggregations and faint outlines of non-basophilic containing erythrocytes. (Hyler and Bradley.)

field has frequently been accepted as an indication of lead poisoning (Figs. 38, 39). Plant physicians, however, will often permit men to continue to work with a count as high as 15. The ordinary stipple count doubtless is of value in plant routine where men are under constant observation and where frequent comparisons are obtained.

(*a*) *Staining Method for Stippled Cells.*—The usual staining methods for demonstrating basophilic granules are well known to all. Ruth A. McKinney and Saul Rosenzweig, of the North End Clinic in Detroit, suggest the following method and enumerate the advantages:

The three reagents required are kept for convenience in covered Coplin jars. The technic is as follows: Fix dried smear in acetone-free methyl alcohol for from three to five minutes and transfer directly to Wright's stain (staining time predetermined for each lot of stain used); wash in tap water and transfer to dilute ammonia water (2.5 cc. of stronger ammonia water in 1000 cc. of distilled water); dip up and down rapidly until blue color runs from slide; wash in tap water, dry and examine.

The finely stippled or coarsely dispersed "basophilic aggregations" in the red blood cells appear distinctly black against the gray or pink of the stained cell (Fig. 40). The white blood cells retain the usual nuclear stain.

The ratio of lead-affected cells to the normal red cells is determined by an adaptation of the Fonio platelet counting technic. A minute opening in a paper disk dropped into the ocular diaphragm gives a suitable counting field. The stipple cells and the normal red cells in each field are counted, but tabulated in separate columns, until 250 normal red cells have been counted. The number of stipple cells is then multiplied by 4, giving the ratio of such cells to 1,000 normal cells. The number of thousands of red blood cells per cubic millimeter multiplied by the number of stipple cells per thousand cells gives the approximate number of lead stipple cells per cubic millimeter of blood.

This staining method offers nothing fundamentally new; its chief advantage lies in the fact that a simplification and combination of two previous technics makes possible:

1. A rapid detection and enumeration of stipple cells in a spread which is also adequately stained for a differential count of the white blood cells
2. The utilization of reagents usually found in any physician's office or clinical laboratory
3. A technic which stains dried smears two months old as satisfactorily as fresh dried smears

McCord,[12] as early as 1924, questioned the value of the ordinary stipple-cell count.

Basophilic Aggregation Test.—McCord[13] and his co-workers assert that in the 1934-35 epidemic of lead poisoning in the automobile industry, 6900 basophilic aggregation examinations of the blood were made. In addition, during this period 1100 tests were made in other industries. This number includes approximately 500 control examinations made on workers who were not exposed to lead. Positive basophilic aggregation tests have served as an index for lead absorption prior to the appearance of clinical manifestations of lead poisoning. This test has proved to be of value in the diagnosis of *early cases* of lead poisoning.

Fig. 40.—A schematic drawing to show the presence of basophilic substance creating a "stippled" appearance.

(*a*) *Principles.*—The procedure is suited to application by any physician or laboratory carrying out any blood examinations. The basic principle in the basophilic aggregation test is the enumeration of red blood cells containing basophilic substance, in contrast to the customary procedure of qualitative or quantitative examination for stipple or polychromatophilic cells. The native state of basophilic material in unaltered red blood cells is not known, but in the process of

Fig. 41.—Typical pre-formed punctate stippling in non-hemolyzed erythrocyte (semi-diagrammatic). (McCord.)

laking and staining red cells this substance may be artificially aggregated into readily visible masses (Figs. 41, 42, and 43). In normal human adults, these aggregates rarely exceed 1 per cent of the total number of erythrocytes, but in persons exposed to lead the percentages ordinarily lie above this normal maximum, when considerable lead is being absorbed or when clinical lead poisoning is imminent. Finding more than from 1 to 1.5 per cent and especially more

Fig. 42.—Typical basophilic aggregation in erythrocyte, showing heavy deposit of basophilic material (semi-diagrammatic). (McCord.)

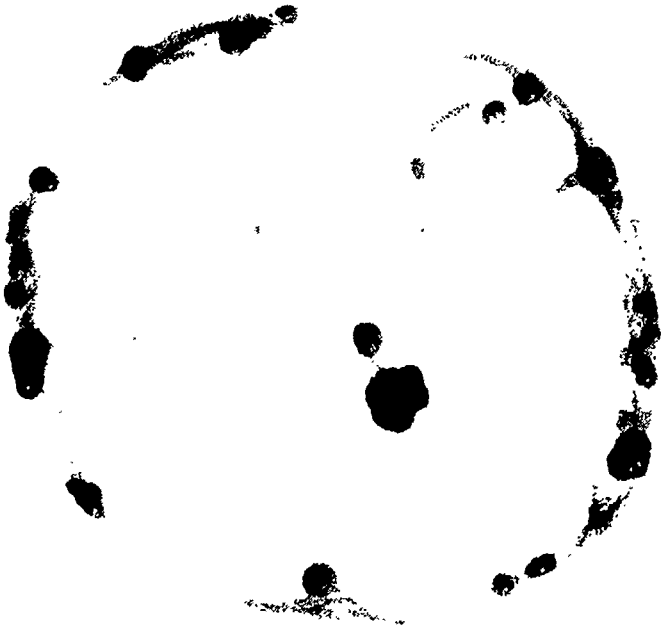


Fig. 43.—Typical basophilic aggregation in hemolyzed erythrocyte, showing scanty basophilic material and thus more difficult to recognize than in Fig. 42. (McCord.)

than 2 per cent in persons exposed to lead at once suggests lead absorption and the possibility of approaching lead poisoning, or the actuality of early lead poisoning. In chronic lead poisoning this test usually is not, but may be, positive. As lead poisoning progresses to extended chronicity, the reliability of the procedure diminishes. The test has been utilized in industries using lead to determine the number of exposed workers absorbing lead, as some proof of existing lead hazards; as a guide for the transfer of workers absorbing lead to lead-free departments; as a measure of the efficacy of preventive devices and practices; and as a means for the detection of malingerers. There are varied types of diseases leading to positive basophilic aggregation tests, but in groups of workers in lead industries, presumably normal except for the possible effects of lead exposures, the positive basophilic aggregation test stands in some relation to lead absorption and its subsequent action.

(*b*) *Technique.*—The preparation of the smears for the aggregation test is best described by Hyler and Bradley,[14] who adopted the best features of McCord's original technique:

> Only the best quality of microscopic slides should be employed, and preferably these should be used only once. Prior to use, slides should be thoroughly washed, followed by submersion in acid and alcohol. The blood smear should be such as to obtain an even distribution with such thinness as is represented by 150 to 200 cells to the microscopic field under an oil immersion objective. These smears, after air drying, may be fixed as later discussed, and stained almost immediately, but standing up to sixty hours appears to make little difference in the counts later obtained. In one instance, smears were made on fifty individuals in duplicate. One-half were fixed and stained in six hours, while the second half were so treated at the end of sixty hours. Reading of these slides indicated a variation in the basophilic aggregation counts of 0.1 per cent.
>
> The fixing of one-half of the slide on a longitudinal basis affords some opportunity for error in that vapors from the methyl alcohol used for fixation purposes of one-half the slide may, in the absence of precaution, tend to fix the remaining half to some degree. Three procedures have been utilized, the first two of which are far more practical. Jones devised a fixation method whereby a longitudinal half of the blood smear is covered with a strip of filter paper and is cautiously wetted with a minimum of methyl alcohol (acetone free). This is allowed to dry until the filter paper becomes loose. The remaining half of the slide is unfixed in order that laking may take place during the process of staining. In the second method, a shallow utensil, such as a petri dish, is filled with alcohol to such depth that one-third of the slide is submerged by dipping to the bottom of this utensil. After

instantaneous dipping, the slide is allowed to dry horizontally on muslin or gauze in such fashion as not to permit any drainage onto that portion of the slide for which fixation is unwanted. In either case, a narrow band of partially fixed cells will exist at the junction of the fixed and unfixed portion of the slide, but this offers no practical problem in the performance of this test. This transition zone is ordinarily less than the width of a microscopic field. For precise purposes, it is possible to score the underside of an annealed glass slide with a good diamond, to make a smear in the usual fashion, and thereafter, prior to fixation, to break the slide into two pieces for fixation purposes. After staining, these halves may be brought into apposition for microscopic examination.

For staining purposes, a variety of vital stains might be utilized provided the solutions used are such that permit of cell laking. In this work, two stains have been utilized with formulae modification from time to time. Jones has expressed a preference for the Sussmann-Weindel stain, about which he states:

All factors considered, the Sussmann-Weindel solution of toluidine blue, methylene blue, and borax furnished the most uniform results in examination of both fixed and non-fixed films. The formula is as follows:

Toluidine blue	0.5 gm.
Borax	0.05 gm.
Methylene blue solution (Loeffler's)	5 cc.
Distilled water	100 cc.

The borax is added to the water, which is heated to dissolve if necessary. The toluidine blue is added and allowed to stand for a few minutes; occasional stirring may hasten solution. Next is added the methylene blue. The solution is then filtered through a single No. 30 filter paper.

Our own preference is for a modified Manson methylene blue stain, prepared as follows:

Sodium borate C. P.	0.3 gm.
Methylene blue chloride	2.0 gm.
Distilled water, to	100.0 cc.

The sodium borate is added to distilled water which is brought to the boiling point. After cooling, the methylene blue chloride is added with vigorous stirring. Filtration may be desirable prior to the addition of methylene blue, and certainly is desirable after the methylene blue addition and thereafter at intervals. The life of the stain is longer if kept in a tightly-stoppered bottle. . . .

Actual staining with this modified Manson stain may be carried out in series of Coplin jars, the time being about ten minutes, but it is almost impossible to over- or under-stain. After staining, the slides should be rinsed in three washings of distilled water and air-dried.

Staining the unfixed portion of the slide is in effect a laking process which removes the hemoglobin, clumps the basophilic material, and brings this into greater visibility. The preliminary examination of the stained slides in making the basophilic aggregation count is done under high power to locate areas of even cell distribution. Final examination is made with the oil immersion objective without the use of a cover slip. Microscopic examination is first made in the laked portion of the smear. It is observed that the normal erythrocytes are very nearly faded from the picture. These cells appear as a faint shadow with an indistinct periphery. The white cells stain deeply and their various forms may be differentiated. The basophilic material in the red cells of this portion of the slide stands out sharply as coarse granules or as combined granules and network, or as reticulation.

The counting technique is facilitated by the use of a Whipple grid and hand counter. All cells in each field containing basophilic substance are counted. If any of these cells are divided by the outside margins of the grid, they are counted as belonging in that field. Those cells displaying basophilic aggregations in twenty consecutive fields are counted or in the case of an uneven smear, two rows of ten consecutive fields each are enumerated. The total number of basophilic cells is recorded.

Examination is next made in the fixed portion of the slide opposite and approximating the area that was examined in the laked portion. All of the red cells within the grid are counted, including those touched by the four margins of the grid.

Five fields are examined in the fixed area. If the examination of the laked portion was made of twenty consecutive fields, then all of the red cells should be counted in every fourth field for a total of five fields. But if the cells containing basophilic material were counted in two rows of ten fields each, then the cells should be counted in each alternate field for a total of five fields. The result should be expressed in percentage. Hence, if there were a total of 63 cells with basophilic aggregations found, this result would be: 63 divided by 4 equals 15.75, and again divided by 786, the total number of unlaked red cells counted in five fields, equals 2.0 per cent of the total erythrocytes as basophilic aggregation cells.

This technique is such that one sufficiently experienced technician may fix and stain 400 blood smears in one day, or may examine from 60 to 100 stained slides in a working day. In many cases, when reading slides made by the proper methods, three technicians have arrived at an agreement within 0.1 per cent of the basophilic aggregation count.

Preparations made approximately in the manner described in this section have been kept for a period of thirteen years without significant deterioration.

In arriving at the approximate number of stippled cells per million red blood cells, the following technique was utilized. A blood smear slightly thicker than the smear for a basophilic aggregation count, was made, usually 200 red blood cells to the field under oil immersion objective. This was then stained with Wright's dye in the usual way. Examination was made with the oil immersion objective of fifty fields or approximately 10,000 red blood cells. The total number of stippled cells found is multiplied by 100 which indicates the approximate number per million red blood cells.

At best, the examination for stippling is difficult, entailing much eye strain. Since much of the stippling is of very fine nature, it requires close scrutiny to avoid missing these cells. The result is that the average technician can examine only 25 to 30 slides per day. Examinations of large numbers of employees exposed to lead hazards in certain industries obviously would require considerably more time than for the basophilic aggregation test.

Possibly because of the work involved in the above described method it has not been generally adopted. From McCord's experience it would appear that, clinically, patients with early lead poisoning regularly exhibit this high basophilic aggregation content of the blood. Certainly it has a confirmatory value especially in routine plant examinations, where the procedure is all set up for a large number of examinations. For the isolated case with a medicolegal aspect, examination of the blood, smear for excessive stippling, excreta, and the whole blood, appears simpler.

Routine Blood Count.—There exists no distinct picture, but the following changes are noted with exceeding regularity.

1. A reduction in the number of red blood cells
2. Changes in the size and shape of the cells out of proportion to the reduction of the red-cell count or hemoglobin
3. A reduction of the hemoglobin usually greater than that of the red blood cells. The color index usually less than 1
4. A fairly frequent leukocytosis. The relationship of the large to the small lymphocyte has already been mentioned

Lead in the Urine. (a) *Twenty-Four-Hour Specimen.*—Using precautionary measures, a complete twenty-four-hour specimen of urine is collected in Pyrex containers and the entire volume submitted to a chemist. A modified dithizone titrimetric extraction is usually employed. Most authorities accept a reading as high as 0.10 mg. per liter

as being within a normal range. Kehoe estimates 0.04 as a mean normal. This test should be run in every case in which there is suspicion of lead poisoning. If the test is conducted accurately, amounts in excess of the normal serve as confirmatory evidence. Diseases of the kidney, dehydration, imbibition, edema, and errors in collection, etc., are factors which may influence the value of the test.

(*b*) *Short-Time or Instantaneous Specimen.*—In large plants or where a great number of men are to be constantly observed, it is difficult to obtain twenty-four-hour specimens without having them become contaminated or having inaccurate amounts collected. Realization of this difficulty with the workmen at the Westinghouse Electric and Manufacturing Company, at East Pittsburgh, led Barnes to investigate the possibility of correlation between short-time or instantaneous specimen and average twenty-four-hour excretions. It was felt that some correlation might be obtained if factors such as time of day, acidity, specific gravity, or total nitrogen were taken into account. After experimenting with exposed, as well as unexposed persons, Barnes[15] concluded that a determination of the lead excreta per hour, as found by analyzing a specimen representing two or three hours' excretion during the working-day, gives a fairly reliable measure of the average excretion for that day. It impresses the author that this procedure suggested by Barnes can serve the plant physician where all the factors can be controlled, but that for a single examination of a patient suspected of lead poisoning the twenty-four-hour specimen should be obtained.

The *instructions* that are to be adhered to in obtaining these spot or instantaneous specimens are as follows:

1. Have the patient empty his bladder. This first specimen is obtained for the purpose of starting at a definite time with an empty bladder and is not to be used in the lead analysis. The first specimen should not be placed in the special container. It may be used for the determination of sugar, albumin, specific gravity, etc., if desired. Record on a data sheet the time that the employee emptied his bladder. The date and the employer's name and occupation should also be recorded.

2. Instruct the employee to return to the medical department at least two hours later (preferably three or four hours later). He should not void his urine until he has returned to the medical department, where the specimen will be taken.

3. When the employee returns, mark the time on the data sheet and have him void directly into the special container. Special care should be exercised to be certain that no dust or dirt is dropped into the container. These containers have been specially cleaned at the

laboratories and should not be rinsed or cleaned in any way. Do not add any preservative. Do not remove any of this sample from the special container, and do not use any of it for other tests.

4. The results of the test are reported in micrograms of lead excreted per hour.

Lead in the Blood.—The normal amount of lead in the blood has been well established in recent years. The reliability depends upon adherence to a most rigid technique to avoid contamination.

(*a*) *Special Uses for Determination.*—According to Smith[10] and his associates, a differential diagnosis of lead poisoning can be accomplished by an accurate determination of lead in the serum, cells, and fibrin fraction of the blood when taken into consideration with the total amount of lead in the whole blood. It is possible by this procedure to make a differentiation between acute, chronic, and mild lead poisoning, as well as to differentiate between mild lead toxemia and such pathological conditions as gastro-enteritis, hypercalcemia, food poisoning, or colic from other causes. The therapeutic use of lead can be controlled by this procedure, since deleading treatment may be instigated before an acute lead crisis develops. Results of 0.001 mg. of lead or less in 10 mg. of serum, cells, and fibrin, or whole blood, may be in error and should be considered qualitatively rather than quantitatively. Such a consideration does not change the interpretations, since the important factor is the appearance and disappearance of pathological lead in the serum fraction of the blood.

(*b*) *Serum Fraction versus Whole-Blood Content.*—The essential difference in the manner in which the analysis of the blood of healthy persons differs from definite clinical cases of acute lead poisoning and periods of exacerbation in chronic lead poisoning lies in the appearance of lead in the serum fraction of the blood, since approximately 34 per cent of these patients show a whole-blood content of less than 0.01 mg. of lead in 10 gm. of blood, and 18 per cent have a whole-blood content bordering on the normal range of healthy persons. In thirty-four cases of latent and early acute phases of chronic plumbism there was a definite appearance of lead in the serum fraction of the blood in all the cases, although the lead content of the whole blood in 58 per cent was less than 0.01 mg. in 10 gm., and 44 per cent fell within the limits of the range established by healthy normal persons and patients with disorders other than plumbism. In inactive chronic plumbism the serum is normal, but there is usually a definite increase in the lead content of the whole blood over that of the control group.

(*c*) *Analysis in Differentiation.*—Smith and his co-workers state that a definite diagnosis may be established in borderline cases by placing the patient on acid therapy as practiced in deleading, when an

increase in the serum fraction and generally in the whole blood will occur. The clinical and physiological course of lead intoxication through its quiescent periods, as well as those of acute and subacute exacerbation, may be followed by means of the analysis of the serum fraction and the whole blood for lead, and a prognosis established. Since it required 10 gm. of blood to make a determination for lead by the diphenylthiocarbazone method, the clinical procedure followed in cases complicated by the presence of malignant growths or blood dyscrasia is first to make an analysis of the lead content of the serum, cells, and fibrin fraction, and whole blood, then follow the trend of the intoxication by means of daily shiftograms and hemograms, and make additional blood analyses when they are hemographically or clinically indicated. However, in uncomplicated cases an analysis is made every second or third day. By such a procedure it is found possible to differentiate between symptoms caused by mild lead poisoning and those which result from pathological conditions. A range of lead values for the healthy normal person is found to be nil in 10 gm. of blood serum, and from 0.002 to 0.011 mg. in 10 gm. of whole blood. This range is independent of sex, age, climatic changes, daily fatigue, violent exercise, meals, menstruation, and ovulation.

Lead Levels. (*a*) *Of Blood and Urine.*—Concerning the evaluation of lead in blood, Kehoe has a different viewpoint. The following quotation is from the letter to the author previously mentioned:

In a study of a rather large number of normal persons, as well as a considerable number of persons with varying degrees of lead absorption and lead intoxication, we have confirmed the observations of Willoughby and his associates. I believe that the lead of the blood is carried almost entirely by the cellular elements. Willoughby examined serum and clot and found most of the lead in the clot. We have separated the plasma and cells by the use of sodium citrate and also heparin, and have found that the cells under these conditions contain practically all of the lead. Where higher concentrations of lead are present in the blood, there is some tendency for the plasma to contain larger proportions of the lead than in the normal case. However, this proportion is never large and in our experience has not exceeded 10 to 15 per cent. It is usually under 10. It appears that lead so transported in the body is not bound in the sense that it cannot be excreted, for in general there is approximately a 10 to 1 relationship in the concentration of the lead in the blood and urine. That is to say, the concentration of the lead in the blood per 100 grams is approximately the same as the concentration in the urine per liter. This does not hold absolutely, there being variations both up and down. Generally speaking, however, where lead absorption to abnormal degrees

has taken place, the urine changes more proportionately than does the blood lead. It seems probable that the equilibrium relationships between the cells and the plasma, with reference to lead, are as unstable as those relationships in the case of certain other ions. The material of the erythrocytes is especially liable with respect to its chemical combinations. So far as I know, no one has studied arterial blood with reference to its lead distribution. We expect to do this, for we suspect that there may be considerable difference in the arterial as compared to the venous blood. In any case, lead is normally carried mainly in the erythrocytes, but is excreted by way of the kidneys.

From the foregoing paragraphs it can be seen that there is as yet no unanimity of opinion regarding the relationship of lead found in the serum to the total blood-lead level, nor upon the relationship of the urinary lead findings with blood-lead determinations. Kehoe believes the urinary-lead findings are of diagnostic value, but less reliance is placed upon this test by Scott and McMillan, Litzner and Weyrauch, or Smith, Rathmell, and Marcil. Tompsett and Anderson[16] recently reported a series of twenty-nine cases in which the lead content of the blood and the excreta was examined. In the cases of plumbism they concluded that the total daily excretion of lead in the urine and feces did not run parallel to the level of the lead in the blood.

(*b*) *After Removal from Exposure.*—Concerning the question of long-continued high blood levels, after the patient has been removed from his exposure, Kehoe believes this is unlikely. This has also been the author's experience. Yet Chaney, of the Los Angeles County Hospital, informs me that continued high blood levels have been in accord with his experience. Also, Blumberg, Scott, and McNair found blood levels very slow in dropping to normal, from six to twelve months being required in some cases. They also state that clinical improvement occurs long before any appreciable drop in the blood level takes place. If this is true, then the commonly accepted viewpoint that symptoms are due to circulating lead will have to be revised. Oddly enough, most of these controversial points were brought out in a single case of ours, which is to be discussed in detail (Case V).

While it may appear that the above discussion is too controversial for the purposes of this book, it has been indulged in to emphasize the fact that the diagnosis of lead poisoning is not so simple a procedure as the average medical textbook would lead one to believe. Conclusions can only be drawn after careful consideration of the history, investigation of the exposure, evaluation of the symptoms, and laboratory study. In the average case *dependence upon the history, symptoms, and a blood smear for stippling, is to be deprecated.* Accuracy

can better be obtained by the additional total daily urine test, or the determination of the lead in blood, or both. In this clinic we examine the twenty-four-hour specimen of urine routinely and frequently check it with an examination of whole blood for lead.

Collecting Blood for Analysis.—Blood samples must be taken in a space which is free of dust. Despite this precaution, it is necessary to use the most meticulous care against the contamination of samples with surface or atmospheric dusts.

The patient must remove any dust-laden clothing before entering the room in which the sample is to be taken, and must wash hands and face or any other parts of the body that may be dusty.

The cork should be removed from the tube in which the blood is to be drawn only during the withdrawal of the blood, and should not be permitted to come in contact with the tabletop or any other potentially unclean surface, but should be held by its topmost part or laid down within its filter-paper wrapping, and replaced as soon as the blood has been obtained. The filter-paper cover should be replaced and secured around the neck of the tube by means of a rubber band.

The sterile lead-free needle provided in a second tube should be used to withdraw approximately 10 cc. of blood directly into the blood tube. Under no circumstances may any anticoagulant or other substance be added or any intermediate container be used, nor may the needle be sterilized by boiling in water. If the latter should be rendered non-sterile accidentally, it should be flamed gently before use. After use it may be rinsed out with cold water and returned to the tube.

The blood sample should be labeled with the name of the patient and the date and hour at which the sample was taken.

Two 10 cc. samples are taken at the same time so that parallel analyses can be made by two different methods.

Sampling Lead Dust in Air.—There are times when a claim can be the better substantiated or denied by sampling the air at the place of employment. A safe atmosphere is considered, at the present time, to be 1.5 mg. per 10 cm. of air. To be of any value in medicolegal controversy, faithful adherence to all the details is necessary in reduplicating the working conditions.

x-Ray Examination.—*x*-Rays to determine the effect of lead poisoning on bones are of no value in adult life. They are of value in children only before the epiphyses unite.

Summary of Diagnosis.—There are more cases accepted as lead poisoning than are justifiable because a distinction between *lead absorption* and *lead intoxication* is not made and because the laboratory criteria for diagnosis are not employed. One should not depend upon

the "cardinal symptoms." It has been shown that lead is normally present in urine and feces. What the profession needs to know today is the difference between normal and abnormal amounts. An equitable opinion in Workmen's Compensation must be based upon the history, the clinical findings, and the laboratory results. For the sake of emphasis and reference they are reiterated as follows:

Urine (24-hour specimen): Normal, 0.01 to .1 mg. of lead per liter
Basophilic aggregation test: Normal, 1 to 1.5 per cent
Blood: The range of lead value in the normal person is as follows:
(*a*) Nil for 10 gm. of blood serum
(*b*) .002 – .011 mg. per 10 gm. of cells and fibrin fraction
(*c*) .001 – .005 mg. per 10 gm. of whole blood

MEDICOLEGAL ASPECTS

Alleged Complications.—A neglected discussion in literature, and one which certainly deserves careful consideration in a book of this type, concerns the complications frequently alleged to follow lead poisoning. Chief among these are: arteriosclerosis, nephritis, hypertension, ulcers of the stomach, neuritis, neuroretinitis, and the effect upon germ cells and fetal life. With the exception of the last-named effect, proof has never been established that any of the other alleged complications can result from lead intoxication or lead assault. Recent opinion is that they are coincidental findings. Why then do we find frequent reference in literature to some of these questionable complications? It is not difficult to explain since it is a well-recognized fact that textbooks and medical articles frequently incorporate the opinions of predecessors, assuming their scientific accuracy even though pathological evidence may be lacking.

Arteriosclerosis.—Years ago in the industrial centers of Europe, it was noted that the "old stagers" when they died and were autopsied revealed arteriosclerosis. Writers of that time, in describing the effects of lead, included in their writings the arterial findings. Thus, there drifted into the literature the observation that "lead caused arteriosclerosis." These observations were made before the study of arterial disease was as thorough and scientific as it is today. They were made in the days when a death certificate of "acute indigestion" was acceptable.

Mayers examined 381 cases of lead workers for the New York State Department of Labor. She found that while arteriosclerosis occurred earlier in all types of industrial workers than it did in office workers and professional men, there was no evidence that lead played any part. The Metropolitan Life Insurance Company's study of ar-

teriosclerosis concluded that while this condition is more common in industrial than in non-industrial workers, lead itself was not shown to be an exciting cause. McNally,[17] after a study of a large group, concluded, "It can no longer be contended in the American practice of manufacturing of lead products that lead causes high blood pressure." According to Aub, there is a lack of correlation between the clinical picture and the pathological findings. Patients who present many symptoms suggestive of pathological changes usually show little of interest at autopsy. Regarding compensation, it is obvious that arteriosclerosis or hypertension in a worker over fifty years of age should not be attributed to lead any more than should the appearance of gray hair or baldness.

Sterility and Abortion.—The observations of numerous investigators leave little doubt that lead in excessive amounts affects the germ cells of both sexes, and as a result, sterility and abortion are fairly common among lead workers. The transmission of lead and arsenic from the mother to the fetus was shown recently in the experimental work of Calvery.[18]

Gastric Ulcers, Nephritis, and Cirrhosis of Liver.—Claims have been made that lead produces these conditions. Statistics are not available to prove or disprove such statements. A review of the literature indicates that such incidence is no higher in laborers subject to plumbism than in any other class of laborer. Such contentions undoubtedly got into print in the day when alcoholism was prevalent among working-men.

Recent studies show that there is no adequate proof for, and ample evidence against, the theory that lead is an etiologic agent in cases of *multiple sclerosis.*

Neuroses.—The neuroses which follow lead poisoning are frequent and accompany the weekly compensation stipend in a manner similar to the traumatic neurosis. They rarely appear in younger patients or those treated early and thoroughly. It behooves the industrial physician to weigh carefully the probability of other causes before assigning to lead the provocative etiology of the so-called complication.

Temporary Disability.—Temporary disability ceases with the disappearance of the symptoms. The period may be from several weeks to several months. Forty-two days was the average temporary disability of sixty-two patients treated at the clinic during 1937 and 1938.

Permanent Disability.—Permanent disability is rare. A review of our records for the past five years reveals not a single instance of actual permanent disability. When severe cases of encephalopathy result, permanent disability (100 per cent) should be allowed. Isolated muscular weakness or nerve paralysis must be estimated for rating

upon the same basis of functional loss as is done in traumatic lesions. Since elimination of lead varies in people, and since persons with functional weaknesses tend to recover, there should be no rating made in these cases for at least two or three years. In 1931, Kessler suggested that after a period of two or three years, compensability of the claim for permanent disability might better be established by a provocative test. Following this, if the blood and excreta are positive for lead, the possibility of reduction of longevity should be considered. In view of recent investigations one should question whether this procedure would be satisfactory.

Illustrative Cases.—Those patients who have been referred to this clinic having been previously diagnosed as having lead poisoning fall into the following groups:

1. The majority have been diagnosed upon the patient's statement that he has lead poisoning because he works "where they handle lead." They have been subjected to no laboratory investigation.
2. A small group with a history of lead exposure has been diagnosed upon the finding of stippled cells in the blood smear.
3. Others upon the presence of a lead line of the gums. Invariably this is not actually present in these patients.
4. A very small number who have been subjected to blood smears, and twenty-four-hour specimens of urine for lead.
5. Rarely has any case been accompanied by a study of the lead in the air at the place of employment.
6. One patient had a whole blood study for lead.

Case I.—A man, aged fifty-eight, had been a Linotype operator for twenty-two years. In the summer of 1935 he developed vague pains in the area of the "stomach." These appeared usually at night and were conspicuously absent during the day. He consulted several physicians, having gastro-intestinal series, gastric analyses, and other tests performed. He continued working but constantly sought medical advice. In 1937, one physician inquiring about his occupation, had a twenty-four-hour specimen of urine run which revealed 0.08 mg. of lead per liter. The patient was told that he had lead poisoning. He stopped working and filed a suit before the Industrial Accident Commission of California for compensation. Following the hearing, his claim was allowed. The award was for total, indefinite, temporary disability. In the spring of 1938, he developed attacks which signified coronary involvement. Upon being informed of this condition, the patient filed a suit for permanent disability due to coronary sclerosis resulting from lead poisoning. The commission again allowed his claim. Two months after this award the patient was sent to the writer for an opinion.

History.—The patient's father died at the age of fifty-eight years of heart disease. The patient denied serious illness or any venereal disease.

Physical Examination.—Complete details of the examination will not be related. The heart rate was 80. No murmurs or apparent enlargement. Blood pressure 158/90. The electrocardiogram revealed no evidence of coronary involvement but did show low amplitude of the QRS interval with notching. There was moderate peripheral arteriosclerosis. The urine showed a good trace of albumin with numerous granular casts. Blood chemistry was normal. The lead content of the twenty-four-hour specimen was normal. Blood lead was not determined. The blood count was normal, and the basophilic aggregation tests were negative.

It was not expected that abnormal lead would be found in any of these tests, but they were run because of the medicolegal dispute.

The conditions of employment were studied. The claimant admitted that they were identical at the time they were studied and tested as they had been during the years he had been employed. Maintaining a faithfulness to detail in order to reduplicate the exact working conditions under which this man served, the Smith-Emery Testing Laboratories obtained a percentage of lead in air less than the accepted standard for safety.

During the trial, it was shown that the patient had orthopnea, dyspnea, and substernal pain on effort. Several physicians testified that these were signs of heart disease common to men in his age bracket regardless of occupation. It was brought out that no one had been able to produce evidence that this man had ever had excessive amounts of lead in his system. It was shown that the diagnosis of lead poisoning had been made upon one finding, coupled with an assumption; namely, the finding of 0.08 mg. per liter of lead in the blood, which is within normal limits, and that he had been employed in a trade at one time considered to have a marked lead hazard.

This is an example of the prevailing misconception of what constitutes lead poisoning. It is an example of attributing to employment a disability not substantiated by findings, instead of attributing the disability to a disease syndrome common to the age of the patient. There existed no evidence that the man ever had lead poisoning, but there was preponderant evidence that his condition found multiple duplication in all walks of life. The case illustrates the urgency for a better informed profession.

The former decisions were reversed in favor of the defendant.

Case II.—A woman, aged thirty-two, presented herself to the clinic complaining of lassitude, weakness, aching all over her body, dry throat, and marked constipation. Four days previously she had

become aware of abdominal cramps, which were increasing in severity.

History.—Occupational history revealed that for six month each year she worked for a tinsel-manufacturing company which made, among other things, Christmas ornaments. Four weeks prior to the onset of her illness, she had been transferred to a small, partitioned room where she dipped ornaments in a hot solution of lead. Ventilation was inadequate, and no respirator was provided. Past medical history revealed that the patient had had an appendectomy in 1920, and a gallbladder drainage in 1926.

Physical Examination.—The gums showed small, spotty deposits along the margins adjacent to the teeth. Blood pressure, 128/70; pulse, 82; temperature, normal. Abdomen: Generalized soreness but no rigidity. No masses or organs palpated. Scars of former operations. Muscles of the extremities were tender to the touch. Neurological examination was negative.

Laboratory Examination.—This revealed red blood cells, 3,100,000; hemoglobin, 68 per cent; white blood cells, 9800. Basophilic aggregation test, 1.8 per cent. Routine urine, negative; twenty-four-hour specimen of urine for lead, 0.52 mg. per liter.

No blood lead was determined. This history was definite, the urine showed excretion above the normal, and the symptoms were sufficient. The patient did not recover for three months, experiencing an acute reversal during the course of deleading.

The case was compensable. Temporary disability of thirteen weeks awarded. No permanent disability.

Case III.—A tire and battery repairman, aged forty-two, consulted his physician regarding abdominal cramps, lassitude, headache, and loss of weight. The workman contended that his illness was due to the acid fumes from the batteries that were constantly being recharged or repaired. The physician found a secondary anemia. The patient was instructed to refrain from work and was given calcium tablets. The insurance carrier was informed that the man had lead poisoning. Seven weeks after stopping work, a gastric hemorrhage occurred, and an operation ensued, which disclosed a perforated gastric ulcer.

No attempt was made in this case to prove the presence of any disease. No lead was looked for in the excreta or blood. A differential diagnosis for pains in the stomach was not made. Seven weeks of absence from the alleged hazard, along with calcium therapy had not improved the condition. The only effect the fumes from these batteries might have had would have been a mild irritation to the upper respiratory tract from the sulfuric acid fumes.

In the department where batteries are charged in the largest storage-battery manufacturing plants, lead poisoning is unknown. This

patient worked in a large, well-ventilated room where, at no time, were there more than four or five batteries being recharged. The point in this illustration is that the evidence for lead was never investigated.

Case IV.—Bartleman and Dukes report the case of a young actress who developed the classic symptoms of lead poisoning. Blood examination revealed anemia and stippled red cells. The diagnosis was confirmed by the finding of large quantities of lead in the urine and feces. The source of the lead poisoning was traced to the use of a greasepaint containing approximately 40 per cent of lead.

There is a great variability in susceptibility to lead poisoning. The greasepaint used by the patient was the same brand as that used by several other members of the cast, but so far as was known, no other case of lead poisoning occurred. There was, however, a good deal of ill health and also some complaints of tiredness and headaches among those of the cast who had used this greasepaint previously.

This case is cited because it is unusual. It is not an example of industrial exposure in the true sense of the word. The possibility of constantly cracked, parched, dry, broken skin in those using cosmetics daily is apparent, although the authors do not record that. They indicate that they ruled out any other cause. No question of compensation is introduced by the investigators. If the patient bought and furnished her own cream, the liability should be her own, but if the cream was furnished by the owner, then the liability is his, and compensation would be in order.

Case V.—(Because the essential interest in this case is to be found in the laboratory findings, the physical examination and other non-essential factors will be omitted.)

A twenty-one-year-old foundry worker presented himself to the author on September 13, 1939, complaining of severe abdominal cramps, together with nausea and vomiting, which he believed were due to exposure to lead. He had worked for the Berg Metal Company for three months, but investigation showed he had been exposed to lead for only three weeks prior to the onset of symptoms. One week before reporting to this clinic he had been seen by his neighborhood physician, who had instituted twice-daily injections of calcium, and calcium gluconate was given orally. The clinical picture was typical for lead intoxication. The patient was immediately hospitalized and a search for lead begun. The blood count revealed a secondary anemia of moderate degree and the basophilic aggregation test showed 1.8 per cent of basophiles. The patient had pyorrhea, and the lead line was questionable. The twenty-four-hour specimen for lead in urine showed 0.01 mg. of lead per liter.

In view of the normal amount of lead in urine, the case incited further search. A hygienist was dispatched to the plant to investigate the conditions of exposure and reported them to be minimal. In spite of calcium therapy and high calcium diet, the patient obtained no relief from his symptoms. Ten days after he was seen by us, a blood test for lead was reported as showing 0.23 mg. per 100 cc. of blood and 0.12 mg. per 100 cc. of serum. The patient sought his family physician who continued calcium therapy and on November 20, 1939—over two months following our first investigation—he showed in the twenty-four-hour specimen of urine 0.03 mg. per liter, but his blood showed 0.2 mg. per 100 cc. of blood, and 0.1 mg. per 100 cc. of serum. At this point it should be admitted that faulty clinical procedure was followed, in that the laboratory investigating the urine was not equipped to do the spectrographic method of blood examination, and therefore two different laboratories were employed. Thus, after two months of intense accepted therapy and the absence from exposure, the lead content in the blood revealed practically no change. Because of the many peculiarities, the facts in the case were submitted to Dr. Robert A. Kehoe of the University of Cincinnati, and to Dr. V. A. Gant in Chicago, for their opinion. It is to be appreciated that they are commenting upon a case they did not see and of one over which they had no laboratory control. Dr. Kehoe wrote to me in part as follows:

In a period of 15 years' experience, we have never seen an adult with lead intoxication without a definitely elevated urinary lead excretion. I know of no renal condition which will interfere with the elimination and, in fact, it is our experience that when water can be excreted, lead is also adequately excreted by way of the kidneys. Relative to the lead content of the blood in which 0.23 mg. per 100 cc. of blood and 0.12 mg. per 100 cc. of serum were supposedly found, I am disposed to believe that these results are grossly in error. First, the result of 0.23 mg. per 100 cc. of blood in an adult is entirely incompatible with the urinary concentration of 0.01 mg. per liter. Second, the distribution of lead between cells in serum is not of a type indicated by these analyses. I have never found less than 85 per cent of the total lead content of the blood in the cells. The plasma, or serum, in our experience has never contained more than five to ten per cent of the lead content. This distribution as reported by you is so unusual, therefore, as to be almost prima facie evidence against its own accuracy.

It is also indicated in your report that on November 20, the blood still contained 0.2 mg. of lead per 100 cc. with 0.1 mg. or 50 per cent in the serum. My same criticism holds here. It seems quite obvious to me that if one of these results is correct the other is incorrect. I repeat that we have studied blood containing lead up to almost twice that which you have given here and have never succeeded in finding

anything like 0.1 mg. of lead per 100 cc. of blood plasma or serum. There is the additional reason for skepticism here in that more than two months elapsed since the cessation of exposure, yet the blood lead had not materially diminished. This is against all experience. Blood leads do not remain at such concentrations, in our experience, for more than a short time unless the exposure and absorption have been so massive as to involve very large quantities of lead in the tissues of the body.

Dr. Gant in his reply stated:

With reference to your letter of December 11, concerning the suspected case of lead poisoning, I must say that the findings are not in agreement with any case which has been called to my attention. In every instance in the past 11 years it has been my experience always to find an abnormal amount of lead in the urine when an abnormal quantity was found in the blood. As a general rule when it is found that the blood contains about 0.2 mg. per 100 cc. the concentration in the urine will be about one-tenth of that, or 0.2 mg. per 1000 cc.

Following the urinary findings and the report of the hygienist who investigated the hazard, the writer was at first in doubt as to the cause of the boy's complaints. But after the report of the lead in blood, he was recommended for compensation.

Certain discrepancies existed which cannot be explained. It is possible that the hygienist who investigated the circumstances of employment was at fault and the patient received a massive exposure which was not recognized. It was established that his exposure had been short and that he wore a respirator. Furthermore, the lead in the urine was determined by a reputable and highly efficient laboratory upon which great reliance has always been placed. Both urine specimens were collected under strict adherence to technique. The lead in blood was examined by Dr. Chaney, of the Los Angeles County Hospital, a man experienced and qualified in this method. Just where the error occurred will never be known. But it is well that it did, for the case has served to bring out the various phases of the controversy and has permitted a discussion of the relationship of lead in blood to that of lead in urine, the relationship of lead in red blood cells to that of lead in serum, and the question of how long lead in blood may persist. The patient was granted temporary disability for three months, and no permanent disability occurred.

Case VI.—A twenty-eight-year-old painter was taken to the County Hospital by a neighbor because he had been acting queerly. He complained of abdominal pains, vomiting, headache, and dizziness. Upon alleging that he thought his illness was due to lead poisoning

from the paint he worked with, he was considered a "compensation case" and refused admission. He was then admitted to a small sanitarium. Following a cursory examination, a diagnosis of lead poisoning was made. He remained in this institution for five days.

Following discharge, he was employed by a new contractor. He worked two weeks, when one day he was observed acting very queerly while painting the woodwork of a room. The foreman discharged him. The patient started home but did not arrive for two days and could not account for the interval of time. At this time he was brought to our clinic. He appeared lucid and claimed constipation, abdominal cramps, and weakness of his wrists. He told the resident that he had lead poisoning and that no one could fool him, for he knew the symptoms. Following a complete study in which there was no evidence of abnormal lead absorption as evidenced by the normal blood and urine, the patient was discharged.

One week later he appeared before the Industrial Accident Commission where he filed a claim for lead poisoning. At the time of trial he submitted a medical report from a physician which stated that this man had "clinical evidence of lead poisoning." No laboratory reports accompanied this diagnosis.

It was brought out at the trial that investigation of this patient's claim revealed the fact that he had been committed to an institution for dementia praecox three years previously. He had since married but had deserted his wife. He had not worked as a painter for more than two weeks at a time and then only about six weeks within the past sixteen months. No abnormal amount of lead had been found in any of the tests run by us, and the physician who contended that the patient had lead poisoning had never investigated it from the laboratory standpoint.

The patient was denied compensation by the commission. Obviously the case presented no basis for claim. It exemplifies the type of case that needlessly crowds the calendar of hearings before the commission.

TREATMENT

The treatment of lead intoxication is at this time not clearly defined. A short time ago a definite regimen could have been outlined for use, but recent experimental work has thrown considerable doubt on the value of the older methods.

Deleading by High Calcium Diet.—Following the work of Aub, Fairhall, Minot, and Reznikoff,[1] which indicated that lead could be either stored or excreted by distorting the calcium metabolism, therapy was directed along these lines. The conclusions drawn from their work were that a negative calcium balance increases the rate

of lead excretion, while a positive calcium balance favors the storage of lead. To this end, then, the treatment for the acute episode in the past has consisted of the following:

1. A high calcium diet, which includes much milk, green vegetables, and potatoes
2. The administration of calcium products and vitamin D orally, *e.g.*, calcium lactate, 1 drachm (4 gm.), once every two hours; or calcium gluconate, 7½ grains (0.5 gm.), in milk at three-hour intervals, so that the patient obtains five or six doses daily
3. Calcium gluconate, 10 cc. of 10 per cent solution, intravenously daily for five days
4. Magnesium sulfate, ½ ounce every morning, to sweep out the relaxed bowel
5. As substitutes for the intravenous calcium gluconate in the relieving of abdominal pain, hypodermic injections of morphine sulfate, ¼ grain (0.015 gm.), and atropine sulfate, 1/100 grain (0.0006 gm.); nitroglycerin, from one to two 1/100 grain (0.0006 gm.) tablets dissolved under the tongue; or the inhalation of the contents of a 5-minim (0.32 cc.) amyl nitrite pearl

Disadvantages of Method.—Recently much doubt has been thrown on this method of treatment by the work of Lederer and Bing.[19] Their work indicated that the deposition of lead in the bones of growing animals is retarded by increasing the calcium content of the diet and that the phosphorus content of the diet had no significant effect on the amount of lead deposited in the bones. This would indicate, then, that a high calcium diet would be the optimum diet for deleading, rather than for the deposition of lead in the bones. The use of the high calcium diet, then, according to this view might actually be dangerous by mobilizing lead for possible storage in the central nervous system in the acutely ill patient. Taeger[20] arrives at such a conclusion and advises against large doses as well as against the intravenous administration of calcium. Shelling and Hopper[21] believed the use of calcium did not induce superior calcification, but rather had the opposite effect, especially when the phosphorus intake was inadequate. Kehoe and Thamann[22] were of the opinion that the use of agents to promote the quick release of lead from the tissues was hazardous. Shelling[23] demonstrated experimentally that the addition of calcium carbonate to an optimal stock diet containing lead carbonate fed to rats resulted in an increased toxicity. These rats evidenced toxicity and died sooner than the rats fed on the same stock diet to which sodium phosphate had been added. Gray and Green-

field[6] reported a small series of cases where prolonged administration of high calcium diet therapy was responsible for permanent, irreparable damage to the nervous system. They[24] concluded that a high calcium regimen was the best means of deleading. In view of this evidence, the advisability of the use of a high calcium diet alone is open to question. The frequently dramatic relief of abdominal pain obtained from the intravenous administration of calcium may represent, then, only a general depressant action of calcium on smooth muscle rather than a specific reaction as regards the lead in the bloodstream.

With High Phosphorus and High Vitamin.—Gray and Greenfield[24] found the use of an optimal calcium diet, with a high phosphorus and high vitamin diet (D particularly), the best treatment in these cases. With this regimen an essential element was the administration of sodium phosphate, two drachms three times daily. Shelling[23] indicated that in order to deposit lead in the skeleton as an insoluble lead phosphate, a certain amount of phosphate was required, and thus when phosphate was inadequate, free lead might remain in circulation. He stated that the addition of sodium phosphate "provides sufficient phosphate for the deposition of both calcium and lead phosphate in the skeleton, for the excretion of lead as the relatively non-toxic lead phosphate, and also for the formation of colloidal lead phosphate in the blood." As indicated, the work of Lederer and Bing directly opposes this statement and seems to indicate that the addition of phosphate had no significant effect. The work of Lederer and Bing further indicated that the beneficial effect of calcium in the diet in retarding the retention of lead by the body was due to reactions which occurred in the intestinal tract and that calcium carbonate orally was the most valuable preparation.

Deleading by Low Calcium Diet. *With High Phosphorus.*—In the past a regimen of a low calcium, high phosphorus diet, phosphoric acid, and magnesium sulfate had been used successfully by Gray[25] in deleading treatment. He used the diet in Table 12 with claims of success.

He also elaborated a similar diet with a calcium to phosphorus ratio of 1:4. To these were added 20 minims of viosterol (250 D) daily, since vitamin D was deficient in the diet. Previously he had used a low calcium diet consisting of meat, liver, potato, rice, tomatoes (cooked without milk), canned corn, bananas, apples (peeled), tea, coffee (without milk), butterfat, bread (prepared without milk, such as salt-free nephritic bread or sodium bicarbonate biscuits or crackers), sugar, salt, and pepper. Phosphoric acid in amounts of from 10 cc. every two hours for six doses daily to 10 cc. once every hour for

ten doses daily was used. In addition, magnesium sulfate, ½ ounce (15 gm.), each morning was given. With this regimen, increase in urinary lead was at first seen, and this was followed by a decrease as the patient was presumably deleaded.

TABLE 12—LOW CALCIUM-HIGH PHOSPHORUS DIET
(Ratio 1 : 3)

Food	*Weight (Gm.)*	*Food*	*Weight (Gm.)*
Applesauce	100	Lamb chops	160
Grapes	103	Liver	90
Banana	100	Bacon	15
Pineapple juice	540	Baked potato	100
Shredded wheat (1)	27	Tomatoes	100
Whole-wheat bread	120	Green peas	100
Macaroni	30	Mushrooms	60
Egg-yolks (4)	80	Celery	30
Butter	30	Almonds	15
Cream, 40 per cent	70	Oranges	200
Lettuce	30		

Total: Protein, 101.78 gm.; calcium, 0.601 gm.; phosphorus, 1.889 gm.; Calories, 2,969.88.

With Other Substances.—Other methods suggested for deleading have included the low calcium diet cited above; ammonium chloride, 15 grains (1 gm.) every hour for ten doses daily; and magnesium sulfate, ½ ounce (15 gm.), every morning. Belknap[26] used fifteen drops of potassium iodide twice daily or seven drops twice daily of sodium iodide in saturated solution. Sodium bicarbonate, from 5 to 8 drachms (20 to 30 gm.), daily, divided into five or six portions has also been used, as has parathyroid extract.

Advisability of Deleading.—As indicated above, there is disagreement concerning the possibility of deposition of lead in bones by changes in diet or by medication. In addition, the advisability, as well as the possibility and the methods of deleading, is distinctly open to question. Most authorities feel that it should not be attempted before from three to four weeks have elapsed after the acute episode. Most feel that the deleading should be carried out only in the hospital and with adequate laboratory checks, although Belknap, cited above, deleads patients in mild cases while they are ambulatory but under close supervision. He requires a blood level of 80 per cent hemoglobin, 4,000,000 or more red blood cells, stippled cells not more than from 1000 to 5000 per million red blood cells for two to three weeks, and a twenty-four-hour urine lead 0.15 mg. or lower before undertaking the deleading. He also deleads no oftener than once in four weeks, and preferably not oftener than once in six or eight weeks.

Benefits versus Drawbacks of Deleading.—Those who oppose deleading feel that the danger of producing lead encephalopathy is too

great, and that the patient cannot be successfully "deleaded" at any rate. Those who favor it urge that the stored lead represents a constant potential hazard, since acute episodes, such as infections with acidosis or other periods of metabolic stress, may cause deleading at an undesirable time. Aub[27] feels that thorough deleading approximately halves the period of disability for lead palsies.

Ascorbic Acid Therapy.—Holmes, Campbell, and Amberg[28] found 100 mg. of ascorbic acid daily of much benefit in lead-intoxication cases, and also observed that the ingestion of alcohol had an unfavorable influence and in several instances nullified the benefits of treatment. Others have had no success with this form of therapy.

Treatment in Lead Palsy.—In this condition, stretching of fibers of paretic muscles should be prevented. In those cases presenting a wrist-drop, a cocked-up splint, to include the fingers and worn night and day, is used until function returns. Hot-water baths (110° F.), electric stimulation by interrupted galvanic current, and gentle massage, preferably by whirlpool, once every day or two are used. Strenuous massage and violent exercise are avoided. Reeducation exercises are of value.

Treatment in Lead Encephalopathy.—Encephalopathy is rare and has not been seen in our clinic in the past five years. When it does occur, lumbar puncture and sedation may be required. Magnesium sulfate, 2 cc. of a 25 per cent solution given intramuscularly from every four to six hours, and barbiturates are used. For convulsions barbiturates, *e.g.*, sodium amytal, from 3¾ to 7½ grains (0.23 to 0.5 gm.), are given intravenously, and avertin is used rectally. Suboccipital or subtemporal decompression in lead encephalopathy has been used successfully.[29]

Therapy without Deleading.—Many feel that removal from the hazard with symptomatic treatment for pain, weakness, lead palsy, and encephalopathy is all that is needed, with no particular attempt to store or remove the lead. We have usually found this procedure successful in our own clinic.

Prophylaxis.—The best method to avoid lead poisoning is, of course, to take measures to prevent it. To this end, Gant[30] suggests the following rules and mechanical features:

1. Hoods with adequate exhaust ventilation to be at any point where dusts or fumes arise
2. All mixing and shaking to be done in enclosed machines
3. All powdered compounds to be transferred by means of adequate vacuum lines

4. Work tables to be provided with grated tops, ventilated from below, and equipped with small narrow troughs filled with water, and attached to each side to prevent dust falling to the floor
5. All inlets to exhaust ventilating ducts to be below the level of the nose, as far as possible, to prevent fine dust particles from being inhaled
6. A chemical analysis of samples of air from various parts of the plant to be made at different intervals during working hours. (Any spot showing more than 1.5 mg. of lead per 10 cu. m. is a potential health hazard)
7. Employees in hazardous spots, as found under "6," to wear respirators. Where movements are confined to a small area, forced-draft respirators (fresh air from the outside) to be used. If exposure is to fumes, masks to be provided with canisters charged with activated carbon. (Respirators are only from 70 to 90 per cent efficient and must be inspected and changed frequently)
8. No dust allowed to accumulate on the floor
9. Floors to be wet at frequent intervals, and powdered compounds used, if possible
10. All cleaning to be done with large vacuum lines. No sweeping
11. To prevent the collection of dust on rafters, sills, etc., ventilating ducts to be installed, equipped with dust-filters near the ceiling or in mid-air
12. To avoid the contamination of outside air and surrounding territory, and as an economy measure, electrostatic precipitators to be installed in the stacks and flues
13. The personal hygiene of the employees to be closely guarded. Locker-rooms with showers, a lunchroom isolated from the plant, and a change of overalls twice a day to be provided. Hands and face to be washed before eating. No eating, chewing, or smoking during working hours

Factory Management.—All of the above find their fulfilment in intelligent plant or factory supervision. The experience of this clinic confirms the observation of others that most cases of lead intoxication arise in small, non-supervised establishments. Frequently those in charge do not realize they are handling a lead compound or lead alloy. As an additional preventive measure substitution of non-toxic materials for the lead compounds may, in certain processes, be practical. For example, aluminum or ferric oxide paint may in many instances be substituted for the paint containing a litharge base.

REFERENCES

1. Magnuson, H. J., and Raulston, B. O.: "Lead Poisoning in Roofers," *J.A.M.A.*, *114* (April 20), 1940.
2. Kehoe, R. A.: Address before Central Society for Clinical Research, November, 1939.
3. Aub, J. C.: "To the Editor," *J.A.M.A.*, *114* (June 1), 1940.
4. Aub, J. C., Fairhall, L. T., Minot, A. S., and Reznikoff, P.: "Lead Poisoning," *Medical Monographs*, Williams and Wilkins Co., Baltimore, 1926.
5. Aub, J. C., Robb, G. P., and Rossmeisl, E.: "Significance of Bone Trabeculae in the Treatment of Lead Poisoning," *Am. J. Pub. Health*, *22:* 285, 1932.
6. Gray, I., and Greenfield, I.: "High Calcium Therapy," *Indust. Med.*, *8:* 507 (Dec.), 1939.
7. Aub, J. C., Reznikoff, P., and Smith, D. E.: "Lead Studies; Effect of Lead on Red Blood Cells. Changes in Hemolysis," *J. Exper. Med.*, *40:* 151–172 (Aug.), 1934.
8. Ferguson, A. E., and Ferguson, T.: "Examination of the Blood Films in Relation to the Prevention of Plumbism among Shipbreakers," *J. Hyg.*, *34:* 293 (Oct.), 1934.
9. Shiels, D. O.: "The Ratio of Large to Small Lymphocytes in Persons Exposed to a Lead Hazard," *Med. J. Australia*, *1:* 847–849 (June 20), 1936.
10. Smith, Fred L., II, Rathmell, T. K., Marcil, G. E.: "Early Diagnosis of Acute and Latent Plumbism, *Am. J. Clin. Path.*, *8:* 471–508 (Sept.), 1938.
11. Blackman, S. S.: "Intranuclear Inclusion Bodies in Kidney and Liver, Caused by Lead Poisoning," *Johns Hopkins Hospital Bulletin*, *58:* 384 (June), 1936.
12. McCord, C. P., Minster, D. K., and Rehm, M.: "Basophilic Aggregation Test in Lead Poisoning," *J.A.M.A.*, *82:* 1759–63 (May 31), 1924.
13. McCord, C. P., Holden, F. R., and Johnston, J.: "Basophilic Aggregation Test for Lead Poisoning and Lead Absorption," *Indust. Med.*, *4:* 180–185 (April), 1935.
14. Hyler, M. D., and Bradley, W. R.: "Basophilic Aggregation Test as Early Diagnostic Factor in Absorption and Incipient Poisoning," *Indust. Med.*, *7:* 184–193 (April), 1938.
15. Barnes, E. C.: "Possibilities of Control of Lead Exposure by Examining Less than 24-Hour Urine Samples, *J. Indust. Hyg. and Toxicol.*, *21:* 9, 464–468 (Nov.), 1939.
16. Tompsett, S. L., and Anderson, A. B.: "Lead Content of Blood and Excreta," *Lancet*, *1:* 553–616 (March 11), 1939.
17. McNally, Wm. D.: *Toxicology*, Industrial Medicine Publishing Co., Chicago, 1937, p. 170.
18. Calvery, H. O., Laug, E. P., and Morris, H. J.: "Chronic Effects on Dogs of Feeding Diets Containing Lead Acetate, Lead Arsenate and Arsenic Trioxide in Varying Concentrations," *J. Pharmacol. and Exper. Therap.*, *64:* 364–387 (Dec.), 1938.
19. Lederer, L. G., and Bing, F. C.: "Effect of Calcium and Phosphorus on Retention of Lead by Growing Organism," *J.A.M.A.*, *114:* 2457–61 (June 22), 1940.
20. Taeger, H.: "Calcium Therapy of Lead Poisoning," *Klin. Wchnschrift.*, *16:* 1613 (Nov. 13), 1937.
21. Shelling; H. D., and Hopper, K. B.: "Calcium and Phosphorus Studies," *Johns Hopkins Hospital Bulletin*, *58:* 196, 1936.
22. Kehoe, R. A., and Thamann, F.: "The Excretion of Lead," *J.A.M.A.*, *92:* 1418, 1929.
23. Shelling, D. H.: "Effect of Dietary Calcium and Phosphorus on Toxicity of Lead in Rat; Rationale of Phosphate Therapy," *Proc. Soc. Exper. Biol. and Med.*, *30:* 248–254 (Nov.), 1932.
24. Gray, I., and Greenfield, I.: "Newer Concepts in the Treatment of Lead Poisoning," *New York State J. Med.*, *38:* 1313 (Oct. 15), 1938.

25. Gray, L.: "Recent Progress in the Treatment of Plumbism," *J.A.M.A.*, *104:* 200 (Jan. 19), 1935.
26. Belknap, E. L.: "Control Lead Poisoning in the Worker," *J.A.M.A.*, *104:* 205–210 (Jan. 19), 1935.
27. Aub, J. C.: "The Biochemical Behavior of Lead in the Body," *J.A.M.A.*, *104:* 87–90 (Jan. 12), 1935.
28. Holmes, H. N., Campbell, K., and Amberg, E. J.: "The Administration of Vitamin C in the Treatment of Lead Poisoning," *J. Lab. and Clin. Med.*, *24:* 1119–27 (August), 1939.
29. Haverfield, T. W., Bucy, P. C., and Elonen, A. S.: "The Surgical Treatment of Lead Encephalopathy," *J.A.M.A.*, *114:* 2432–37 (June 22), 1940.
30. Gant, V. A.: "Lead Poisoning," *Indust. Med.*, *7:* 693 (Nov.), 1938.

CHAPTER XX

MERCURY

THE case of mercurial poisoning as one sees it in accidental, suicidal, or therapeutic instances is not similar to industrial mercurialism. Industrial exposure to mercury was known to the Greeks as early as 400 B.C., but like lead it is still not well known to the average physician. Freeman reported its occurrence in fur-cutters and hatters in America in 1859.

Occupational Hazards.—There are about 100 occupations in which this metal offers a hazard. All of these need not be listed. The following list is but a few of the hazards and shows the form of mercury used:

Mercury	Manufacture of scientific instruments
Mercury nitrate ($HgNO_3$, $Hg(NO_3)_2$)	Felt-hat industry; decorating porcelain
Mercury fulminate ($Hg(CNO)_2$)	Constituent of all percussion caps
Mercury sulfide (cinnabar, HgS)	Mining and use as a pigment
Mercury cyanide ($Hg(CN)_2$)	Making cyanogen
Mercuric chloride ($HgCl_2$)	Manufacture of antiseptic dressings
Hatting industry ($Hg(NO_3)_2$)	Treatment of felt
Fire-gilding	Applying gold mercury amalgam to base metal and driving off mercury by heat
Mercury amalgam	For extracting gold and silver from ore
Mercury pumps	To exhaust air from incandescent lamps
Chemical works	As $HgCl_2$, etc.
Solder for dry batteries	
Dentists	Mercury amalgams
Mercury boilers	
Welders	

Pathology and Action.—Comparable to lead, the chief mode of entrance of mercury into the body in industry is by the respiratory tract. Ingestion, absorption through the skin, and injection into the

subcutaneous tissue are primarily used in therapeutic endeavor. The *inhalation of mercury* produces a reaction peculiar to body temperature, according to Biondi. If the inhaled vapors are lower than the body temperature, they circulate in the body as such or in combination, eventually to irritate the nervous system. If the mercury vapor in the air is of a higher temperature than the body or mucosa, the mercury is condensed on the mucosa to be absorbed as a mercury compound, giving rise to stomatitis, nephritis, or colitis. When this occurs, it causes inflammation at the point of elimination, as nephritis in the kidneys, stomatitis in the salivary glands, and colitis in the large intestines.

Mercury may be stored in the kidneys, liver, spleen, or the bones. It is eliminated in the urine, feces, saliva, sweat, or from the large intestines, bile, or milk. Its effect upon the nervous system may represent a diffuse encephalitis or symptoms of the particular center which it happens to predominantly affect. The ethyl group of organic mercurial compounds has an affinity for the cerebral lipoids resulting in a disturbance not unlike that of tetraethyl lead.

Mercury vapors in the body affect the lipoid tissue. The pathology of chronic mercurialism is not well understood. Mercury circulates in the bloodstream as an oxychloro-albuminate. Young people and females are more susceptible to its constant effects, while infection or alcoholism predisposes to chronic mercurial poisoning.

DIAGNOSIS

Signs and Symptoms. *Tremor.*—The most startling symptom, and one which leads to much confusion in the diagnosis, is the tremor which occurs in the voluntary movements. It is an intentional tremor, similar to that observed in paralysis agitans (not so regular), multiple sclerosis, or Basedow's disease. The hands, limbs, head, lips, tongue, or jaw may be affected, and this may be bilateral or unilateral. Textbooks for years have illustrated the characteristic handwriting of these victims, where the first few words are normal but succeeding words become more and more illegible. An illustration is found in the Mad Hatter in *Alice in Wonderland*. In New England they were called the "Danbury shakes."

Psychic Disturbances.—Various forms may occur, *e.g.*, unrest, irritableness, depression, fear, lack of concentration, and loss of self-confidence. In addition, generalized weakness, pains in the chest, vomiting, metallic taste in the mouth, and cramps in the leg muscles are sometimes present.

Tests. *Acute Poisoning.*—Acute poisoning is rare in industry. Marked gastric disturbance, characterized by alternating diarrhea and

constipation, varying degrees of distention with distress, stomatitis, and inflammation of the kidneys may occur.

Chronic Poisoning.—Symptoms of chronic poisoning are referable chiefly to the nervous system. When it is suspected or alleged, the urine or feces should be examined for the presence of mercury. If found, its presence does not mean that the patient is poisoned. Like lead, its finding must be combined with the history and symptoms. Blood examination is not of much value. There is frequently a relative lymphocytosis. The blood chlorides may be decreased.

A safe atmosphere for working conditions is that of 2.5 mg. per 10 cubic meters of air, or less.

MEDICOLEGAL ASPECTS

Temporary Disability.—Most patients respond to treatment and removal from exposure. Patients in mildly severe cases may have a persistence of symptoms from two to three months; in severe cases, they may be disabled for a period of eight to twelve months.

Permanent Disability.—Before the present state of hygiene, permanent tremors were not uncommon. Today, one rarely sees them. If they are present in older persons and those exposed for many years, Kessler[1] suggests a 25 per cent total award.

Illustrative Cases.—Mercury mining in California was a prominent industry during the first World War, but there followed a period of inactivity. Today these mines are booming because of the removal of Spain as a source of mercury, due to the second World War. However, the process is now so carefully guarded that mercurial poisoning rarely occurs.

In the city of Los Angeles there are 120 concerns engaged in the manufacturing of hats and furs; yet in the past five years not a single patient with mercurial poisoning has presented himself to our clinic. This is an indication of the high state of preventive medicine in an industry with known hazards.

The following case has been selected for illustration because of the description of the tremors that existed and the eventual outcome. The man was a patient of Dr. F. S. Ryan, of San José, California, and the case was reported by Dr. Alice Hamilton.[2]

Case I.—The patient is a Spaniard, employed first as a miner and then as a furnace man, and he was evidently unusually susceptible, for he had several attacks of poisoning while mining cinnabar, where the danger is very slight.

An attack would begin with a queer sweetish taste in the mouth, followed by colicky pains and diarrhea. There was never an inflammation of the mouth or salivation, nor had there been a loss of teeth.

The most serious symptom was tremor. From the mines the patient went to work in a reduction plant and was put on a Scott furnace where the fumes were very bad. During this time, his tremor was practically constant and very troublesome, especially if he was excited, sometimes making it impossible for him to feed himself. He drank a good deal of native California wine but no whisky.

In spite of the doctor's warning, he continued to work on the furnaces; then one afternoon at about three o'clock in his home in the mining camp, he suddenly began to have violent clonic spasms of the right leg, which, as he described it, "began to work up and down like a piledriver." The thigh would be drawn up on the abdomen, the leg flexed against the thigh, and then violently extended again, and all the efforts of his fellow-workmen failed to hold it still. According to Dr. Ryan, who saw him about three hours later, the right leg was jerking back and forth rhythmically about sixty times per minute without any variation and with great violence.

Morphine had no effect on the spasm, although it induced drowsiness. There was no change in his condition under treatment by purging, by applying hot packs, or by putting ice to the head, nor could he be made to sleep even with morphine. The next morning a supinator spasm of the right arm developed, synchronous with that of the leg. Chloroform was then tried, but the spasms did not stop until the stage of surgical anesthesia was reached, and then began again before consciousness was regained. There was no cerebral symptom except the insomnia.

Thirty-six hours after the onset, sleep was obtained under chloral, and the spasms diminished in violence. Under the continual administration of chloral, purgatives, and sweating, the spasms gradually died down, but did not cease entirely until the seventh day. The exhaustion was extreme, especially as the patient could only take liquid food and only a very little at a time, for he could not keep still long enough to swallow much.

He was discharged from the hospital, emaciated and weak, and for a year after that he could not work. He was a prey to headaches, insomnia, bad dreams, depression, and irritability. Nevertheless, he went back to the furnaces the following year and worked for six or eight months until his increasing tremor finally compelled him to give it up. I saw him at the works where he was employed as a teamster and he told me that he had never recovered his former health. He is always nervous, irritable, and easily upset, a statement which was heartily endorsed by the bystanders. Just recently he had had an operation for gastric ulcers, and under the excitement of preparing for it, his tremor returned for several days.

This case-report is an instance of marked tremor, unusual susceptibility, chronic ill health, and the return of tremors under excitement.

It should be pointed out that disability occurred some time prior to 1925. The standards of hygiene as carried out in plant or mines today would not have permitted this patient to remain in the hazard after symptoms became so pronounced, nor would they have permitted him to return to this hazard. One wonders what the status of this case was under the Workmen's Compensation Act in California, where occupational disease was a part of that law at that time. There appears to be no question but that the patient was disabled because of his work and that he was eligible for temporary disability for at least one year. Why this employer or insurance carrier permitted him to return to work is most peculiar in the light of present-day working standards.

Case II.—In the *Archiv für Gewerbepathologie und Gewerbehygiene*, Berlin, December, 1938, an observation not mentioned in American literature was found. Fellinger and Schweitzer report the clinical histories of three patients in whom severe vascular impairments developed in connection with mercurial poisoning. To be sure it is justifiable to inquire whether the vascular lesions are actually the result of a mercurial intoxication or whether their concurrence with mercurial poisoning is merely accidental.

Although positive proof is lacking, many factors militate for a causal rôle of the mercurial poisoning. In all three cases the vascular lesions developed at a time at which, although the early signs of mercurial poisoning had disappeared, the chronic manifestations and sequelae (dental lesions, tremor, and psychic changes) were completely developed. This appearance at a time when late lesions and sequelae develop seems to indicate that the vascular changes belong to the other manifestations of mercurial poisoning, the more so as the chronological connection is similar to that which is observed in lead poisoning, in which vascular impairments are comparatively frequent. Moreover it appears improbable that in comparatively young and hitherto entirely healthy persons, vascular lesions should develop with great rapidity, when it is considered that Raynaud's and Buerger's diseases generally develop at first sight insidiously, causing only minor symptoms.

These and other considerations make it probable that these vascular lesions and their sequelae have a causal connection with mercurial poisoning. It was impossible to make anatomic studies on the vessels of the three patients with mercurial poisoning, but the clinical manifestations of the vascular lesions resemble those of the various forms of obliterating endarteritis; however, in their unusually rapid, almost fulminant, course they differ from idiopathic vascular diseases.[3]

Case III.—Williams and Schram[4] report an incident of mercurial poisoning in 1937 when thirty-eight men suffered effects from exposure to this metal following cutting out the tops of stills by means of oxy-

acetylene torches. The lids of these stills were closed by mercury seals, and over a period of years small quantities of mercury would be spilled on the lids. Those particles which were not removed would be painted over from time to time by a spray process. The symptoms were gingivitis, salivation, headache, abdominal cramps, and in the more severely affected, diarrhea with bloody stools. One patient showed alternate albuminuria and glycosuria. The temporary disability was short in all cases, the longest being six days.

Case IV.—A fifty-seven-year-old general workman for a metal concern, of Los Angeles, presented a claim before the Industrial Accident Commission (January, 1941) for permanent disability due to: (1) loss of all teeth and (2) loss of all nails as a result of exposure to mercury. His total length of employment had been two years and nine months, although he had been absent from this process for one year before his final year of service. In 1938, at intervals, the patient was annoyed by "indigestion." His family doctor noted many decayed teeth and advised their removal, believing this would relieve the indigestion. All teeth were extracted in August of 1938. About five months before being seen by the writer, the patient noted that he was losing his nails, a condition which led to his stopping work.

The company for which this claimant worked is engaged in a process of obtaining mercury and antimony by the reduction of livingstonite concentrate, obtained from Mexico. This workman would take sacks of concentrate to a scale to be weighed; then to the elevator where the material was carried to a platform where it was dumped into a hopper (Fig. 44). He then carried the cinders which fell to the bottom of the furnace to a pile where they cooled and were re-sacked. These cinders when hot gave off a strong sulfur fume. The cinders also contained antimony that had been separated from the mercury. For ten minutes every hour the patient worked at the hoe table where mud containing mercury was spread out and under which was a fire. Also, every hour he drained mercury from the condensers into a flask.

An industrial engineer, after investigating the plant, reported that the process was carried on in a large building, which had a roof but no side walls. He found only one point where there could be any possibility of inhalation of mercury fumes, although the concentration was well within the permissible limits. This was after the mercury antimony had passed through the rotary kiln. Antimony and mercury are vaporized in this kiln and drawn by suction into a condenser for the first recovery of the metals. Antimony oxide resulting from kiln action is eliminated from the rotary kiln by gravity into a hopper, from which it is conveyed by bucket elevator to an elevated hopper. The oxide is then released from the hopper by means of a sliding door into small muck cars. Farther along in the process in the scrubbing and retort recovery of the mercury and antimony, sulfur dioxide is

generated, but this gas is carried off by suction control up to the exhaust chimney which is 102 feet high. The whole process is one designed to prevent an escape of mercury fumes, for such escape would mean a financial loss.

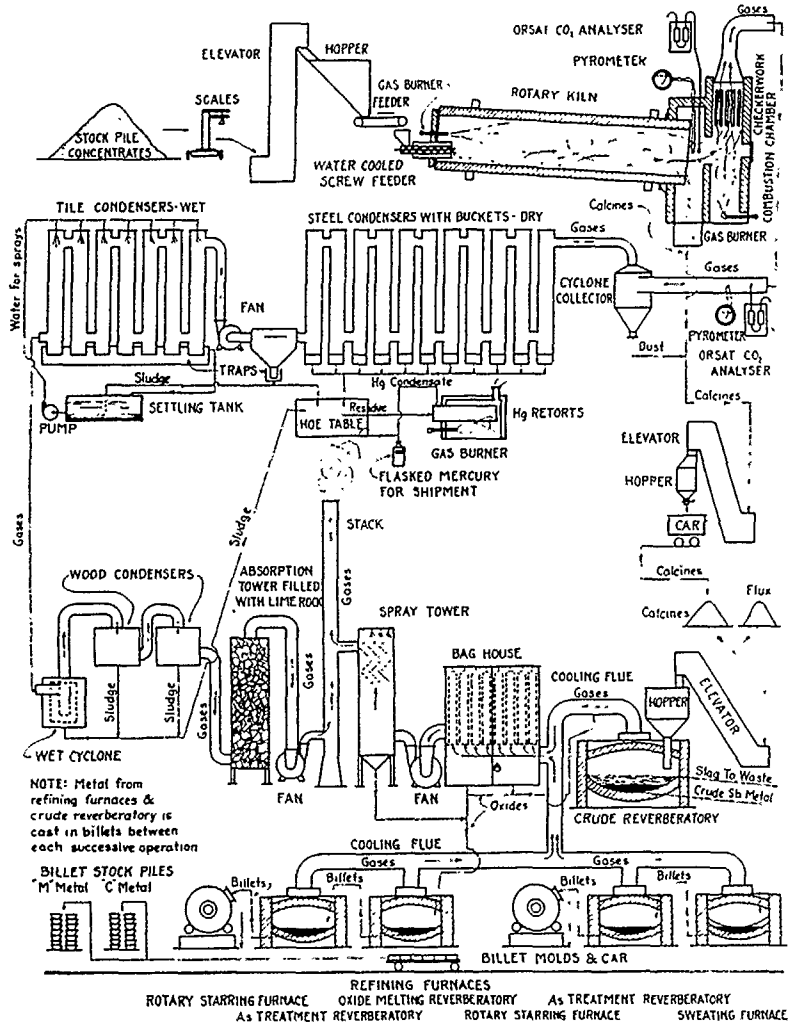


Fig. 44.—Reduction plant for treating livingstonite concentrate for the production of mercury and antimony metal. (*Mining and Metallurgical Bulletin*, Phenhoel-Menardi Engineering Co., Ltd.)

Physical Examination.—The patient is an adult male whose given age is fifty-seven. He appeared somewhat underweight. The scalp and hair were normal in appearance. The pupils reacted to light and accommodation, but were irregular and unequal. Teeth all false. Gums

clean; no discoloration or pigmentation. Throat: normal. Lungs: clear throughout; no adventitious sounds noted. Heart: sounds somewhat distant; no murmurs heard. Rate of 78, regular. Blood pressure, 138/90. Abdomen: normal in contour; no organs or masses palpated. There was a scar in the right quadrant due to a former appendectomy. Neurological examination revealed no abnormal findings. Right knee jerk normal. Left knee jerk exaggerated. Romberg negative. Gait normal. Examination of the hands revealed a loss of the fingernails of all fingers. This loss was not complete in every instance, there remaining at the tip of most of the fingers a small portion of normal nail. There existed no tenderness of the finger tips or to pressure on the nail bed. Examination of the feet revealed the large and second toes to have lost their nails in a manner similar to that noted of the hands.

x-Ray Examination.—Chest: No alteration of either pulmonary field is demonstrated. The markings are not unusually prominent for a patient of the stated age. No evidence of pleural thickening seen. The left ventricle is beginning to be prominent in keeping with the patient's age, but no other changes are recognized. The findings are essentially negative.

Laboratory Examination:

Blood		Urine
Hemoglobin	88 per cent	Voided specimen
Erythrocytes per cu. mm.	5,200,000	Yellow color
Leukocytes per cu. mm.	7,800	Clear appearance
Polynuclear neutrophiles	58	Specific gravity, 1.014
Lymphocytes	36	Acid reaction
Large mononuclears	3	Sugar, 0
Eosinophiles	2	Albumin, faint trace
Basophiles	1	Acetone, 0
Blood Wassermann: Negative		Microscopic, 0–1 pus cells

A consultant ruled out arsenic poisoning and fungous infection by laboratory examination of the nails. In his opinion even though the blood Wassermann was negative, the probable etiology was lues. A spinal Wassermann was not permitted. Since arsenic was eliminated from an etiological consideration, there remained antimony, sulfur dioxide, and mercury.

Little is known about the effects from chronic exposure to antimony, and it is believed that it is relatively non-toxic. In the foreign literature mention is made of a pustular, necrotic dermatitis com-

mencing as a folliculitis and resulting in atrophic scars. Colic, diarrhea, and loss of weight have also been mentioned by foreign writers. The claimant presented none of these conditions unless the vague type of indigestion he complained of could be considered a result of exposure.

Sulfur dioxide produces irritation to the upper respiratory system when inhaled in large doses, but the effect is usually transitory.

Mercury primarily affects the central nervous system, and tremors of the hands, legs, head, jaw, lips, and tongue may be noted. Psychic disturbances such as mental depressions, fear, and lack of concentration are sometimes present. In 1935 Neal and Jones[5] made a study of the effect of mercury upon workers in the fur-cutting industry. They found no evidence that mercury fumes or dust affected the lungs, nor was there a single case of pulmonary tuberculosis among their group. A fine intention tremor was noted in forty-three workmen, while tremor of the eyelids, tongue, jaw, head, and legs was also present in less frequency. These two investigators noted a frequency of two types of discoloration of the oral and nasal membranes, one a white line and the other a rich, copper-colored line. With both the membrane was dry, glistening, and when the condition was present it usually extended over the entire oral mucous membrane as far as the upper portion of the pharynx and vestibular portion of the nasal mucous membrane. In their study of the blood picture, no significant changes were noted. The percentage of lymphocytes in the blood was slightly higher than normal, but the percentage of monocytes was within normal limits, so they attached no importance to the slight increase in lymphocytes. Nor could they determine that the amount of mercury in the urine had any significant value, since in persons with pronounced mercurial poisoning, the mercury in the urine was consistently found not to be high. In discussing the paper of Neal and Jones, McCord[6] pointed out that in the cartridge industry the mercury fulminate is in a wet state, and therefore the type of exposure is somewhat different from what it is where fumes are in existence. He stated that in those who handle the wet mercury fulminate, ulcerated fingertips, erosion of the nails, and sore fingers appear.

In a report to the Industrial Accident Commission concerning our case under discussion, the author took the position that he could not state with any certainty whether mercury was or was not the cause of the disturbance to the nails. Onychomadesis is usually considered to be the result of some metabolic disturbance, such as diabetes, or of syphilis of the central nervous system. In this instance, diabetes was ruled out, but syphilis was not completely, and had to be considered as a likely cause.

Too little is known about the industries using mercury to be dog-

matic. Our claimant's exposure does not appear to be exactly comparable to the workers in mercury fulminate referred to by McCord, for in the latter instance it is inferred that these men handled the wet substance, whereas our claimant did not touch the mercury mud. However, the case herein reported is worth while in that it calls attention to the possibility of nail erosion from this or similar types of mercury exposure. It does not represent a permanent total disability, from the standpoint of loss of either teeth or nails.

TREATMENT

Acute Poisoning.—This is usually the result of intentional or erroneous ingestion of the bichloride of mercury and so will not frequently be seen in industry. However, as indicated by the cases reported by Williams and Schram, acute exposure may result in symptoms primarily involving the gastro-intestinal tract.

Gastro-intestinal Involvement.—In such instances gastric lavage is indicated at once. A number of chemicals have been used with the wash water to precipitate the mercury and to delay its absorption. The most promising agent so far seems to be sodium formaldehyde sulfoxylate. The chief difficulty is that its effect is not great if as long as fifteen or thirty minutes elapse before it is given. A freshly prepared solution of 10 per cent of the sulfoxylate with 5 per cent of sodium bicarbonate is used for the gastric lavage, and about 200 cc. of the mixture is left in the stomach. An intravenous injection of from 10 to 20 gm. of sulfoxylate in a 10 per cent solution should also be given over a period of from 20 to 30 minutes.

Its use except in the acute case seen quite early is open to question, since there is some experimental evidence demonstrating that while sodium formaldehyde sulfoxylate is of value in the prevention of the manifestations of poisoning, *it may actually aggravate preexisting intoxication.* Monte and Hull[7] felt that its repeated use was inadvisable and strongly doubted its efficacy in any instance. It is probable that acute mercury poisoning will be seen earlier in industrial than in private practice, and hence the therapy outlined may be of considerable value.

Lavage with three tablespoonfuls of charcoal and 20 gm. of magnesium sulfate to a pint of water is advised by McNally. One gram of charcoal binds 180 mg. of bichloride of mercury. Following this lavage two eggs in water and a glass of milk are administered. This is followed by another lavage with water, and finally four tablespoonfuls of charcoal are given by mouth (not to be removed). The milk which is used should have the cream removed, since fats dissolve mercury salts more quickly and aid in the absorption.

Pain and Prevention of Shock.—In these conditions, morphine sulfate, $^1/_4$ grain (0.016 gm.), every four to six hours is used. For further treatment in shock, caffeine sodium benzoate, $7^1/_2$ grains (0.5 gm.), or epinephrine, 1 cc. of 1:1000 solution, may be given hypodermically, and an intravenous infusion of 10 per cent dextrose in saline (1000 cc.) is indicated.

Subsequent Treatment.—Various therapies have been outlined for the subsequent treatment of the patient. Good results have been claimed for the intravenous use of sodium thiosulfate, 15 grains (1 gm.), in 10 cc. aqueous solution, from one to six times daily, for from three to five days. Hashinger and Simon[8] reported a patient treated chiefly by exsanguination-transfusion, with complete recovery. Korányi[9] reported complete recovery of a patient with beginning oliguria, hematuria, and albuminuria by the use of daily venesection (300 cc.), daily oral administration of 5000 cc. Ringer's solution, and the intravenous administration of 20 cc. of a 10 per cent solution of sodium chloride. Others have advised multiple transfusions of from 300 to 350 cc. Cecostomy has been used with good results.

It is very important that the lowered blood chloride level should be treated by intravenous administration of physiological saline, and that acidosis be treated as described under "Methyl Alcohol." Sodium citrate or acetate in amounts sufficient to keep the urine alkaline is given by mouth.

A regimen consisting of 250 cc. of the following mixture, 250 cc. of milk at hourly intervals has been used: potassium bitartrate, 1 drachm (4 gm.); sugar, 1 drachm (4 gm.); lactose, $^1/_2$ ounce (15 gm.); lemon juice, 1 ounce (30 cc.); boiled water, 16 ounces (500 cc). In combination with gastric lavage (with six quarts of sodium bicarbonate solution) and colon irrigation twice daily, daily sweating in a hot pack, and continuous rectal irrigation with a solution of potassium acetate (a drachm to the pint), it has given some good results.

For muscular twitchings of uremia, calcium gluconate, 10 cc. of a 10 per cent solution intravenously, is of value.

Chronic Poisoning.—No treatment of demonstrated value in chronic poisoning is reported. Removal from the hazard is, of course, absolutely essential. The use of sodium thiosulfate might be of value, 15 grains (1 gm.), in 10 cc. of sterile water intravenously every other day for a short time.

Nervous Disorders.—Most of the treatment in the chronic case is directed toward alleviation of the nervous affections. Since many of these patients have symptoms similar to those seen with chronic encephalitic parkinsonism, it has been suggested that a trial of drugs used in that disease be made. The older drugs of this group are scopol-

amine hydrobromide (hyoscine), from $\frac{1}{200}$ to $\frac{1}{100}$ grain (0.0003 to 0.0006 gm.), two or three times per day, or tincture of stramonium, from 60 to 90 minims three times daily. A drug used more recently has been amphetamine sulfate (benzedrine sulfate), from 20 to 30 mg. twice daily, at 8 A.M. and at noon, in conjunction with a drug of the atropine group, or genoscopolamine, in dosage of 0.5 mg. granules, one three times a day, increasing the dosage after several days to two or three granules three times a day. The latter is said to have far less toxic effects than atropine, hyoscine, and stramonium.[10]

Stomatitis.—For this, the following mouth washes may be used: tincture of myrrh, 1 part to from 25 to 50 parts of water; potassium permanganate, 1:8000; or

	Ounces	*cc.*
Thymol (saturated solution)	2	64.0
Hydrogen dioxide	2	64.0
Glycerine	2	64.0
Potassium chlorate (saturated solution) q.s. ad.	8	250.0

Good oral hygiene is imperative.

A diet high in vitamins with the vitamin B complex particularly added should be of value. Diarrhea or constipation, if present, should be controlled.

REFERENCES

1. Kessler, H. H.: *Accidental Injuries*, Lea and Febiger, Philadelphia, 1931, p. 718.
2. Hamilton, A.: *Industrial Poisons in the United States*, Macmillan Co., New York, 1925, p. 240.
3. Fellinger, K., and Schweitzer, F.: "Vascular Diseases after Poisoning with Mercury," *J.A.M.A., 112* (March 18), 1939.
4. Williams, J. E., and Schram, C. F. N.: "Acute Mercurial Poisoning," *Indust. Med., 6* (Sept.), 1937.
5. Neal, P. A., and Jones, R. R.: "Chronic Mercurialism in Hatter's Fur Cutting Industry," *J.A.M.A., 110:* 1938.
6. McCord, C. P.: Discussion of above paper.
7. Monte, L. A., and Hull, E.: "Mercury Bichloride Poisoning Treated with Sodium Formaldehyde Sulfoxylate," *J.A.M.A., 114:* 1433 (April 13), 1940.
8. Hashinger, E. H., and Simon, J. F.: "A Case of Mercuric Chloride Poisoning Treated by Exsanguination-Transfusion," *J. Lab. and Clin. Med., 20:* 231 (Dec.), 1934.
9. Korányi, A.: "Successful Treatment of Corrosive Mercuric Chloride Poisoning, with Repeated Venipunctures and Administration of Ringer's Solution," *Klin. Wchnschr., 14:* 753 (May 25), 1935.
10. Scharf, J. H.: "Genoscopolamine: Its Use in Parkinsonism," *J. Nerv. and Ment. Dis., 89:* 682 (May), 1939.

CHAPTER XXI

MANGANESE AND CHROMIUM

MANGANESE

MANGANESE poisoning is not a common industrial disease. Little has been written about it. Yet manganese is finding increasing use, and it is to be expected that the cases of manganese poisoning will be increased. The industrial demands for manganese ore have been met mostly by importing it, but it is produced in Montana, Georgia, Arkansas, Virginia, and to a lesser extent in Arizona, New Mexico, Nevada, and California.

Occupational Hazards.—Manganese, because it imparts strength and hardness to steel products, is employed in the steel industry in the manufacturing of alloys, such as silicomanganese, silicospiegel, ferromanganese and spiegeleisen, and in combination with copper, zinc, aluminum, and other metals. It is also used extensively in the manufacture of dry-cell batteries and to a lesser extent in calico printing, making of dye, fireworks, pottery, linoleum, and photographic reagents, and in germicides, deodorizers, and fertilizers.

Pathology and Action. *Effect on Nervous System.*—Information regarding its effect has come largely from animal experimentation. From these observations, combined with occasional autopsies on victims of this poisoning, it would appear that the central nervous system is primarily involved. Degenerative changes in the pons, caudate nucleus, striate nucleus, large ganglion cells, cortex, and basal ganglia have been variously reported. Interstitial nephritis, biliary cirrhosis, and proliferation of connective tissue in the lungs have also been noted.

Effect on Blood.—According to a United States Public Health Service[1] report, extensive laboratory examinations of workers indicated that a low white-cell count with a decreased percentage of neutrophiles, a slightly lowered blood calcium content, and a slight reduction in the middle zone of Lange's test, were often associated with manganese poisoning. A lowered hemoglobin content was observed in several of the manganese workers. Analysis of the blood for manganese content was of no assistance in this study.

Urinary manganese appears to reflect a present or past exposure to manganese compounds rather than evidence of intoxication. Impairment of kidney or liver function was not observed.

Effect on Lungs.—Chemical examination of the lungs of a person dying from manganese poisoning was reported to show from 3.07 to 4.87 mg. of manganese per 100 gm. of dry tissues in the right and left lungs, respectively, and very much smaller amounts in the other organs, indicating that he still had a reservoir of manganese in the lungs seven years after his last exposure to this dust. A few months before death he had been found to be excreting small amounts of manganese in the urine.

Mild and Severe Effects in Industry.—According to Voss,[2] the use of manganese is greater in Europe than in America. He points out that in comparison to the wide technical use of manganese and its compounds, the number of cases of manganese intoxication is small. He believes this is due to the fact that only a small number of persons have a predisposition for this poisoning; that not all manganese compounds are capable of exerting a toxic action; and that the danger of occupational poisoning occurs only where manganese compounds are inhaled or swallowed in adequate quantities for a comparatively long time. Voss observed a case of a man who had had a mild exposure for ten months and a severe exposure for seventeen months. Other men working under the same conditions developed no signs or symptoms. This patient showed a masklike face, amyostatic symptoms, disturbances in speech and gait, micrographia, laughing, weeping, and stuttering. Stored manganese was indicated by the presence of this substance in the feces eight months after the cessation of exposure. Nervous lesions in severe poisoning usually remain constant or increase in severity (as noted in the case reported by McNally), but the patient observed by Voss improved.

Bryan[3] reported a case of manganese poisoning in a forty-seven-year-old man who sought medical treatment because of disturbance of gait. The initial symptom in three of six patients examined by Gayle[4] was a disturbance of gait. Canavan, Cobb, and Drinker[5] were fortunate to observe the autopsy findings in a sixty-nine-year-old man who died of cardiorenal disease fourteen years after the onset of symptoms resulting from this occupational hazard. This man had been exposed to manganese for four years. His symptoms had been disturbance of gait, stiffness of the legs, frequent falls, fatigability, and sleepiness. The chief autopsy findings were degeneration of the nerve cells, satellitosis, and gliosis in the basal ganglia. There was also widespread atrophy in various portions of the brain. The bulletin of the United States Public Health Service showed pictures of patients walking but dragging their feet, their difficulty in attempting to walk up an inclined surface, and their falling down when they tried to walk backward (Fig. 45).

PATIENTS DRAGGED THEIR FEET,

HAD DIFFICULTY WALKING ON INCLINE,

AND FELL DOWN TRYING TO WALK BACKWARD

Fig. 45.—Patients with chronic manganese poisoning. Photographs taken with a motion-picture camera. (U. S. Public Health Service, *Bulletin 247.*)

Diagnosis

Signs and Symptoms.—Languor, sleepiness, stolid, masklike facial expression, low monotonous voice, muscular twitching, cramps in the leg muscles, increase in tendon reflexes, ankle and patellar clonus, retropulsion and propulsion, slapping gait, and uncontrollable laughter are some of the signs and symptoms in manganese poisoning. The patient is usually aware of muscle stiffness and twitching first, and he then notices difficulty in walking.

Differential Diagnosis.—Paralysis agitans, multiple sclerosis, and progressive lenticular degeneration must be differentiated from chronic manganese poisoning. The occupational history is, therefore, important in initiating suspicion that one is dealing with manganese poisoning.

Tests.—There is no single laboratory finding which offers conclusive evidence of manganese poisoning. Manganese may be found in the excreta, but this only indicates the fact that manganese has been absorbed. In suspected cases a low white-cell count with a reduced percentage of neutrophilic leukocytes is a significant confirmatory aid. In the work conducted by the group under the supervision of the United States Public Health Service they failed to find any significant changes in the bones upon *x*-raying a series of men poisoned by manganese; nor did they find any value in the study of blood analyzed for manganese. They did note that the blood calcium was lower on the average in the poisoned patients than in the non-affected. A slight rise in the middle zone of the colloidal gold curve was thought to be noteworthy. These workers also noted a reduction in the middle tubes of Lange's test similar to that observed in paralysis agitans. It was held that the Lange test may prove helpful in differentiating manganese poisoning from active multiple sclerosis.

Test of Atmosphere.—In cases where a questionable history of exposure exists, it may be of advantage to test the atmosphere at the place of occupation. It is thought that 50 mg. of manganese per 10 cubic meters of air is a safe atmosphere for workmen.

Test of Blood or Urine.—If it is felt necessary to establish the presence of manganese in the body, the urine or blood may be examined in the following manner: Blood or urine is evaporated to dryness in a silica dish in the presence of sodium carbonate and ashed in an electric muffle. Add sulfuric acid until effervescence ceases, then a few drops of nitric (1:3 dilution). Filter, and to the filtrate add five drops of 2.5 per cent silver nitrate, and 0.3 gm. of crystallized potassium persulfate. Place beaker or test-tube containing the unknown in warm water. In a few minutes a purple color of permanganate appears. Run a blank on all chemicals and dishes used. Make

up to 50 cc., compare with a standard permanganate solution containing from .002 to .025 mg. of manganese. (See Sutton's *Volumetric Analysis*, 11th ed., 1924, p. 269, for details.)

MEDICOLEGAL ASPECTS

Temporary Disability.—With a short exposure and the removal of the workman early from the hazard, complete recovery may be expected. The length of this temporary disability is extremely variable. It is rarely short.

Permanent Disability.—If a severe exposure has occurred and marked neurological symptoms are present, it may be expected that the disability is permanent and may be progressive. However, life may not be necessarily shortened, for patients often live for years, eventually to die of some unrelated cause.

Illustrative Cases:

Case I.—White male, aged fifty.

Occupational History.—This man quit working in the manganese plant in 1918, after having worked there for two years running a pulverizing machine. He stated that he was grinding both chrome and manganese ore at that time, working inside the whole time. He stated that the dust conditions were particularly bad, and he did not wear a respirator. He was transferred out of this plant in 1918 because of difficulty in walking. Following this, he worked with the mixing pans in the brickyard for six years and after that running the pump and tending boilers in the brickyard for three years. He was assigned to this light work on account of partial disability. Prior to this industrial work, he had farmed for thirteen years and had been a mule-tender in a woolen mill for three years. He had not worked at all since 1928.

Personal History.—His wife was living and well. They had two children living. His wife had one miscarriage after influenza in 1927. He admitted being a light user of liquor and a rather heavy user of tobacco, mostly in the form of cigars.

Past Medical History.—The patient stated that he had had frequent severe attacks of tonsillitis. He had typhoid fever at the age of thirteen and was in the hospital for five weeks. He had neuralgia of the face years before when his teeth were bad. He was operated on for hemorrhoids in 1930. He had a chronic, severe cough while working in the manganese mill, lasting about one and one-half years.

Present Medical History.—Patient dated the onset of his present condition back to 1918, about the time he was transferred out of the manganese plant. It began with a slight weakness, which grew worse and still persists. Marked disturbances in gait, with stumbling, began

to trouble him, with inability to lift the right foot. He was troubled with weakness and cramps in the legs, falling down on going backwards, inability to turn around, and difficulty in going up and down hills, especially the latter. He noticed moderate drowsiness, which has persisted. Metallic taste and loss of appetite were noted. He had generalized tremor of the muscles, muscular weakness, especially of the right leg, and muscular cramps, worse at night, throughout both legs. He was troubled with numbness and tingling of the forearms, hands, feet, and legs, which was intermittent, and a stuttering, halting speech. He had some difficulty in swallowing food.

Since 1925 he had noticed short attacks of impulsive weeping. There had been no change in his sexual functions. His wife had had three pregnancies during this illness. Since the onset of these symptoms he had noticed a slight shortness of breath on exertion. He stated that he had noticed black spots and stars in front of his eyes at various times during the last twelve years. He had occasional slight attacks of palpitation of the heart. He had occasional twitching of the fingers and legs, mostly when sitting down. Several of these symptoms had been improving in the past few years. The trembling of the extremities, and muscle tremor in general, was improving, being intermittent in character and troubling him only occasionally at the time. However, the weakness in the right leg seemed to be getting worse. He believed his ability to talk was improved considerably.

Physical Examination.—White male, aged fifty, not acutely ill. Height, 71 inches; weight, 142 pounds; temperature, 97.4° F.; almost white hair; eyes and ears, essentially normal; moderate obstruction to breathing was present in nose; rather marked pyorrhea alveolaris, a slight amount of dental caries, all upper teeth were out; a coarse, moderate tremor of the tongue was noted; neck, normal.

(*a*) *Chest.*—Girth at inspiration, 94 cm.; at expiration, 86 cm. The physical examination of the chest was essentially negative. The stereographic *x*-ray films of the chest were essentially negative.

(*b*) *Circulatory System.*—The heart examination was essentially negative except for some diminution of all heart sounds. Before exercise the heart rate was 80; immediately after exercise, 96; and three minutes after exercise, 80. The blood pressure before exercise was 124/84; immediately after exercise, 126 86. No peripheral arteriosclerosis was noted. The electrocardiogram showed a heart rate of 76; conduction time, 0.12 seconds; left axis deviation and low voltage, 0.45 millivolts.

(*c*) *Abdomen.*—Except for a left inguinal hernia, supported by a truss, the examination of the abdomen was negative.

(*d*) *Extremities.*—Examination of the extremities revealed atrophy of the left thigh and calf, the circumference of the left calf being 2½ cm. less than the right.

Neurologic Examination.—Besides the atrophy noted in the left thigh and calf, there was a general diminution in power of all groups of leg muscles. The facial muscles were not affected, except for those of the forehead. Only tongue tremor was observed. The ankle jerks were slightly decreased. Both Achilles tendons were abnormally sensitive to compression. The Romberg test was slightly suggestive but not positive. The gait was spastic and slapping in character. He had a right foot drop and had to swing the leg to walk. Retropulsion and propulsion were noted, with the subject falling down on walking backward. On the inclined plane he had marked difficulty walking up, owing to the right foot drop, and he fell down in walking down backwards. Walking sideways was quite slow and tedious. There was no intention tremor in lifting the water glass. There was a slight stuttering observed with fast economical speech, indistinct at times. Masked facies was not observed. A specimen of handwriting revealed no intention tremor or micrographia. The examination of the skin was negative. The psyche was unaltered.

Laboratory Findings. (*a*) *Urine.*—Specific gravity, 1.013; amber; acid; no albumin or sugar; 0.006 mg. Mn per 1; no casts; occasional red blood cell; 1 or 2 pus cells per high-power field.

(*b*) *Blood.*—Erythrocyte count, 4,900,000; 14.5 gm. hemoglobin per 100 cc. (86 per cent); color index, 0.88; reticulocytes, 0.30 per cent; white blood count, 10,200; platelet count, 255,000.

(*c*) *Schilling Differential Count.*—Showed 53 per cent segmented neutrophiles, 39 per cent lymphocytes, 6 per cent monocytes, 1 per cent eosinophiles, 1 per cent basophiles.

(*d*) *Coagulation Time.*—1 minute 50 seconds; bleeding time, 3 minutes.

(*e*) *Kahn test*, negative.

(*f*) *Blood Chemistry* (mg. per 100 cc.).—Blood glucose, 93; total non-protein nitrogen, 40; serum chloride, 366; serum calcium, 9.5 and serum phosphorus, 3.9.

(*g*) *Cerebrospinal Fluid.*—Clear and colorless; very faint trace of globulin; cell count, 2; mastic negative; colloidal gold curve, 0001000000; Kahn test, negative.

(*h*) *Galactose Tolerance Test.*—Only very slight trace of galactose eliminated.

Diagnosis.—Chronic manganese poisoning, well advanced.

It is interesting to note that although this man left his manganese exposure in 1918 (some 19 years previously), he still had rather marked disability, especially in the use and control of his legs. While many of the symptoms had improved considerably, this case demonstrates that there are certain permanent organic changes.[1]

Case II.—McNally[6] reports a patient seen by him in 1925. The patient gave a history of having worked in the ore department of a battery company, transporting, loading, and handling in various ways 58 per cent manganese in powdered form. After fourteen months of this type of work, he first noticed that after dumping a wheelbarrow of dust he stepped back and had difficulty in stopping himself from falling. In a week's time the condition became progressively worse. Within the next six months the patient noticed that if he walked too far he developed a rapid gait which ended in a fall unless someone was present to assist him. This tendency progressed until he had great difficulty in walking alone and would not trust himself to do so. The condition was aggravated by fatigue and excitement. He subsequently developed a coarse tremor of the right leg, marked disturbance in walking; he had propulsion, retropulsion, and lateropulsion, and tremor on effort, such as dressing himself. The tone of his voice became more monotonous, and a mask-like expression of his face persisted. McNally noted in the laboratory examination a slight increase in the lymphocytes and also in the monocytes. The liver-function test was negative.

Treatment

Prophylaxis.—The prevalence of this disease can be limited by engineering control. Isolating the process under hoods or exhaust ventilation, control of the dust by wet processes, the use of mechanical conveyors, and examination of the atmosphere at intervals are all preventive measures. Jones[3] observed no cases of intoxication among men working in an atmosphere of less than 30 mg. of manganese per cubic meter of air. He states that this should not be considered a threshold limit and suggests an atmosphere of 50 mg. per 10 cubic meters as a safer one. Quarterly medical examinations should be routine in any mill or place where manganese compounds are handled in order to detect early signs or symptoms. Mild neurological signs or symptoms, such as drowsiness, languor, muscular cramps, and twitching, perhaps coupled with an otherwise unaccountable low white-cell count, indicate removal of the workman from the hazard. Daily showers following work should be encouraged by installation of shower baths and education of the employee in this regard.

Nothing of real value in the treatment of these patients has been described. For those mildly affected, much improvement, perhaps total, will occur on their removal from the exposure. For those severely affected, little improvement may be expected.

Vitamin B_1 and Calcium.—Well-rounded, adequate diets should be given these patients, and general tonics may be used. There is some question concerning the use of thiamine hydrochloride (vitamin B_1) since experimentally large doses of vitamin B_1 encourage retention

of manganese, and hence B_1 might be *contraindicated* here. Since low blood calcium levels have been demonstrated by some, oral calcium preparations and milk may well be included in the diet. The patient should be encouraged to drink adequate amounts of fluids and should be given cathartics, if necessary, in order to secure adequate elimination.

Sodium Thiosulfate and Liver Extract.—McNally[6] thought a patient of his showed some improvement while 15 grains (1 gm.) of sodium thiosulfate in 10 cc. aqueous solution was given intravenously every other day. He cites the feeding by Charles[7] of liver to these patients with what appeared to be improvement in some. When improvement under liver was noted, it was rapid and occurred in the first few days or so of the therapy. The use of liver extract intramuscularly, then, in dosages of from 15 to 30 units a day for four consecutive days, and the repetition of this dose two or three times a week over an indefinite period of time, depending upon the individual patient, is suggested.

REFERENCES

1. "Chronic Manganese Poisoning in an Ore-Crushing Mill," *Public Health Bulletin No. 247*, 1940. (Case 71.) United States Public Health Service, Division of Industrial Hygiene.
2. Voss, H.: "Occurrence of Occupational Manganese Intoxication in the Steel Industry," *Arch. f. Gewerbepath. u. Gewerbehyg., 9:* 407–507 (July), 1939.
3. Bryan, A. W.: "Chronic Manganese Poisoning," *Arch. Neurol. and Psychiat., 37:* 1448, 1937.
4. Gayle, R. F.: "Manganese Poisoning and Its Effect on the Central Nervous System, Report of Six Cases," *J.A.M.A., 85:* 2008–2011, 1925.
5. Canavan, M. M., Cobb, S., and Drinker, C. K.: "Chronic Manganese Poisoning. Report of a Case with Autopsy," *Arch. Neurol. and Psychiat., 32:* 501–512, 1934.
6. McNally, Wm. D.: "Manganese Poisoning Case," *Indust. Med., 4:* 349–350 (July), 1935.
7. Charles, J. R.: "Manganese Toxemia; with Special Reference to the Effects of Liver Feeding," *Brain, 50:* 30, 1927.

CHROMIUM

Chromium is found in several minerals, chiefly as chromic iron or chromite. Industrially the bichromates of potassium and sodium and chromic acid concern us. Chromium as a metal is inert, but chromic acid and its compounds, because of their caustic action, affect the skin and mucous membranes.

Occupational Hazards.—In the manufacturing of coal-tar dyes, wallpaper, electric batteries, and explosives; in textile printing; in certain paints, such as lead chromate; in lithography; and most commonly of all in photography and electroplating.

Signs and Symptoms.—There have been a few cases reported in literature where some form of the chromium has been ingested with

serious or fatal results. These do not deserve consideration in this discussion.

Ulcers.—Industrially, the acid produces lesions around the base of the nails of the hands, on the knuckles, dorsum of the hands, and the forearms. Occasionally they affect the eyelids and often the nasal mucosa. The ulcers usually develop slowly, and the workman is often unaware of their presence until they become big enough to note upon inspection. Sometimes they may be painful, especially if they occur over the knuckles, where flexion is hindered. These ulcers may become fairly deep (Fig. 46). The ulcers are usually small, varying in size from the head of a match to the end of a lead pencil, but often quite

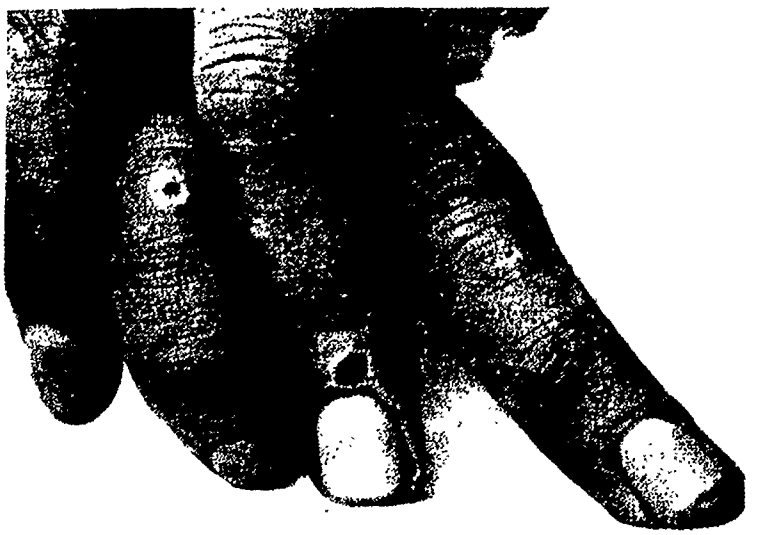


Fig. 46.—Healing chrome holes. The permanent scarring is well seen. (White: *The Dermatergoses or The Occupational Affections of the Skin*, Paul B. Hoeber, New York.)

deep. The edges are thick, rounded, often glossy and rarely inflamed. They may persist indefinitely unless treated. On healing there is usually a permanent residual scar. Invariably they cause no apprehension until they occur in the cartilaginous portion of the nose.

Allergic Reactions.—Instances of allergic reaction to the chromates have been reported in which the affected persons developed in addition to a dermatitis, asthma, fever, nephritis, and the like.

Medicolegal Aspects. *Temporary Disability.*—This is short. Treatment usually stops the progress of the lesions, and removal from exposure or the employment of preventive measures permits the affected person to continue his work.

Permanent disability is not to be considered. Claims for disability almost always are based upon impairment of function of the nose owing to ulcerations of the nasal mucosa or perforation of the septum. Many nasal perforations due to chrome have been seen by the writer, but in no instance did it impair function or constitute a disabling condition. The British observers failed to note any carcinomatous lesions resulting from chrome ulcers. Teleky[1] reports an incidence of lung cancer among chromate workers. When and if such a causative agent for cancer is claimed, the basis for decision must rest upon the precepts outlined under "The Medicolegal Relationship of Trauma to Disease."

Treatment. *Scrubbing Tissue.*—If the lesion or lesions ("chrome holes" or ulcers) are small and superficial, the damaged tissue may be scrubbed thoroughly with 5 per cent sodium hyposulfite, water, or saline solutions. The purpose of this treatment is to dislodge all tissue containing chromium compounds so that additional damage by burrowing will not occur. After this scrubbing, the wound may be treated like any abrasion of similar severity.

Wet Dressings.—If the ulcers are already too deep for such scrubbing, wet dressings of sodium hyposulfite for a period of from three to five days should be applied to aid in the reduction of the chromium compound. Since the chromium in the flesh is not dislodged by sodium hyposulfite, wet dressings of from 5 to 10 per cent solution of sodium citrate, sodium lactate, or potassium and sodium tartrate should be applied to accomplish this. These wet dressings are continued for from three to five days. At times necrotic tissue in the ulcer will require curettage. Suppuration rarely occurs in chrome ulcers, but cleansing with a mild antiseptic agent following use of the reducing agents may be used. Treatment appropriate for any ulcer is used following the above-outlined therapy.

It is frequently desirable to remove the worker from his customary duties, at least during the early stages of treatment.

Prophylaxis.—Prophylactic measures consist of removal of fumes and dust from the atmosphere, cleanliness within the plant, and confining the process within enclosures wherever possible. Respirators or gas-masks should be worn. Shower baths and change of clothing should follow the day's work, and the installation of showerbaths and locker-rooms will encourage this procedure. The skin should be protected by suitable clothing, rubber gloves, protective ointments, or oils. The nasal mucosa should be sprayed with an oil prior to going to work.

REFERENCE

1. Teleky, T.: "Occupational Cancer," *J. Indust. Hyg. and Toxicol.*, *19*: 2, 1937.

CHAPTER XXII

CADMIUM

FROM the amount of space devoted to cadmium poisoning in textbooks on toxicology and industrial hygiene, one would be led to believe that such an intoxication is extremely rare. There is good reason to believe, however, that cadmium poisoning occurs with relative frequency but remains unrecognized as such. Furthermore, it can be safely assumed that the incidence of cadmium poisoning will increase, especially during these years of intensive production for national defense. As is the situation with all industrial hazards, the large well-supervised establishments will prevent the occurrence of this illness by precautionary methods, but the small plant is apt unwittingly to expose workmen to the fumes of this metal. It is obligatory, then, that all who deal with industrial health be better informed regarding cadmium. A recent personal experience with an acute fatal case of cadmium intoxication enables me to present an accurate description of the various phases of this illness.

Cadmium was first used as a coating for piano strings and other musical instruments because it formed a protective covering against rust. It was subsequently found to take on a high polish. Cadmium is also included in specialized solders. Since it has a relatively high melting point and is capable of withstanding excessive wear, it is being used extensively in the automobile industry. Undoubtedly with the demand for machinery of all types, the need for cadmium will be increased. Pennsylvania produces a large part of this country's supply of this metal in the form of cadmium sulfide. The more important compounds of cadmium are the oxides, sulfate, sulfide, chloride, iodide, and bromide.

The failure to be concerned about, or to recognize, cadmium as an industrial poisoning is largely attributable to the fact that it is used with other metals and to a peculiarity of the symptoms it produces on undue exposure. Unquestionably, ill effects from the fumes of this metal have invariably been ascribed to lead or to metal-fume fever, as our two cases will show. Also, because the serious symptoms are sometimes delayed, cadmium poisoning may be confused with diseases of non-occupational origin. Because of this delayed reaction, a workman may fail to withdraw immediately from his exposure.

Occupational Hazards.—These are encountered largely in the smelting of ores; in the making of alloys, vapor lamps, and storage batteries; in electroplating, glass coloring, spraying of pigments, plating of automobile parts and musical instruments, and especially in welding.

Pathology.—Because of animal experimentation, the reports have indicated a more extensive pathology than is probably found in the human. Prodan[1] fed cadmium to cats to find subsequent involvement of nearly all of the viscera. But industrial cadmium poisoning results from inhalation, not ingestion. Legge[2] reported an autopsy of a workman exposed to the fumes of cadmium resulting from the melting of cadmium ingots. According to Legge, the autopsy of this man revealed inflammation of the lungs, gastro-intestinal tract, kidneys, and spleen. In 1935 from two plants located in Ontario, fifteen cases of cadmium poisoning occurred and were reported by Bulmer, Rothwell, and Frankish.[3] Two of these men died and were autopsied, at which time the findings indicated predominant involvement of the lungs. According to these men, the postmortem examination gave the following anatomical diagnosis:

Cadmium poisoning, edema and congestion of the lungs (chemical), hemorrhages into the lung (chemical), partial collapse of lungs, proliferative interstitial pneumonitis (chemical), catarrhal bronchitis (chemical), cloudy swelling of the liver and kidneys, congestion of the spleen, and fatty infiltration of the pancreas.

Microscopic examination of the lungs showed congestion, hemorrhage, edema, alveolar collapse, desquamation of the lining cells of the alveoli, and marked proliferation of the lining cells of the alveolar spaces.

This report of these Canadian investigators indicates that the lungs bear the brunt of the assault from cadmium-fume inhalation. Testimony to this is found in the autopsy of our patient, to be subsequently presented in detail.

DIAGNOSIS

Signs and Symptoms.—The first symptom is that of a distressing cough, to be followed within a short time to several hours by headache, dizziness, and eventually by a feeling of constriction within the chest. Accompanying the early symptoms are often dryness of the throat and sometimes nausea or vomiting. At the time the chest pain sets in, shortness of breath is also noted. The patient may feel alternately hot and cold, but it is an interesting observation that an elevation of temperature does not usually occur early but invariably at the terminal stage of the fatal cases.

Pulmonary Involvement.—If the exposure is severe, the symptoms become intensified. Of these, the excruciating chest pain and the excessively increased respiratory rate are most striking. While the above symptoms point to profound pulmonary involvement, signs within the lungs are meager, if any. The breath sounds may be of the exaggerated bronchovesicular type, but râles are often few or absent until the terminal stage. Also, while the *x*-ray may reveal widespread patchy bronchopneumonia, indications of large areas of consolidation are not heard.

MEDICOLEGAL ASPECTS

No toxic limit has as yet been established. From the author's experience, inhalation of even minimal doses is hazardous. Nor is it known how long after cadmium poisoning has occurred that the presence of this metal can be found in the body tissues. The cases so far reported have resulted from short, severe exposures. Unless death occurs, the illness is rarely longer than one or two weeks at the most. To date there exists no reason to believe that a permanent disability results.

Illustrative Cases.—Before reciting the more interesting details of a fatal case of the writer's experience, a mild illness resulting from inhalation of cadmium fumes will be reported.

Case I.—In June 1940, a twenty-four-year-old welder came to the clinic stating that on the day previously he had welded some "galvanized rods." His respirator was defective, but knowing that the job would be of short duration, he did not secure another respirator. Shortly after finishing this work he noticed an irritating cough, headache, and slight dizziness. That night he had chills, noted a dryness of the throat, the cough became more constant, and by morning he was aware of a heavy, oppressive feeling in his chest.

His examination revealed nothing unusual except increase in the pulse and respiratory rate. The temperature was normal; pulse, 90; respirations, 26; blood pressure, 116/78. The urinalysis and blood count were not significant, and the *x*-rays of the lungs were negative for evidence of any pathological process.

Since the patient's illness resembled in some respects a metal-fume fever, inquiry was made at the plant where he worked regarding the substance being welded. I was informed that these rods were cadmium-coated. The patient returned to his work six days after exposure free of all symptoms.

Similar to the above case is a report[4] of an instance of intoxication due to the overheating of cadmium. The exposure, although brief, was followed shortly by a sensation of dryness of the throat, weakness,

difficulty in breathing, together with pain across the anterior chest. The slightest effort precipitated paroxysms of coughing. The patient was confined to bed for five days and returned to work thirteen days after exposure.

Case II.—The following case of the author's experience represents a contribution to the study of cadmium poisoning in that he was not only able to observe this case clinically, but secured complete laboratory studies, postmortem findings, and microscopic slides of the various tissues of the body.

History.—A twenty-seven-year-old Mexican laborer employed by a metal company, of Los Angeles, was referred to me on April 14, 1941, with a diagnosis of metal-fume fever. Before proceeding with the details of this case, a defense of the physician who made the diagnosis of metal-fume fever is justifiable and illustrates the confusion regarding cadmium poisoning which was mentioned in an earlier paragraph. This doctor has for years treated workmen from this plant for the ordinary traumata incident to industry, and has also treated numerous patients from this same plant for metal-fume fever and lead poisoning. He was never informed that cadmium was present in a new process used by this company. Furthermore, although a competent industrial physician, he told me in discussing this case that his "reading material" had never mentioned the toxic effects of this metal. This digression well illustrates the fact that employers fail to realize that they expose their workmen to a hazard when cadmium is heated and that the physician has likewise not yet been impressed with the extreme toxicity of cadmium fumes.

To return to our patient: While he managed to walk into the examining room, he was so exhausted that he was unable to speak for several minutes. He was obviously extremely ill, and after speaking a few words would stop to regain his breath. No ambulatory cardiac, or even one suffering from a cardiac insufficiency of severe degree and confined to bed, presented more extreme dyspnea. While this patient talked he clutched his upper chest. After considerable waiting, the following meager history was obtained.

Three days previously, April 11, 1941, he had crawled into a rotary furnace, thirty inches in diameter, to cut V-shaped openings from two longitudinal fins. (Details of his activities will be discussed in the comment on this case.) He wore no mask, and although the fumes caused him to cough, he finished the work, without stopping, in about forty minutes. Upon emerging from the furnace he was coughing considerably, but continued with other duties for the rest of that day. During the latter part of the afternoon his coughing became distressing, and he felt dizzy. That night severe headache set in, coughing became constant, and during the night he became aware of a gripping chest pain. Sleep was impossible. He related that he would try

sleeping in bed, then in a chair, then back in bed again. This stirring around increased his cough as well as his shortness of breath. The morning following his exposure, his illness was reported to his employer, who sent him to the physician referred to previously.

The afternoon of the day following his exposure, the patient's wife had him look into a mirror that he might see how blue his lips were. In spite of the administration of codeine, his chest pain increased in severity. The patient described this chest pain by closing his hands as if he were tightly squeezing something.

Physical Examination.—Inspection revealed this Mexican workman to be cyanotic and extremely dyspneic, with a facies that indicated he was suffering severe pain. His temperature was 98.4° F. upon admission; the throat was not inflamed; and in spite of a respiratory rate of 40 per minute the lung examination showed evidence of no moisture or consolidation. The heart had a regular rate of 122 beats per minute, while the blood pressure was 140 systolic; 90 diastolic. The rest of the examination was not remarkable.

Twelve hours after admission, eighty-four hours after exposure, his temperature rose to 100° F.; respirations remained at the rate of 40; the pulse was 130 beats per minute; the lungs remained clear. Pantopon, ⅓ grain, did not relieve his chest pain. Unbearable headache accompanied his chest pain, as did dryness of the throat and cough upon the least exertion.

Laboratory Examination.—The initial laboratory findings were as follows:

Urine

Specimen	Voided
Color	Yellow
Appearance	Cloudy
Specific gravity	1.021
Reaction	Acid
Albumin	One plus
Amorphous deposits	None
Pus cells	1–2
Mucous threads	Numerous

Blood

Hemoglobin	89 per cent
Erythrocytes	5,200,800
Leukocytes per cu. mm.	14,800
Polynuclear neutrophiles	84 per cent
Lymphocytes	12 per cent
Large mononuclears	2 per cent
Eosinophiles	1 per cent
Basophiles	1 per cent
Stippled R.B.C.	None

x-Ray of Lungs.—There were mottled increases in density throughout both lung fields. The pleural margins were clear. The cardiac shadow appeared to be normal in size.

On April 16, two days after admission to the hospital and five days after his exposure, the temperature rose to 104° F., the pulse rate to 140, and the respirations to 50. The leukocyte count was 21,100, with 87 per cent polynuclear neutrophiles; the urine remained unchanged. A bedside *x*-ray film revealed multiple patches of increased density throughout the entire lung fields.

Worthy of comment is the fact that although this patient had been placed in an oxygen tent at the time of admission, it did not affect his respiratory rate. When under the tent he was definitely more comfortable and the chest pain less severe, but the breathing continued to be rapid and shallow, with movement confined to the area of the diaphragm. While the breath sounds were of the exaggerated bronchovesicular type, no moisture could be detected within the lungs.

For the next two days there was little change in the patient's general condition except for a drop in the temperature and pulse rate, but the respirations continued to be excessively rapid. There was also no change in the lung findings, although the *x*-ray film showed widespread patchy bronchopneumonia. Thinking that a secondary invasion of the pneumococci might be present, we instituted sulfathiazole treatment, without any effect. During all this time the patient remained perfectly lucid. However, if it became necessary to remove the oxygen tent momentarily for any reason, he would fight for air, toss to and fro, and appear frantic.

On the evening of the 19th of April, nine days after exposure, the pulse rate increased to 160, the respirations to 70, but the temperature did not rise until just before death. One hour before death he was mentally clear. During the last three days there was no chest pain, but cyanosis became more pronounced. On the morning of his death, April 19, the bedside *x*-ray revealed the entire lung field to be overshadowed by widespread patchy pneumonia, but there was no evidence of pleural fluid or solidary consolidation. On this last day scattered râles were heard within the lungs, but there was nowhere any tubular breathing. The blood count and urinalysis taken on the day of death were as follows:

Urine

Specimen	Voided
Color	Yellow
Appearance	Slightly cloudy
Specific gravity	1.020
Reaction	Acid
Albumin	Trace
Pus cells	0–1

Blood

Hemoglobin	90 per cent
Erythrocytes per cu. mm.	4,500,000
Leukocytes per cu. mm.	30,500
Polynuclear neutrophiles	88 per cent
Lymphocytes	7 per cent
Large mononuclears	2 per cent
Eosinophiles	2 per cent
Basophiles	1 per cent
Stabs	15

During the height of his illness a blood chemistry, blood Wassermann, and blood culture were made. The blood chemistry was normal; the blood Wassermann, negative; and the blood culture showed no growth. Sputum was never obtained for examination, since this patient's cough was non-productive. A dark-brown urine has been reported as present in cases of cadmium poisoning. It was not so at any time in our case.

Autopsy Report.—The body is that of a well-developed and well-nourished male of approximately the age stated on the record, twenty-seven. There are no gross deformities. There are no evidences on the external surface of the body of any injury. The muscles are all well developed. The pleural cavity contains a thin, red fluid. The parietal pleura is smooth and glistening. The lungs do not collapse when the chest is opened. The right lung weighs approximately 900 gm. It feels solid. Frothy fluid escapes from the air passages when pressure is applied, but there are regions, particularly in the lower lobe, in which a seropurulent fluid can be expressed from the smaller divisions of the bronchi. All of the small air passages appear to be distended. The cut surfaces are quite wet. The visceral pleura, particularly of the lower lobe, is red and somewhat thickened. The left lung is similar, except that in the lower lobe there are regions up to 6 mm. in diameter, which are quite solid, from which seropurulent fluid can be expressed. Edema of this side is the same as that on the right.

The heart is not enlarged. All the valves are normal. No scars are found in the myocardium, but the muscle is somewhat flabby. Both right and left coronary arteries have common origin in the root of the aorta above the aortic cusps. Both arteries are patent throughout their extent. The liver appears to be normal. The capsule is smooth and glistening, and the normal markings are present on the cut surfaces. The gallbladder contains black bile. There is no obstruction to the major bile ducts. The spleen is somewhat enlarged and soft, and the cut surfaces are a grayish red. There are no abnormalities of the pancreas. Both kidneys are normal in size. The capsules strip off easily. The cut surfaces are somewhat swollen and cloudy, but the normal markings are present. Except for slight grayish discoloration of the medulla in the lower pole, the right adrenal gland appears normal.

In the left adrenal gland, the medulla in the lower pole appears to be replaced by gray-white tissue. This is of cartilaginous firmness. This tissue replaces less than one sixth of the medulla and except for this, the gland appears to be normal. The urinary bladder contains clear, yellow urine. There are no abnormalities in the bladder. The mucosa of the stomach is intact. Some swelling and redness are present in the mucosa of the first portion of the duodenum. Except for this, there are no gross changes in the small bowel. The colon is normal. The gross appearance of the brain is normal.

From the gross examination, the cause of death in this case is bronchopneumonia, associated with marked edema of the lungs.

Microscopic Examination.—Sections of the following tissues were stained with hematoxylin and eosin:

(*a*) *Lung.*—In the sections taken from the lower lobes of the lung most of the alveoli are collapsed. There has been desquamation of the lining epithelium, and mingled with these cells there are numerous plasma cells and a few polymorphonuclear leukocytes. The interstitial tissue is thickened. The cells are swollen and in many places are separated by relatively large clear spaces. In other regions the tissue is quite compact. In the former regions there has been some infiltration with plasma cells and an occasional polymorphonuclear leukocyte. The alveoli which are open contain plasma cells, a few leukocytes, and large pale-staining round cells with centrally placed nuclei and pale-staining acellular debris. The lining epithelium of these alveoli is swollen and stains poorly.

(*b*) *Heart.*—There are no noteworthy alterations in the sections of the myocardium.

(*c*) *Liver.*—There are no alterations of the hepatic cells, and the lobular arrangement of the cells is intact. The capsule is normal.

(*d*) *Kidney.*—Except for some swelling of the tubular epithelium there are no noteworthy sections of the kidney. The glomeruli are intact, and the tubules are patent. There is no scarring.

(*e*) *Adrenal Gland.*—The sections have been taken through the lower poles of the adrenal glands. There are numerous, fairly thick strands of fibrous tissue replacing portions of the medulla. The medullary cells, however, appear normal, and there are no alterations of the cortex.

(*f*) *Duodenum.*—Sections cut through the first portion of the duodenum: The mucosa is intact and appears normal in approximately half the section. It is absent down to the submucosa, and there is some round-cell infiltration along the remaining surface. The submucosal glands are normal, and there are no alterations in the muscular layers. The blood vessels are distended.

(*g*) *Brain.*—No noteworthy alterations noted.

The manner in which this patient received his cadmium intoxication is not only interesting but instructive. The plant where this workman labored had been engaged for a number of years in reclaiming metal, chiefly lead, antimony, and tin, from discarded scrap metal. During 1940 they noted that another metal was present in the recovered substance, which upon analysis proved to be cadmium. They, therefore, devised a rotary furnace into which the scrap was thrown to be "sweated." This rotary furnace was thirty feet long and thirty inches in diameter. Within the furnace on opposite sides and extending the length of this tubular furnace were two fins which projected about four inches from the walls, the purpose of which was to tumble the scrap as the furnace revolved. It was decided that the recovery process could be made more efficient if V-shaped openings were cut at intervals in these two fins. Into this narrow, confined space went this Mexican workman without a mask, to cut with an acetylene torch the fins coated with cadmium residue. The victim did not know he was going to volatilize cadmium, and had he known he would not have appreciated its dangers. The superintendent admitted he was not aware of the toxicity of cadmium, but contended that all the workmen are instructed to wear masks whenever using an acetylene torch to cut metal. Had someone been present to enforce that ruling, this death would not have occurred.

When this patient first presented himself with a diagnosis of metal-fume fever, it was felt that the diagnosis was erroneous because of two factors, (1) the complaint of severe chest pain, and (2), the presence of extreme dyspnea. There is no acute industrial metal poison, to the writer's knowledge, which results in such distressing conditions. Just why they are present in severe cadmium poisoning is not quite clear, but the author believes it can be explained upon the basis of anoxemia. The immediate reaction to the fumes is apparently an acute inflammation of the respiratory tract so intense and so extensive that in order to lessen pain, massive splinting of the lungs occurs similar to that occurring in pleurisy, but more marked. As the diffuse, patchy bronchopneumonia spreads and edema obliterates the alveolar spaces, the available area for aeration decreases until the blood receives no further oxygen. Successive *x*-ray films of the lungs of this patient revealed this gradual diminution of available normal lung space. Likewise, the autopsy showed the distention of the first portion of the bronchial vessels which accounts for the bronchovesicular breathing heard throughout the upper chest. The plugging of the alveolar spaces, particularly in the lower two thirds of the lungs explains why râles were not prominent. Both the autopsy findings and

the microscopic study indicate a chemical, rather than an infectious, nature of the inflammation.

The death in this instance was considered occupational in origin, and the widow was awarded the full death benefits. The only other cause of death to be considered in this case was possibly that due to nitrogen dioxide (NO_2), which would still make the case a compensable one. However, cadmium was believed to be the etiological factor in this death because of the following facts:

1. It has been shown by investigators that one of the outstanding complaints in severe cadmium poisoning is that of chest pain with severe chest constriction. Our patient complained of this from the beginning, and it remained an outstanding constant symptom.
2. The autopsy findings were in keeping with those reported by others.
3. Cadmium was the metal being recovered in the process to which this patient had been exposed.

TREATMENT

Drugs for Respiratory Relief.—The less seriously affected usually experience sensations similar to an upper respiratory infection, and measures commonly used for the latter often afford relief. These include the use of acetylsalicylic acid, 10 grains (0.65 gm.), every four to six hours; capsules of acetylsalicylic acid, 5 grains (0.32 gm.), phenacetin, 2½ grains (0.160 gm.), and caffeine citrate, ½ grain (0.032 gm.), at three-hour intervals; or capsules of codeine sulfate, ¼ grain (0.016 gm.), and papaverine hydrochloride, ¼ grain (0.016 gm.), at from four- to six-hour periods. A combination of ephedrine sulfate, ⅛ grain (0.008 gm.), and amytal, ⅜ grain (0.024 gm.), at from three- to four-hour intervals, may afford relief in some. Any of numerous cough syrups containing codeine phosphate or sulfate, pantopon, or dilaudid hydrochloride in combination with expectorants, such as ammonium chloride, may be of value, *e.g.*:

Codeine sulfate	7½ grains	0.45 gm.
Ammonium chloride	4 drachms	16.00 cc.
Syrup of citric acid	1 ounce	30.00 cc.
Water to make	4 ounces	120.00 cc.

Sig.: Teaspoonful every three to four hours.

Irrigations and Gargles.—Irrigation of the throat with warm salt and soda solution (a teaspoonful each of sodium chloride and sodium bicarbonate in one pint of water), or warm glucose solution ("corn syrup" one part and water two parts) every two hours, may be under-

taken. If irrigations are not possible, then gargling either with the salt and soda solution, with 1:5000 potassium permanganate, or with five aspirin tablets crushed in a glass of water may be substituted. Neither the use of silver preparations nor the frequent vigorous swabbing of the throat with other preparations is recommended.

Inhalations.—In some instances inhalations of the following might prove soothing:

Menthol Camphor	.....	aa	15.0 gm.
Oil of eucalyptus Oil of dwarf pine needles	.....	aa	30.0 cc.

Sig.: One teaspoonful in one pint of water, steamed, and inhaled. Sedation in the form of barbiturates and intranasal drops may be needed, of from 0.25 to 0.5 per cent neosynephrin, or 0.33 per cent ephedrine in aqueous solution.

Oxygen Therapy.—Whenever a patient appears to be moderately or severely overcome by these fumes, placing him immediately within an oxygen tent is strongly recommended. This should be instituted without waiting for any signs of pneumonia for, as indicated by our fatal case, these may be absent. Furthermore, irrespective of the presence or absence of a pneumonia, oxygen therapy affords a relief for the air hunger which exists. If a specific organism is suspected to be present as a secondary invader, appropriate chemotherapy should be introduced.

Prophylaxis.—Those responsible for the health of workmen should recognize the danger of cadmium and adopt adequate means of removing fumes by exhaust systems. Workmen should wear a respirator at all times when in proximity to cadmium fumes.

REFERENCES

1. Prodan, L.: "Experimental Cadmium Poisoning," *J. Indust. Hyg.*, *14*: 174, 1932.
2. Legge, T. M.: "Cadmium Poisoning," *Ann. Report Chief Inspector of Factories for 1923*, London, 72, 1924.
3. Bulmer, F. M. R., Rothwell, H. E., and Frankish, E. R.: "Industrial Cadmium Poisoning, a Report of Fifteen Cases, Including Two Deaths," *Canad. Pub. Health J.*, *29* (January), 1938.
4. Hayhurst, E. R.: "Cadmium Poisoning," *Ohio Indust. Hygiene Bull.*, *1* (May), 1939.

CHAPTER XXIII

SELENIUM AND VANADIUM

SELENIUM

Relegated to an obscure place in the textbooks of chemistry and absent from books on toxicology, this metal is practically unknown so far as its industrial uses or hazards are concerned. Chemically, it forms the same compounds as sulfur does. For instance, with hydrogen and the more reactive metals, selenides such as H_2Se or FeSe are formed. Many organic selenium compounds have been formed in which Se replaces S. Certain areas of the country contain a soil high in selenium. In 1936 Smith[1] and his co-workers studied the possibility of toxic effects upon the rural population in Wyoming, South Dakota, and Nebraska from the ingestion of selenium-bearing foodstuffs. The most persistent investigator of this substance has been Dudley,[2] who has pointed out that this metal is at present an unrecognized hazard in the processing of selenium-bearing materials. His warning can be better appreciated when it is realized that the domestic consumption of selenium increased from 57,000 pounds in 1921 to 412,000 pounds in 1935.

Occupational Hazards:

Glass decolorizing
Production of ruby glass
Production of red and yellow glazes
Production of paint and ink pigments
Production and coloring of plastics
Manufacture of photoelectric apparatus
Alloying of machineable stainless steels
Alloying of free machining copper base alloys
Fireproofing of electric cable

Pathology.—The pathology of selenium has not, as yet, been adequately studied, although considerable animal experimentation has been done. There is, apparently, a divergence between its action upon animals and that upon humans. In animals, subacute poisoning has produced bronchial pneumonia, fatty degeneration of the liver, degeneration of the kidney cells, and other minor alterations, which clear up without residual pathology. Gastro-intestinal disturbances noted in the human have been absent in animal experimentation.

Signs and Symptoms.—Respiratory irritation, cough, edema of the lungs, vomiting, diarrhea, abdominal pain or cramps, loss of reflexes, cerebral irritation as manifested by convulsions, and death.

Acute Reactions.—Concerning the severe reactions resulting in convulsions or death, one finds these mentioned, but the author was unable to find any reference to actual death from selenium poisoning other than that of Gadamer in 1909. The garlic odor to the breath has been frequently referred to.

Chronic Reactions.—Possibly the best picture of the chronic effects from constant exposure is reported by Smith, Franke, and Westfall.[1]

A series of 111 families was studied for clinical evidence of selenium intoxication, and a series of 127 specimens of urine of as many subjects, representing 90 families, was analyzed for this element. The results of the urinary analysis showed that only 8 per cent of the cases were free, or nearly free, of selenium, while 92 per cent contained amounts varying from 2 to 133 micrograms of selenium per 100 cubic centimeters. This affords definite proof of the absorption of selenium by some of the rural population in the foregoing states. In the 111 families visited, the following disorders, exclusive of the more vague symptoms of anorexia, indigestion, general pallor and malnutrition, were observed:

1. Bad teeth, varying from marked discoloration through all stages of decay, were seen in one or more members of 48 families.
2. Yellowish discoloration of the skin, in many cases a very definite icterus, and in some cases seemingly associated with more or less definite liver disease, was seen in about 46 subjects.
3. Skin eruptions of varying degrees of severity, but not conforming to any one particular type, were seen in 20 subjects.
4. Chronic arthritis with more or less permanent changes in the joints was present in 15 subjects, varying from the milder types of rheumatoid arthritis to a more severely deforming type of arthritis deformans.
5. Diseased nails of the fingers, and in some cases also of the toes, were observed in 8 subjects. They were usually asymmetrical, atrophic, brittle, and irregular, and often presented transverse and at times, longitudinal ridging.
6. Subcutaneous edema of probably cardiorenal origin was diagnosed in 5 cases, and peripheral neuritis of doubtful etiology in 2 subjects. Fifteen subjects gave a history of more or less protracted gastro-intestinal disturbances.

While this study of Smith, Franke, and Westfall cannot be considered an observation of industrial exposure, yet it does offer the

largest group of cases yet noted in the literature, and many of the symptoms are analogous to those resulting from industrial exposure. Certain observers have noted a close similarity between selenium and tellurium poisoning. Alice Hamilton[3] in 1917 investigated selenium poisoning in copper foundries. She reports the chief symptoms were bronchial irritation and digestive disturbances.

K. Halter[4] reports the case of a man working as a mixer in a plant where colored glass was produced. After working with elementary selenium and especially with sodium selenite, used for bleaching greenish glass, he developed headaches, then irritation of the mucosa. After about two days, reddening and swelling of the uncovered skin followed. These symptoms appeared often and increased in severity.

Clinical examination disclosed edematous erythema of the face and neck, and several hard, infiltrated, ill-defined foci on the back of hands and fingers. No change appeared on skin protected by clothing. Nasal and laryngeal mucosa was reddened and somewhat thickened. Slight conjunctivitis was present. There was an enlarged liver and increase of porphyrine in the urine. Selenium was detectable in the urine. Basal metabolism increased. No nervous or gastro-intestinal symptoms (in contrast to arsenic poisoning). Inflammation of nasal mucosa and conjunctiva is the direct result of the irritating action of selenium, as is the injury to liver function.

Tests.—Selenium can be detected in the urine. The relationship has not yet been determined between the amount of selenium present in the urine and the symptoms. That is, there has not been sufficient experience with this metal to form standards of acceptance such as there has been with lead, for instance. Air also can be sampled for the presence of selenium contamination.

Medicolegal Aspects.—As yet there is no recognized toxic limit of exposure, nor has there been a sufficient number of cases studied to form any basis for the estimation of temporary disability. It is held that massive doses cause death. Possibly by the end of this decade, experience will have established a better bas-relief of this metal, as well as of tellurium, thallium, and vanadium. With few exceptions these three closely resemble selenium in regard to toxic effects.

Treatment. *Prophylaxis.*—The air breathed by the workers should be free of this substance, or its presence reduced to a minimum. Frequent sampling is necessary to accomplish this. If this substance is subjected to high temperatures, exhaust ventilation should be in operation. The skin should be protected to prevent involvement as noted by Halter. Workmen with known respiratory conditions, impaired liver function, or arthritic tendencies should be excluded from this hazard.

In chemists synthesizing organoselenium compounds, absorption through the skin is prevented by frequent changes of new gloves, protective cream, and scrupulous care in handling products. Protective devices, such as rubber and synthetic rubber gloves, give only temporary protection, since some compounds, *e.g.*, methylbenzoselenazole, readily penetrate them.

Diuretics and Cathartics.—No specific treatment for this poisoning is outlined, but if intoxication has occurred, removal from exposure with possible hospitalization is necessary. The use of diuretics to secure adequate elimination may be indicated, *e.g.*, ammonium chloride, 15 grains (1 gm.), from four to six times daily; or theophylline, 3 grains (0.195 gm.), three times daily. In this regard saline cathartics are useful, such as magnesium sulfate, from ½ to 1 ounce (16 to 32 gm.); the effervescent preparation of magnesium citrate, 12 ounces; sodium phosphate, 1 drachm (4 gm.); or the effervescent preparation of sodium phosphate, 2½ drachms (10 gm.).

Relief of Symptoms.—Upper respiratory symptoms may be treated as described under "Cadmium." Intravenous 50 per cent glucose (50 to 100 cc.) may aid in treatment of edema of the lungs, as may from ½ to 2 cc. of salyrgan intravenously at from three- to four-day intervals. If kidney degeneration is present, the use of salyrgan is probably contraindicated, although some believe that salyrgan may be used even in the presence of definite kidney pathology. Inhalations of oxygen by intranasal tube or by a tent may be needed, and intravenous infusions of 10 per cent dextrose in physiological saline (1000 to 3000 cc. daily) may be necessary if vomiting or diarrhea is present.

Diet.—The use of a high carbohydrate diet with an increased water intake has been suggested, as has the use of concentrates of the vitamin B complex. Experimental work[5, 6, 7] on animals indicated the protective action of a diet high in protein and low in carbohydrate against selenium. There was also some evidence that the ratio of selenium to the protein in the diet seems to determine the toxicity of selenium in food and that the quality of protein, as well as the quantity, was of importance. For example, casein and lactalbumin tend to counteract the toxicity of selenium while edestin and gelatin are not effective.[5, 6, 7]

The trial of similar diets in selenium poisoning in human beings has not been reported, but the use of diets high in proteins, such as casein, lactalbumin, etc., might well be worth while in treatment of these patients. In addition, when liver damage is present, the use of a high carbohydrate, high protein diet should be followed.

Also experimentally,[8] sodium arsenite, five parts arsenic per million of drinking water, completely prevented symptoms of selenium

poisoning. Clinical use of this in treatment of selenium poisoning is not reported.

REFERENCES

1. Smith, M. I., Franke, K. W., and Westfall, B. B.: "Selenium Problem in Relation to Public Health; Preliminary Survey to Determine Possibility of Selenium Intoxication in Rural Population Living on Seleniferous Soil," *Pub. Health Rep., 51:* 1496–1505 (Oct. 30), 1926.
2. Dudley, H. C.: "Selenium as Potential Hazard," *Pub. Health. Rep., 53:* 281–292 (Feb. 25), 1938; *Indust. Med., 7:* 233–236 (May), 1938.
3. Hamilton, A.: *Industrial Poisons in the United States,* The Macmillan Co., New York, 1925.
4. Abstracts: *J. Indust. Hyg. and Toxicol., 21:* 8 (Oct.), 1935.
5. Editorial: *J.A.M.A., 115:* 2001 (Dec. 7), 1940.
6. Smith, M. K.: "The Influence of Diet on the Chronic Toxicity of Selenium," *Pub. Health Rep., 54:* 1441 (Aug. 4), 1939.
7. Gortner, R. A., Jr.: "Chronic Selenium Poisoning as Influenced by Dietary Protein," *J. Nutrition, 19:* 105 (Feb. 10), 1940.
8. Maxon, A. L., and DuBois, K. P.: *J. Nutrition, 18:* 447 (Nov.), 1939, cited in editorial, *J.A.M.A., 114:* 1083 (March 23), 1940.

VANADIUM

There is not much in American literature regarding the effects from exposure to vanadium dust. Dutton[1] reported a case in 1911. He, with others, has indicated an irritating cough, gastro-intestinal symptoms, and anemia to be present following exposure. In contrast to these reports, the investigation of Symanski suggests that the affection is confined to the eye, the upper respiratory tract, and the lungs.

Vanadium Pentoxide as Toxic Product.—After calling attention to the fact that the use of vanadium has greatly increased in recent years, Symanski[2] reviews the literature on the disorders supposedly caused by it. Reviewing the complicated procedure by which vanadium is produced, he shows that of the various transformation products, it is only the vanadium pentoxide, particularly in the readily absorbable form of dust, which is harmful to the health of the workers. He subjected the workers who came in contact with this substance to a careful examination. He describes observations on nineteen men.

Signs and Symptoms.—Symanski found a number of characteristic disorders. He observed conjunctivitis with inflammatory injection of the conjunctivae, suppurating discharge, and corresponding complaints such as burning of the eyes, rhinitis with reddishness of the nasal mucosa, and coryza with watery discharge; and also a feeling of soreness in the pharynx, continuous coughing with more or less profuse expectoration, occasionally sanguineous pharyngeal secretion, a feeling of dryness in the pharynx, sensations of constriction of the chest, occasionally piercing pains, the typical aspects of a more or less severe subacute or chronic bronchitis, considerable sonorous and sibi-

lant râles in the absence of signs, indicating inflammatory infiltration of the pulmonary parenchyma; he observed no tuberculous complications, but there were roentgenological signs indicating a chronic bronchitis.

In view of the fact that all examined persons had been active in the production of vanadium for only a few years, Symanski thinks that it is necessary to consider the possibility of the development of chronic changes. Contrary to reports in the literature, he never observed reabsorptive gastro-intestinal disturbances, renal symptoms, increased nervous sensitivity, disturbances of the central nervous system, or impairment of vision. Moreover, he found no characteristic changes in the composition of the white or red blood picture, especially signs of anemia.

Treatment. *Symptomatic.*—Since but few cases have been reported, and there is no general agreement about the symptoms, treatment should be directed to the individual case of proved exposure. If Symanski is correct, symptomatic treatment would be directed at the conjunctivitis, the nasopharyngitis, and the bronchitis. Some of the detailed therapy directed at the upper respiratory symptoms as described under "Cadmium" may be followed.

Prophylaxis.—All processes in which vanadic acid is liberated in the detailed therapy directed at the upper respiratory symptoms as possible. In mixing rooms, respirators or masks should be worn, and suction apparatus should be installed. Rotation of workers should be practiced, so that they would have days or weeks in which they could recuperate from effects of contact with vanadium.

REFERENCES

1. Dutton, W. F.: "Vanadiumism," *J.A.M.A.*, *56:* 1648, 1911.
2. Symanski, J.: "Gewerbliche Vanadinschadigungen ihre Entstehung und Symptomatologie, *Arch. f. Gewerbepath. u. Gewerbehyg.*, *19:* 295–313, 1939. (As reviewed in *J.A.M.A.*, May, 1939.)

CHAPTER XXIV

ARSENIC

THE solid compounds of arsenic usually cause no serious disturbance to the body or its functions. Skin lesions in persons handling these solid compounds are fairly common. On the other hand, exposure to the gaseous form of arsenic (arsine or arseniuretted hydrogen) is dangerous. Arsine has a strong hemolytic action, but the destruction to the red cells is not immediate, there being an interim of from several hours to a day or two.

Arsine is never intentionally used or produced in industry. There is always the chance, however, that it may escape in certain processes. Wherever nascent hydrogen is used in a reduction process, as in the making of aniline dyes or coal-tar products, there is a possibility that arsine may be formed. Another source of danger is in the cleaning out of tank cars which have contained sulfuric acid.

Occupational Hazards.—Among the following workers, arsenic affects the skin chiefly:

Arsenic-roasters
Artificial-flower makers
Artificial-leather makers
Bookbinders
Brass-founders
Briquet-makers
Bronzers
Calico-printers
Carpet-makers
Carroters (felt hats)
Chargers (zinc-smelting)
Colored-paper workers
Color-makers
Compounders (rubber)
Copper-founders
Copper-smelters
Curriers (tannery)
Cut-glass workers
Decorators (pottery)
Dye-makers
Electroplaters
Enamelers
Enamel-makers
Farmers and Gardeners
Feather workers
Felt-hat workers
Ferrosilicon workers
Picklers

Pathology.—Pathology consists chiefly of a local irritation causing lesions to the skin, mucous membranes, and appendages. Acute poisoning, as in suicide, murder, or accident by ingestion, will cause irritation of the stomach as well as degenerative changes of the heart, liver, and kidneys. Chronic absorption may lead to changes in the nerve and muscle fibers.

Diagnosis. *Signs and Symptoms.*—Headache, eruptions, and ulcerations of the skin, perforation of the nasal septum, loss of nails and hair, inflammation of the mucous membranes, nausea, vomiting, diarrhea, and abdominal pains, peripheral neuritis, muscular weakness, and paralysis. Workers with paris-green have complained of an irritation of the larynx and bronchial tubes. Many of these exhibit a cough or husky voice, which quickly disappears upon withdrawal from exposure.

Differential Diagnosis.—Arsenic poisoning must be differentiated from botulism, mercurialism, and from certain poisons such as carbolic acid. In the latter, the burns on the lips or mouth and the carbolic odor are characteristic. Mercurialism is differentiated by the salivation and pains in the mouth. Botulism is confined mostly to a gastro-enteritis of a day or two's duration.

Tests.—Arsenic may be found in the urine, feces, hair, and nails. In chronic arsenic poisoning, it remains for a long time in the hair and nails. It has been found in the hair of an employee two years after cessation of his employment. This substance may be recovered from the bones and other organs after death.

Medicolegal Aspects. *Temporary Disability.*—This varies, but is usually short. Skin lesions may be resistant to treatment, resulting in a temporary disability of several months. Gastro-intestinal symptoms clear up shortly after exposure has ceased.

Permanent Disability.—When this occurs, it is usually from a peripheral-nerve involvement. If it is present after a two-year period, the functional loss should be estimated, as in any other partial permanent disability.

Illustrative Cases:

Case I.—A fifty-seven-year-old white male, single, had been unemployed since November, 1935. In June, 1936, he presented himself with a disabling condition which he attributed to chronic arsenic poisoning from his last employment, that of dye-making.

Examination.—Weight 210 pounds. Florid complexion, red bulbous nose. Huge barrel-shaped chest with râles at both bases. Markedly dyspneic. Heart sounds distant and barely audible. No murmurs elicited. Blood pressure, 200/120. Abdomen obese with possible fluid present. The liver was palpable and down to the level of the umbilicus. Pitting edema of both legs. Urine: Albumin, two plus, with occasional casts. Blood count not significant. Blood Wassermann negative. Nails and hair negative for arsenic.

It was freely admitted by the patient that he had drunk heavily all his life. During the past ten years he had consumed nearly a pint

of whisky a day, with frequent week-end bouts. During the past year he had become a pronounced drunkard. His occupational history revealed that prior to coming to California in 1925, he had been a section hand on a railroad in Illinois. From 1925 to 1932 he was a longshoreman. He was unemployed during the greater part of the time from 1932 until May of 1935. At this time he obtained employment in a dye-manufacturing concern. He worked there for nearly seven months, when he was discharged. While there a young employee alleged that his father had died of arsenic poisoning from working in dyes. Therefore, when this patient became ill, he claimed arsenic as the cause. He appeared honest in his belief. However, his "job" was that of a trucker, conveying finished material to the shipping platform.

The details of the case have been given to show how often an obviously non-compensable case must be carefully investigated.

Diagnosis.—Cardiovascular renal disease, with hypertension. Cirrhosis of the liver (alcoholic?). The case was not compensable.

Case II.—A young Chinese laborer employed in spraying vegetables became weak, lost some weight, and had abdominal pains. He consulted a physician in a nearby town, who diagnosed the case as arsenic poisoning. He was removed from his work, but the symptoms continued. Four weeks later a stool examination revealed numerous eggs of the tapeworm, which was then recovered by administering male fern.

No comment is needed.

Treatment. *General Measures.*—As with mercury, phenol, and others, the cases of acute poisoning from arsenic will be rare in industry. When they do occur, the symptoms of vomiting, painful diarrhea, nervousness, thirst, cyanosis, and circulatory collapse must be treated. Early abundant gastric lavage with warm water is indicated. This is followed by the administration of warm milk. Previously lavage of the stomach with colloidal ferric hydroxide suspension, prepared by adding magnesium oxide to tincture of ferric chloride or a solution of ferric sulfate, was advised. This is now considered by most to be obsolete, but as McNally points out, it can do no harm and may delay absorption while the stomach is repeatedly washed out.

Relief of Pain and Diarrhea.—Morphine sulfate, $\frac{1}{4}$ grain (0.016 gm.), every four to six hours, may be needed for relief of the pain and may aid in controlling the diarrhea. Other drugs which may be used for relief of the diarrhea and abdominal pain include bismuth subcarbonate or subnitrate, 15 grains (1 gm.), every four to six hours; tincture of opium, from 10 to 15 drops, every four to six hours; or camphorated tincture of opium (paregoric), two drachms

(8 cc.), every three to four hours. Approximately two hours prior to starting these latter drugs, it is advisable to give a saline cathartic in full dose.

For the diarrhea the drugs mentioned above, *e.g.*, tincture of opium, bismuth subcarbonate or subnitrate, or paregoric, may be used. Porter[2] has reported prompt improvement of the diarrhea and neuritis by the use of 2.6 mg. of thiamine hydrochloride daily, but if this vitamin is used, larger dosages, from 50 to 60 mg. per day parenterally, are indicated. The use of nicotinic acid, from 100 to 200 mg. daily, would possibly be of value in treatment of the diarrhea.

Cyanosis, Circulatory Collapse, and Shock.—For cyanosis, oxygen inhalations are given, and circulatory collapse and shock are treated as described in other sections, *e.g.*, "Mercury" and "Fluorine." The intravenous infusion of from 5 to 10 per cent dextrose in 1000 cc. physiological saline is indicated not only for treatment of the shock syndrome, but also for the dehydration and chloride loss which may follow prolonged vomiting and diarrhea. The amount of this intravenous therapy needed will be determined by the patient's course.

Sodium Thiosulfate.—The use of sodium thiosulfate intravenously in both the acute and chronic cases is advised. There has been considerable controversy concerning the use of this drug, but clinically it appears to be of value. Ayres and Anderson[1] demonstrated an increase in the excretion of arsenic in the urine following the injection of sodium thiosulfate in 80 per cent of forty-nine cases. The amount to be used may vary from 15 grains (1 gm.) in 10 cc. of sterile distilled water every four to six hours during the first twenty-four hours of the acute stage to a similar injection two or three times a week over a period of weeks in the chronic case.

Skin Reactions.—The ulcerations, pigmentation, and varied forms of a scaling dermatitis which may be present in the chronically exposed usually disappear after removal from exposure. The following prescriptions may be of value for the dermatitis:

I.	Salicylic acid	1.5 gm.
	Sulfur (ppt.)	1.5 gm.
	Lanolin	6.0 gm.
	Ung. aqua rosae q.s.	30.0 gm.
II.	Salicylic acid	6.0 gm.
	Menthol	1.2 cc.
	Lard q.s. ad.	60.0 gm.

Fluids should be forced, and some of the drinking-water should be normal saline. Milk and a high caloric, high vitamin diet are of general value.

Peripheral Neuritis.—Vilter, Aring, and Spies[3] report striking improvement in a case of arsenic peripheral neuritis, in which 20 mg. of synthetic vitamin B_6 in sterile physiological solution of sodium chloride was given twice daily. Improvement was more marked when 50 mg. of alpha-tocopherol was given intramuscularly in conjunction with the vitamin B_6; 50 mg. of thiamine hydrochloride intravenously for three weeks had previously failed to influence the condition. Physiotherapy may also improve the neuritis. Anemia, if present, should be treated by ferrous sulfate, 5 grains (0.32 gm.), two tablets three times daily.

Arsine Poisoning.—No specific treatment is available for arsine poisoning. Repeated transfusions are of value, and the administration of iron compounds orally is indicated. Oxygen inhalations will be needed in many cases. The remainder of the treatment should consist of an adequate fluid intake, supplemented by frequent intravenous infusions of from 5 to 10 per cent glucose in physiological saline. The administration, in the early morning, of saturated solutions of magnesium sulfate, from ½ to 2 ounces (15 to 60 cc.), flavored with a small amount of compound tincture of cardamom, may help in encouraging flow of the thick bile plugging the biliary passages; this, however, is doubtful. A similar attempt might be made by introduction of from 40 to 50 cc. of 33 per cent magnesium sulfate through a duodenal tube (Lyon non-surgical drainage). The stimulants frequently mentioned in other chapters, *e.g.*, "Mercury," are to be used when symptoms of circulatory or respiratory failure appear.

REFERENCES

1. Ayres, S., and Anderson, N. P.: "Sodium Thiosulphate and the Elimination of Arsenic," *J.A.M.A., 110:* 886–887 (March 19), 1938.
2. Porter, W. A.: "Acute Arsenic Poisoning," *Virginia M. Monthly, 66:* 148 (March), 1939.
3. Vilter, R. W., Aring, C. D., and Spies, T. D.: "A Case of Arsenic Peripheral Neuritis Treated with Synthetic B_6 and Alpha-Tocopherol," *J.A.M.A., 115:* 208 (July 20), 1940.

CHAPTER XXV

ZINC, ANTIMONY, COPPER, AND TIN

ZINC

THE acute intoxication in which zinc plays a part has been described under "Metal-Fume Fever."

Chronic Poisoning.—Chronic zinc poisoning is doubted by most investigators. In the older textbooks and literature, instances of chronic zinc poisoning were recorded. It is now felt that other metals present in the process in which zinc is used provide the intoxication. In 1926, McCord and his co-workers[1] studied the employees in two galvanizing plants. In one plant, where the working conditions were poor and the men had been engaged in this work for a long period of time, they exhibited gastric disturbances varying from gastro-enteritis to gastric and duodenal ulcers. In the second plant, where the hygienic conditions were fairly good and the men had a short term of exposure (less than six years), no gastro-intestinal disease was found. They concluded that a long period of time expressed in years (five to twenty), plus poor working conditions and a poor type of worker, favored the production of chronic zinc poisoning. In this same year, Drinker[2] and her colleagues found no acute or chronic illness attributable to zinc in twenty-four workmen exposed from two to thirty-five years. Writers in the recent literature are of this same opinion, although duBray[3] reported a case in 1938, believed by him to be due to chronic zinc intoxication.

Zinc is found in various human tissues and the excreta. Some zinc is found in practically all food. In industry, zinc is involved in the process of mining, smelting, manufacturing of brass and other alloys, galvanizing, and in many other less important procedures.

Anyone alleging illness due to zinc warrants thorough study for causes not industrial in origin, as well as careful inquiry into the process to which he is exposed in order to determine the presence of other metals.

Medicolegal Aspects.—An allowable concentration is 14 mg. per cubic meter. Temporary disability is short, a few days at the most. There is no permanent disability.

Treatment. *Prophylaxis.*—The preventive measures are the same as those to be adopted with a lead hazard, or any dust hazard apt to

contain lead. Adequate ventilation, avoidance of the deposits of dust, frequent cleaning of all parts of machinery, floors, and walls of dust collections are factors in reducing exposure. Men should be selected who are free from focal infection. Some authors advise periodic changes of work and comparatively short working hours. The inclusion of adequate fat and milk in the diet is thought to prevent to some degree the occurrence of symptoms in those working with zinc.

If a case of *chronic zinc poisoning* has been established, the patient should be removed from his hazard at once. Anemia, if present, is treated by ferrous sulfate, 5 grains (0.32 gm.), two tablets three times a day. In *acute intoxication*, a few days' absence from the work and treatment as described under "Metal-Fume Fever" will suffice.

Zinc Chloride Burns.—Treatment of zinc chloride burns consists of removal of the necrotic slough and of filling the wound with sodium bicarbonate. Compresses of a warm solution of sodium bicarbonate are then used. After the lesion has cleaned up to some extent, it is dressed with borated petrolatum, painted with merthiolate, or covered with metaphen in collodion. Gloves should be worn by workers handling flux containing zinc chloride. When zinc chloride powder is handled, ordinary washing with soap and water will not remove it as a rule, while a 5 per cent solution of hydrochloric acid will.[3]

REFERENCES

1. McCord, C. P., and Friedlander, A.: "Occupational Syndrome among Workers in Zinc," *Am. J. Pub. Health, 16:* 274–280 (March), 1926.
2. Batchelor, R. P., and others: "Clinical and Laboratory Investigation of Effect of Metallic Zinc Oxide and of Zinc Sulphide upon Health of Workmen," *J. Indust. Hyg., 8:* 322–363 (August), 1926.
3. duBray, E. S.: "Chronic Zinc Intoxication; Instance of Chronic Zinc Poisoning from Zinc Chloride Used in Pillow Manufacturing Industry," *J.A.M.A., 108:* 383–385 (Jan. 30), 1937.

ANTIMONY, COPPER, AND TIN

These three metals are considered jointly because they represent a group about which little is known. This is especially true of copper and tin, whereas more is being learned regarding the toxicity of antimony and its compounds.

Tin.—Tin is rarely ever used in a form that could cause any symptoms. A transient irritation of the nose and eyes has been noted from the use of tin tetrachloride in silk mills.

Copper.—Copper oxide is frequently present with other metals in processes which cause metal fume fever. Following Mallory's contention that bronze diabetes, characterized by cirrhosis of the liver, diabetes mellitus, and pigmentation of the skin resulted from chronic

copper poisoning, the British investigators made a careful survey regarding the incidence of such a syndrome among copper workers. They found no evidence that such a disease occurred among copper workers.

Antimony.—While antimony has received slight consideration by the toxicologist, it is likely to be subjected to more detailed study, since it is now being more widely used in certain national-defense projects.

Occupational Hazards.—The usual exposure to antimony occurs where the ore is being mined, smelted, or refined; and also in processes where it is combined as an alloy with other metals, especially lead, tin, and copper. These alloys find use in storage-battery grids, pewters, metal bearings, and type metal. In the defense program it is used especially in the manufacturing of munitions.

Pathology.—Most of the references regarding the effects of antimony occur in the foreign literature, and a great deal of it is of an experimental nature. A very recent summary of the literature can be found in an excellent paper by Bradley and Fredrick.[1] These two workers quote the report of Schrumpf and Zabel, whose observation of workers in a type foundry revealed very few cases of lead poisoning, but a number of men with "remarkable facial expression, complaints of nervousness, irritability, sleeplessness, fatigue, dizziness, muscular and neuralgic pains, loss of appetite, nausea, gastro-intestinal disturbance, and constipation." Examination of the blood revealed a diminished leukocyte count and a notable eosinophilia. The blood pressure was lowered. Antimony was recovered in the stools.

Experimentally the work of Bradley and Fredrick indicates that the most important pathological effect was the consistent *injury to the heart muscle.* They could draw no conclusions from their hematological studies.

The author has never observed a case of intoxication by antimony, although his experience with the metals has been fairly comprehensive. It is possible that antimony has been an unrecognized factor of illness in certain cases thought to be lead poisoning. Obviously, but few industrial processes use antimony except in combination with other metals. A true estimation of its harmful effects upon the human body will have to await the compilation of more clinical data. The experimental investigation strongly suggests strict hygienic measures in those industries using this metal.

REFERENCE

1. Bradley, Wm. T., and Fredrick, Wm. G.: "The Toxicity of Antimony—Animal Studies." *Indust. Med., 10* (April), 1941.

PART IV

THE DUSTS

Recognition of the irritating effects from inhaled dust is not recent. "Fatal dust" was mentioned in the writings of Pliny; and Celsus in the first century described "dust phthisis." Studies of silicosis were conducted in England, Germany, Italy, South Africa, and Australia many years ago, yet it was not until 1915 that the first real investigation (by Lanza and Higgins) was conducted in this country. Since then much time has been devoted to the dust diseases, especially silicosis, by the hygienist and pathologist, and volumes have been written concerning them.

Confusion about Effects.—In the early years of the depression, the physician and the layman were awakened to a disease called "silicosis." Unscrupulous lawyers grabbed the chance to obtain fees from idle workmen willing to allege a disability to obtain remuneration. Judges and juries were shown *x*-ray films with "spots" on them. Untaught physicians testified that such films could represent silicosis. Large awards were made to fraudulent persons. It has been estimated that the cost of these to industry was in excess of $100,000,000. The silicosis racket has abated, but it is not extinct, nor will it be so long as confusion remains in the minds of the court, industrial commission, or the practicing physician, as to the interpretation of the word "harmful." What dusts are harmful? what factors constitute a harmful exposure? and when is the worker to be considered harmed or disabled? These questions have been answered repeatedly in the literature, but possibly they have been obscured by scientific detail. It is hoped that those seeking a ready basis for diagnosis and especially a basis for the determination of compensability will find the following discussion adequate.

Distinction between Dusts.—The distinction between gases, fumes, and dusts in an industrial atmosphere is a very fine one. It has been indicated in the chapter on metals that the body is affected by the inhalation of finely divided dust particles of lead, zinc, mercury, cadmium, manganese, etc. These metallic dusts produce a toxic reaction which is usually acute and temporary; whereas the dusts to be discussed in the succeeding chapters have a specific action upon the lung tissue, are insidious rather than acute, and their damage is permanent. This statement excludes those dusts which may affect the upper respiratory tract but which produce no pathological change in the

lung tissue. Frequently a patient comes to the clinic with a diagnosis of silicosis because he has been in an atmosphere of dust, just any dust, without any effort having been expended to determine if silica or the silicates existed in the exposed atmosphere.

Organic and Inorganic Dusts.—The first act to be considered by the physician is the type of dust in the occupation. For this purpose the dusts may be divided into two groups: (1) the organic, and (2) the inorganic. Organic dusts do not cause pulmonary lesions, and, most important of all, very few inorganic dusts can be definitely considered to be offending agents.

Definition of "Pneumoconiosis."—Pneumoconiosis means a chronic pulmonary fibrosis due to the inhalation of irritating dusts which produce a proliferative reaction. The term is comprehensive and too often misleading. Investigators in dust diseases use the term correctly, but the average physician is prone to assume that it implies a disabling condition. Since silicosis is a condition of pneumoconiosis, the implications of this disease are apt to be applied to any dusty exposure. In addition to silicosis, the term "pneumoconiosis" includes asbestosis, anthracosis, aluminosis, siderosis, chalicosis, byssinosis, tabacosis, and other forms not fully understood.

Silica versus Silicates.—Confusion also exists regarding the use of the word "silica." I have heard physicians, attorneys, and referees discussing the possible presence of silicosis in a claimant who had been exposed to the silicates without appreciating that there is a world of difference between silica and the silicates. Because that difference is so important, the author feels it expedient to present an elementary review of the chemistry of silica and the silicates.

Chemistry.—The chemical individuals that go to make up the igneous rocks, resulting from originally molten material by a process of crystallization, are called "primary minerals." When these primary minerals have been altered by heat, pressure, or water, secondary minerals result. Silica is silicon dioxide, SiO_2. The most important crystalline form of silica is quartz. Next to feldspar, it is the most abundant of all minerals. Minute aquatic organisms such as diatoms, infusoria, and certain varieties of sponges develop siliceous skeletons which, following death, accumulate on the bottom of the ocean. This substance is known as infusorial or diatomaceous earth.

Silica, SiO_2, is composed of silicate tetrahedra, SiO_4^{----}, a structural unit which was first revealed by *x*-ray. In studying the internal structure of crystals, among other things it was noted that separate SiO_4^{----} tetrahedra alternate in the space lattice with positively charged metallic ions in certain silicates, while others are linked together in pairs, with an oxygen ion in common. "Since each oxygen ion is shared between two different silicate tetrahedra, there are only

half as many oxygen ions in the complete solid structure as the formula SiO_4 would seem to suggest; in other words, the formula of the solid structure, extended indefinitely in three dimensions of space, is a triple infinite multiple of SiO_2."[1] However, the chemistry of the silicates is not so simple as the foregoing would indicate, owing to the arrangement of the oxygen ions in the structure, for some groups possess oxygen ions which are not linked with other groups, while two ions may be shared with other groups. A chain, for instance, of infinite multiples of SiO_3 may be formed. The fibrous structure of asbestos is due to the linking of silicate groups in continuous chains.

Silicates in Industry.—The silicates of industrial importance are too numerous to consider at this time, but a few deserve mention. The talc used in cosmetics is hydrated magnesium silicate. Closely related to talc is soapstone. Hydrated potassium-aluminum silicate is known as mica and is chiefly of use in electrical condensers. Waterglass has many uses and is formed artificially by fusing sodium or potassium carbonate with sand. When added to water in its molten state a viscous colloidal dispersion takes place, and it is in this form that it is most widely used. Various heavy metals may be added to silica to form colored silicates, such as cobalt, copper, chromium, manganese, or the like. Carborundum (silicon carbide) results from adding carbon (coke) to silica at high temperature.

Silica Locked in Silicates.—The point in the foregoing discussion is this: The silicates are abundant in industry, whereas pure silica is less so. In the silicates, the silica is bound by the chemical structure so that it is not released to act as a harmful agent to the lungs. Therefore, in dealing with all persons presenting a claim involving silicosis, one should first differentiate between a hazard in which silica as such is present and one in which the silica is *locked* within the chemical structure of the silicates and is, therefore, inert. This fact must be kept in mind when one reads a chemical analysis of a substance which shows the presence of a high content of silicates, but does not reveal the actual content of the silica as such in the mineral. In fact, a chemical analysis of a material is not satisfactory for the determination of the exact percentage of silica present.

Estimating Silica Content.—When a question arises about the amount of silica present in a substance alleged to be an offending agent, it might be better to resort to the *x*-ray diffraction method as discussed in Case XIII under "Silicosis." As a final exhortation prior to the consideration of silicosis, the author warns all interested parties that the silicates should be differentiated from silica in a claim of this type.

REFERENCE

1. Deming, G. H.: *General Chemistry*, Wylie and Sons, Inc., New York, 1935, p. 482.

CHAPTER XXVI

SILICOSIS

Definition.—The Committee on Pneumoconiosis of the American Public Health Association defined silicosis as a ". . . disease due to breathing air containing silica (SiO_2), characterized anatomically by generalized fibrotic changes and the development of miliary nodules in both lungs, and clinically by shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis (some or all of which symptoms may be present) and characteristic *x*-ray findings."[1]

Factors in Development of Silicosis.—Four important factors are necessary:

Size of Dust Particle.—If dust is to exert a harmful effect upon the lung tissue, it must be small enough to enter the smallest divisions of the lung. Only particles of less than 10 μ are capable of getting into the lungs. Seventy per cent of the dust in industry measures between 0.5 and 3 μ. Larger particles settle out of an atmosphere more quickly than the smaller ones. A particle of 5 μ will fall, according to measurement, twenty times as fast in one hour as a particle of from 1 to 3 μ. Therefore, an atmosphere which appears to the workman as being too dusty because he can see the dust is apt to be less dangerous than one which appears free of the particles.

Number of Dust Particles per Cubic Foot of Air in Breathing Zone.—An allowable concentration is 10,000,000 particles of a size between 0.5 and 5 μ per cubic foot of air.

Mineral Composition of Dust Breathed.—The content of the dust should determine the standard fixed for allowable concentration. If a standard of 15,000,000 particles is the limit for a dust containing 35 per cent free silica, then a mineral dust containing 70 per cent free silica should be limited to a standard of 7,500,000 particles per cubic foot of air. Concerning the composition of the dust breathed, there exists controversial opinion over the action of silica combined with other substances. Some hold that the action of silica is aggravated by the presence of an alkali, while others maintain it is retarded.

McCord[2] and his co-workers concluded that ". . . the often encountered assertion that co-existence of silica and alkali dust in industry constitutes extra hazardous working conditions, is unproved but not necessarily unfounded."

Denny, Robson, and Irwin[3] contend that if metallic aluminum (hydrated alumina) is dispersed in an atmosphere containing silica, silicosis will be prevented.

Length of Exposure.—No set time can be given as an estimate for development of silicosis. The older literature indicated that ten years or longer was the average. In the more severe exposure, silicosis may develop in from three to five years. Cases resulting from an exposure of less than one year have been reported. Such instances are extremely rare and certainly must involve unusual factors.

Occupational Hazards:

Mining of hard siliceous rock (copper, gold, silver, zinc, iron, and hard coal)
Quarrying of granite, sandstone, quartz, slate rock, and crystal
Pottery-making. Ceramic industries
Glass-manufacturing
Working with talc and abrasive material (sand, sandstone, sandpaper, scouring material, and soaps)
Brick-manufacturing where silica is used
Stone-finishing
Construction work (tubes, tunnels, aqueducts, etc.)
Industries where spraying occurs

DIAGNOSIS

Signs and Symptoms.—Climate, season, soil, heredity, race, age, and sex seem to have no effect upon the development of silicosis. But individual susceptibility varies a great deal, some patients showing marked pathological changes after a comparatively short exposure.

Nodulation and Fibrosis in Absence of Symptoms.—The initial effect of a collection of silicotic nodules within the lung is attended by no symptoms. Many persons may develop what appears to be intense nodulation and fibrosis without evidencing any signs or symptoms during their life. Râles will not be heard, the chest will usually be barrel-shaped, and inspection will generally show some bilateral limitation of excursion. Otherwise these persons appear normal. Even respiratory diseases of another nature, such as pneumonia or bronchitis, may be overcome without aggravating silicosis.

Progressive Symptoms.—If the patient does show symptoms, the first is that of shortness of breath upon minor effort. This increases in severity, and palpitation of the heart may be noted. At about this time, a dry, infrequent cough may ensue, which as time goes on becomes moist and frequent. The sputum is usually scant, stringy, and sometimes discolored. Fever is conspicuously absent. Unaware of his

true condition, the patient may request lighter work. Further progression may include pleurisy pains, epigastric pains, loss of appetite, and marked fatigue. As dyspnea increases, cyanosis appears. Progressive heart failure may continue until death, but these cases are rare. It is usually at this late stage that tuberculosis enters the picture. This condition, silicotuberculosis, will be described later.

Symptoms in Acute Silicosis.—This is an extremely rare form and results when enormous amounts of fine silica dust become imbedded in the lungs. Within from a few months to a year symptoms may appear. These may even precede conclusive *x*-ray findings. Acute and frequently fatal pneumonia often complicates or terminates this type of silicosis.

Tests.—The important evidence of pneumoconiosis is obtained by *x*-ray. The advent of tuberculosis may be proved by the finding of the tubercle bacilli in the sputum. In the absence of the tubercle bacilli, early suspicion may be strengthened by an increased blood sedimentation rate.

Pathology. *Elimination Mechanisms.*—That only a small portion of the dust inhaled ever reaches the lung is a fact not realized by the layman and frequently forgotten by the physician. Most of it is expelled before it reaches the lung by the upward current created by the ciliated epithelium. Further elimination occurs by phagocytosis, the particles being carried back to the ciliated epithelium or to the point where the cough mechanism becomes effective. Inert dusts which pass these barriers usually disintegrate and become short-lived. Substances, such as coal dust, which actually enter the lung tissue may cause pigmentation but no pulmonary destruction.

Fibrosis at Hilus of Lung.—The pathology of silicosis is one involving the lymphatic drainage of the lung. The normal lymphatic drainage is toward the hilum. A small area beneath the visceral pleura is drained into the subpleural cistern and from there along the lobar septa to the hilum. Dust which reaches the wall of an alveolus is picked up by the phagocytic cells, and by ameboid movement they pass into the lymph spaces. From here they slowly move on to the minute lymph islands which guard the entrance to the small lymphatic vessels, pass through these and are finally picked up by the lymph glands at the hilus of the lung. It is believed that at this point the dust cells, acted upon by the alkaline body fluids, form a colloidal silica hydroxide which is a strong protoplasmic poison, and its action causes death to the dust cell and necrosis to the immediate surrounding tissue. Scar (fibrous) tissue is formed, which acts as a blockage to the lymphatic channels, and the removal of further inhaled dust becomes impossible.

Spread of Fibrosis.—Additional fibrous tissue forms along the lymphatic vessels which accompany the blood vessels of the lung, invades the septa between the lobes and spreads into the lung tissue itself. The minute masses of lymphatic tissue become fibrotic nodules (Fig. 47), and these, as time goes on, increase in number to block off large portions of the lung. This blocking of the lymph flow toward the hilus increases the spread of dust cells toward the pleura. These minute nodules of fibrotic material are thus scattered throughout the field and are surrounded by apparently normal lung tissue.

Fig. 47.—Simple discrete nodulation of uncomplicated silicosis. (Gardner.)

Alveolar Consolidation.—As the process proceeds they tend to coalesce to form massive areas of fibrosis (Figs. 48, 49, 50). This increase of fibrosis tends to destroy the alveoli and thus provokes enlargement of the surrounding alveoli. Such enlargement constitutes emphysema. With the thickening of the alveoli, the elasticity of the lung is interfered with, and aeration decreases. If compensatory emphysema is adequate and infection is not present, the condition is that of simple silicosis. But if compensation is not adequate, the first symptom of silicosis appears, namely dyspnea. However, since only one fourth of one lung is necessary for life, fibrosis must be enormous before death can occur from deficient aeration alone.

Production of Dyspnea.—That encroachment upon the normal

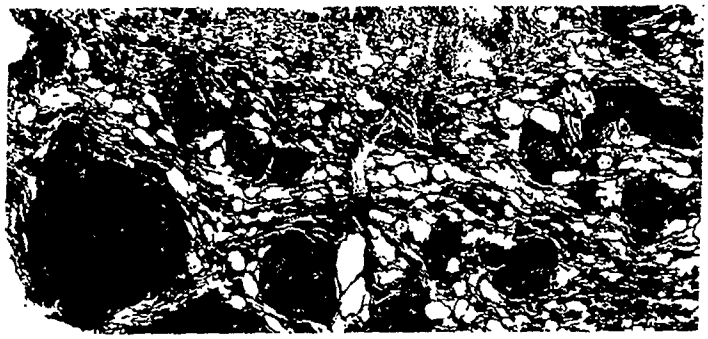


Fig. 48.—Massive areas of confluent nodules. Note the thickened pleura. (U. S. Public Health Service, *Bulletin 244.*)

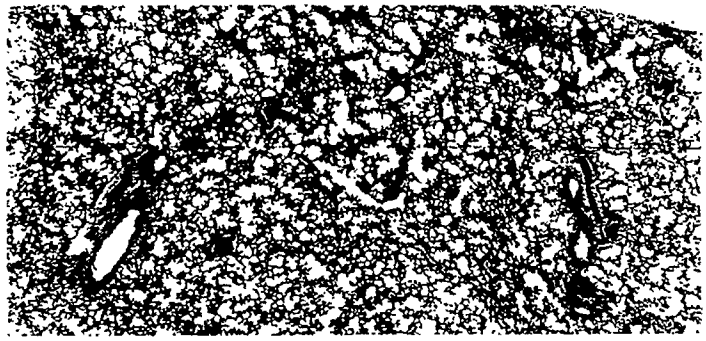


Fig. 49.—Confluent mass of silicotic nodules adjacent to small bronchus and blood vessels. Note the presence of extensive emphysema in patent lung tissue. (U. S. Public Health Service, *Bulletin 244.*)

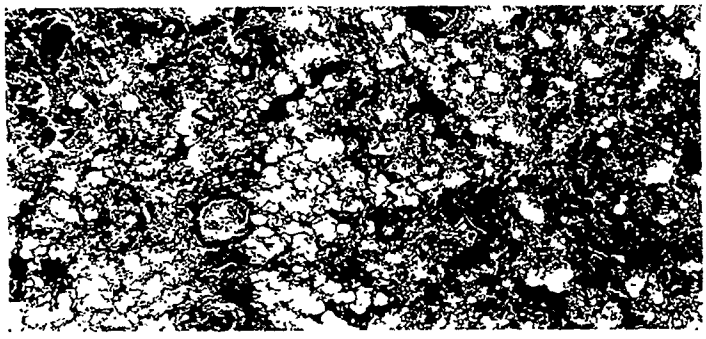


Fig. 50.—Section through lung, showing massive areas of confluent, fibrous nodules. Close inspection will reveal the extensive emphysema. (U. S. Public Health Service, *Bulletin 244.*)

lung fields by nodulation is the cause of dyspnea is questioned by Cole and Cole[1]:

It is universally conceded that small, hard, dense nodules in the lung are pathognomonic of silicosis, yet these small, hard, dense, nodules are either absent or relatively infrequent in some cases of advanced silicosis in which dyspnea is an outstanding symptom. These silicotic nodules as observed roentgenographically often progress to an extreme stage before they cause dyspnea, yet dyspnea may occur and terminate fatally without a preponderance of the typical well defined nodules. Spherical whorls or nodules are considered pathognomonic of silicosis, yet we believe they constitute a protective mechanism—a constructive rather than a destructive process—and are not responsible for the outstanding symptom of silicosis, dyspnea.

Advanced silicosis is frequently found only by accident, when the patient is being examined roentgenologically for some other lesion or injury, such as a fractured rib. Such accidental discoveries are frequent, but, on the other hand, roentgenograms may show no evidence of typical nodulation even in a case of advanced silicosis.

They are convinced, after making an intensive study of the pathological changes of acute silicosis, that "dyspnea is caused by morbid changes in the blood and endothelial reticular structures, which in turn result in changes in blood cells, observed in localized regions of the lung." They conclude that large microscopic sections of silicotic lungs show three general manifestations of morbid change: (1) avascular areas, (2) overvascular areas, and (3) a region of invasion.

The region of invasion, where collagen constricts the capillaries, is the most important region for intensive study. In the avascular portion, capillary occlusion by external pressure causes a traffic jam of red blood cells, with a damming back of the blood into the arterioles and a lack of drainage of the veins. The blood in the larger vessels in the avascular areas disintegrates. Overvascular areas, where there is immense dilatation of the capillaries and engorgement of the veins and arteries with viable red blood cells, seem to compensate for avascular regions in different portions of the section.

Dyspnea has been explained by two pathological factors: (1) nodulation, and (2) alveolar consolidation. To these Cole and Cole add a third; namely, capillary obstruction and dilatation.

From a study of microscopic sections they have made certain deductions which constitute their conception of the life history of silicosis at least as it occurs in some groups of cases. They believe that the dyspnea of silicosis is due to capillary occlusion in large localized regions of the lung, with compensating capillary dilatation in other regions.

Variety of Morbid Changes.—Most diseases exhibit morbid changes which can be recognized in relatively small sections; for example, certain types of neoplasm can be determined by small groups of characteristic cells; but in silicosis there is a vast variety of morbid changes in various portions of the lung. Different varieties of dust result in different types of morbid change in varying regions of the lung.

TABLE 13—A TABULATION OF THE ROENTGENOLOGICAL CHANGES FOUND IN SILICOSIS TOGETHER WITH THE UNDERLYING HISTOLOGICAL CONDITIONS[3]

HEALTHY LUNGS AND ADNEXA

Roentgenological Appearances	*Histological Appearances*
1. *Healthy lungs*, as defined by the N.T.A. Committee Report (National Tuberculosis Association)	1. Essentially the normal tissues of the vascular tree, the mediastinum, the bronchi, and trachea
2. Irregular exaggeration of the linear markings, with possibly some beading, confined to the trunks	2. Cellular connective tissue proliferation about lymphatic trunks in the walls of vessels and bronchi. Beading may be due to various causes, such as blood vessels seen end on, arteriosclerosis, minute areas of fibrosis in lymphoid tissues along the trunks
3. Increased root shadow	3. Cellular reaction in the tracheobronchial lymph nodes with extensions along afferent lymphatic trunks

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

SIMPLE SILICOSIS

Roentgenological Appearances	*Histological Appearances*
4. *Nodulation.* Discrete shadows not exceeding 6 mm. in diameter tending to uniformity in size, density, and bilateral distribution, with well-defined borders surrounded by apparently normal lung shadow. The outer and lower lung fields characteristically show fewer nodules	4. Circumscribed nodules of hyaline fibrosis located in the parenchyma of the lung. Occasionally some of these nodules may show microscopic foci of central necrosis
5. *Conglomerate shadows* that appear to result from a combination or consolidation of nodulation usually with associated emphysema manifested by: (*a*) Localized increased transparency of the lung with loss of fine detail (*b*) Intensification of the trunk shadows by contrast (*c*) Depression of the domes with possible tendency toward individualization of the costal components of the diaphragm (*d*) Lateral view. Increase in the pre-aortic and retrocardiac spaces with exaggerated forward bowing of the sternum and backward curving of the spine. Widening of the spaces between the ribs may or may not be present (Fig. 31)	5. The result of coalescence of discrete nodules; an area in which the nodules are closely packed and most of the intervening lung is replaced by more or less hyaline fibrous tissue. The lung architecture is partially obscured. No demonstrable evidence of infection. Emphysema is a compensatory dilatation of the air spaces with or without thickening of the septa

TABLE 13.—A TABULATION OF THE ROENTGENOLOGICAL CHANGES FOUND IN SILICOSIS TOGETHER WITH THE UNDERLYING HISTOLOGICAL CONDITIONS—*Continued*

SILICOSIS WITH INFECTION

The characteristic appearances described under "Simple Silicosis" are modified by infection as follows:

Roentgenological Appearances	*Histological Appearances*
6. *Localized discrete densities* and- or string-like shadows accompanying those of simple silicosis described above	6. Strands of fibrous tissue, often along trunks and septa, with or without areas of calcification; indicative of "healed" infection
7. *Mottling.* Shadows varying in size, with ill-defined borders and lacking uniformity in density and distribution accompanying simple silicosis	7. (*a*) Areas of bronchopneumonia with or without caseation (acute infection) (*b*) Lobar areas of proliferative reaction with or without caseation (subacute or chronic infection)
8. *Soft nodulation.* The nodular shadows described under "Simple Silicosis" (4) have now assumed fuzzy borders and- or irregularities in distribution. This change may or may not accompany No. 7	8. Perinodular cellular reaction either exudate or proliferative in character
9. *Massive shadows* of homogeneous density not of pleural origin symmetrically or asymmetrically distributed	9. Extensive areas of fibrosis probably due to organized pneumonia of tuberculous or non-tuberculous origin superimposed upon a coexistent silicotic process. Outlines of normal structures may be partially destroyed

Finally, it must always be remembered that nodulation and formation of new fibrous tissue continues in a victim after the exposure to a dust has ceased. The damage produced in the lung is permanent.

Roentgen-Ray Diagnosis.—The most important evidence of pneumoconiosis is presented by *x*-ray films. These should be in stereo. and in questionable cases a lateral view may be of some value. Table 13 was prepared by experts following the Saranac Symposium in 1934.

Differential Diagnosis.—In interpreting the films of suspected early silicosis, one must rule out passive congestion from cardiac decompensation, advanced bilateral bronchiectasis, asthma, malignant disease, and mycotic infections.

The *x*-ray diagnosis of silicotuberculosis from that of uncomplicated tuberculosis is frequently difficult. Pancoast and Pendergrass[5] classify these cases into seven groups:

1. Cases showing *x*-ray evidence of tuberculosis only, in spite of history of dust exposure
2. Cases showing obvious pulmonary tuberculosis with mild silicosis or pneumoconiosis. (Pancoast believes that these two groups should not be included in the *x*-ray diagnosis of silicotuberculosis.)

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

SCHEME REPRESENTING THE SEQUENCE OF LUNG-FIELD MARKINGS IN A TYPICAL CASE OF UNCOMPLICATED SILICOSIS

LINEAR	NORMAL LUNG MARKINGS & 1ST DEGREE EXAGGERATION OF LINEAR PULMONIC MARKINGS.		This is the range of markings usually seen on X-ray examination of persons who have never worked in a dusty trade.
	2ND DEGREE EXAGGERATION OF LINEAR PULMONIC MARKINGS, WITH OR WITHOUT BEADING.		
GRANULAR	1ST DEGREE DIFFUSE GROUND GLASS OR GRAINY APPEARANCE, NOT OBLITERATING LINEAR MARKINGS.		These are the earliest markings in the sequence of dust-induced changes which can be clearly differentiated from the changes that usually accompany advancing age, bronchitis, cardiac stasis, etc.
	2ND DEGREE DIFFUSE GROUND GLASS OR GRAINY APPEARANCE, OBLITERATING LINEAR MARKINGS.		
NODULAR	1ST DEGREE DISSEMINATED NODULES UP TO SIZE OF MILIARY TUBERCLES.		Cases with granular or nodular markings may be diagnosed as stage I or stage II silicosis depending on signs and symptoms present.
	2ND DEGREE DISSEMINATED NODULES EXCEEDING 1 M M IN SIZE, EMPHYSEMA PRESENT, USUALLY.		
COALESCENT NODULES AND CONGLOMERATE SHADOWS (EMPHYSEMA ALWAYS PRESENT)			May be diagnosed stage II or stage III depending on signs and symptoms present and degree of disability

Fig. 51.—This scheme gives an excellent graphic impression of the advancing stages of uncomplicated silicosis as interpreted from the *x*-ray markings. (U. S. Public Health Service, *Bulletin 244*, 1939.)

Silicotuberculosis.—It has been previously stated that silicosis may exist in a pronounced form without causing death or interfering with health. Tuberculosis likewise may be present in a person for years, causing no impairment of health, or may be easily controlled. But *tuberculosis in the presence of silicosis defies treatment or control.* To understand this, it must be realized that these two diseases do not represent a constant entity but rather a combination of all degrees of the two diseases. Tuberculosis may be present in either a quiescent or active stage before the lung is exposed to silica, or the tubercle bacillus may be implanted in a lung already silicotic.

Evidence of Tuberculosis.—If silicosis is superimposed upon an old inactive tuberculosis, certain characteristics of the preexisting tuberculosis may be in evidence. Gloyne[6] lists these as: (1) dense pleural adhesions, especially in the upper part of the pleural sac, tethering the apex of the lung to the apical pleura; (2) fibrotic contraction and scarring of the visceral pleura without adhesions, again especially well marked at the apices; (3) calcified nodules. Gardner, Gloyne, and others have pointed out that the calcified nodules of old healed tuberculosis have certain distinguished characteristics. They are usually beneath the pleura, attached to the wall of the small bronchus or in a mediastinal gland.

Silicosis "with Infection."—As to whether the frequency of silicotuberculosis is greater in those harboring the tubercle bacilli prior to exposure or in those who become infected with tuberculosis after silicosis has transpired is of interest to the experimentalist or pathologist but not to the practicing physician. What should interest him is that it is estimated that 75 per cent of all silicotic persons die of tuberculosis. There are many instances when it cannot be proved that tuberculosis is the coexisting infection. Under such circumstances, the diagnosis of silicosis with infection is preferred, but the chances are great that the unproved infection is tuberculosis. Silicosis with infection produces certain recognized *x*-ray findings. The nodules lose their distinct outline to become fuzzy and "soft" in appearance. They lack uniformity in density and outline. They coalesce, and conglomerate masses appear. While this is in progress, râles may be heard, fever may be present, and tubercle bacilli may be found. Marked weight loss, flattening of the chest, increased productive cough, pleural adhesions with chest pain, and cavitation may become prominent.

Complications and Sequelae of Silicosis. *Emphysema.*—This is an almost constant condition accompanying silicosis. Its production explains the barrel chest. Occasionally large emphysematous areas are mistaken for tuberculous cavitation.

Cardiac Involvement.—Many of these silicotic persons die apparently from cardiac failure, yet postmortem examinations rarely reveal valvular lesions or particular involvement of the myocardium. It seems evident to the writer that months or years of cardiac effort against resistance could result in myocardial weakness and a cardiac death. Certainly in the presence of arteriosclerosis, silicosis becomes an added burden on heart function. Electrocardiograms taken of silicotics reveal no significant findings except evidence of displacement to the right, due to overstraining of the right heart.

Bronchitis and Bronchopneumonia.—Secondary infection from

septic invasion is common. Its frequency, compared to that in persons not exposed to silicosis, is not revealed by statistics.

Carcinoma.—No evidence exists that malignant disease of the lungs commonly results from silicosis. From a compensation angle it should not be assumed that a worker in a silicosis hazard has incurred cancer because of his exposure. Gardner[7] reviews a case reported by Bradshaw and Chodoff in which roentgenograms of an ex-coalminer with cough, expectoration, loss of weight, and dyspnea showed a localized mass the size of a half dollar in the right hilum. There was no evidence of generalized silicotic nodulation. The sputum was negative for tubercle bacilli; bronchoscopic examination showed an obstructive lesion compressing the right upper lobe bronchus with a tendency to mucosal bleeding. Films taken at the end of expiration showed an obstructive emphysema of the right upper lobe. Tuberculosis was apparently excluded by the absence of hemoptysis, elevation of pulse and temperature, and a persistently normal sputum. Although two bronchial biopsies revealed no evidence of tumor but merely "inflammatory exudate, blood clot, and anthracotic tissue," the probable diagnosis of bronchiogenic carcinoma superimposed upon anthracosilicosis was entertained. Exploratory thoracotomy was advised but refused.

One year and five months later, the patient's films showed marked symmetrical elevation of both hila with increased "fibrosis" radiating upward and laterally from both hila. The trachea was displaced to the right, and there was emphysema in the left lower lung field. On bronchoscopic examination the orifice of the upper lobe bronchus was now clearly visualized, but there was an obstruction of the main tube at the level of the middle lobe bronchus. Another biopsy produced no cellular elements. Temperature and pulse had remained normal, but the patient had lost 21 pounds, and his dyspnea had increased.

A thoracotomy was performed, and the surgeon palpated a stony hard mass in the upper lung covered by dense adhesions between the apex of the lung and the anterolateral chest wall. Only a few small nodes were felt about the hilum of the lung and along the trachea. He then performed a total pneumonectomy, but pneumothorax developed on the opposite side and the patient died. Pathological examination revealed an anthracosilicosis of conglomerate type involving the upper three fourths of the upper lobe. "The remainder of the lung contains a moderate amount of pigmentation and of fibrotic foci." On autopsy a localized lesion, inferred to be similar in character, was found in the apex of the upper lobe.

The authors cite the difficulties in diagnosis, point out that gen-

eralized nodulation was not observed in the roentgenogram and hence silicosis could only be inferred, and plead for more exploratory thoracotomies.

The reviewer admits the difficulties but points out that "fibrotic foci" were reported in the lower part of the right lung after removal from the body. They may have been too small to cast definite shadows on a film. In his mind the localization of the massive fibrosis in both upper lobes suggests reaction to dust localized in the scars of healed tuberculosis. In such cases much of the inhaled dust seems to come to rest in the vicinity of the scars, and subsequent retraction of the lung tends to pull nodules upward toward the conglomerate focus. Compensatory emphysema produces the impression that there are few nodules in the lower lung. Conglomerate fibrosis is much more prone to develop in the modified anthracosilicosis than in uncomplicated silicosis. The stony hard consistence of such lesions is one of their major characteristics. The tracheobronchial lymph nodes in carcinoma are generally enlarged and more matted together than in pure anthracosilicosis.

Extrapulmonary Tuberculosis.—A survey made by Cohen at the White Haven, Pennsylvania, Sanitorium, and reviewed by Schnurer,[8] corroborates the established fact that pulmonary tuberculosis is more common in anthracite-coal miners than in non-miners. Of 541 miners and 730 male non-miners of the same age who came to autopsy, it was the cause of death in more than twice as many miners as non-miners. Cohen reports on the occurrence of extrapulmonary tuberculosis in a series of autopsies. Only 20 per cent of 50 anthracosilicotics with pulmonary tuberculosis had intestinal tuberculosis, while in 84 patients with non-anthracosilicotic pulmonary tuberculosis, it was present in 51 per cent. The degree of anthracosilicosis, rather than the age, determines the frequency with which intestinal tuberculosis occurs. It was seen in miners under thirty years of age in 67 per cent because the existing anthracosilicosis was early or only moderately advanced. In those above thirty years, when the coniosis was advanced, it occurred in only 14 per cent of cases as compared to 41 per cent in the non-mining group of the same age. The author explains the comparative rarity of intestinal tuberculosis in advanced anthracosilicotuberculosis on the basis of (1) chronicity of the pulmonary process, and (2) the extensive pulmonary fibrosis, particularly the vascular sclerosis which tends to prevent the spread of tubercle bacilli. There are no definite symptoms referable to the gastro-intestinal tract in those having intestinal tuberculosis associated with anthracosilicosis.

During the same period of time, laryngeal examinations were

made on 365 subjects. Of 100 tuberculous miners, only 27 per cent had laryngeal tuberculosis as compared to 31 per cent of 276 tuberculous non-miners, despite the fact that the tuberculosis in the miners was much more severe. Thus, 57 per cent of the miners died of tuberculosis, while only 12 per cent of the non-miners died of the disease. In those who died, laryngeal involvement was seen in 33 per cent of the miners, and in 59 per cent of the non-mining group. Again, age *per se* was not important in the incidence of laryngeal tuberculosis. In the same series of autopsies, 32 per cent of the miners had extrapulmonary lesions other than those mentioned, as compared to 46 per cent in non-mining adults.

Basis for Diagnosis. *Reliable History of Adequate Exposure.*—The patient's statement that he has been working in a silica-laden atmosphere cannot be accepted without investigation. To accept or deny a case for compensation without investigation often instigates unnecessary litigation. An adequate exposure constitutes working within this dusty atmosphere over a period of several years. The claimant may be employed by a firm engaged in handling silica, but he may be a janitor or shipping clerk or employed in an endeavor outside the actual hazard. Furthermore, the time one works in such an atmosphere is exceedingly important. From ten to twenty years is the average length of time in an average concentration. In excessive concentrations, this average time may be as low as two years. Less than two years is to be viewed with skepticism until evidence of unusual factors is obtained.

Occupational History.—A claimant may be engaged in an occupation foreign to silicosis at the time he becomes disabled. His last exposure to silica may have been ten or fifteen years previously. To allocate the responsibility under compensation properly, his occupational history since he left school is necessary.

x-Ray Findings.—Chest films, preferably in stereo, should be interpreted by one thoroughly acquainted with the roentgenological manifestations this disease presents.

Sputum Examination.

x-Ray Diffraction Analysis.—(See discussion of this method under Case XIII.) Of occasional value only.

MEDICOLEGAL ASPECTS

The fact that a workman has evidence of silicosis does not necessarily entitle him to compensation. A scar, evident in the pulmonary tissue is comparable to a scar on the surface of the body—if neither interferes with the function of the body or part, it is therefore not compensable. However, if the fibrous tissue of a scar pre-

vents contraction of a finger, for instance, loss of function is to be compensated. Similarly, when the scar tissue in the lung interferes with its function to the point that one becomes disabled, then compensation is due. Concisely, silicosis is to be compensated for only when disability is present.

Evaluation of Disability.—But upon what basis is disability to be evaluated or its extent determined? Two men may present comparable results from physical examination, as well as almost identical *x*-ray pictures, yet one claims no disability and the other does. Not only physical examination but also history, roentgen-ray studies, exercise-tolerance tests, and estimations of cardiac and respiratory efficiency have been suggested as measuring rods. Mayer[9] states that pulmonary function may be disturbed in one of three ways: (*a*) in ventilation, (*b*) in gas exchange, and (*c*) in pulmonary circulation.

Testing Ventilation.—Ventilation is tested by calculating the vital capacity and multiplying this by the highest rate per minute of respirations which the patient can accomplish. Pulmonary fibrosis associated with emphysema interferes with the capacity of the lung to inflate and deflate. There results an increase in minute volume of ventilation, while at the same time maximal breathing capacity is reduced.

Testing Gas Exchange.—The gas-exchange test, with the use of a spirometer, estimates the oxygen intake and the carbon dioxide output. According to Mayer, it is expressed in the following three ways:

(*a*) *Oxygen Debt.*—This is the amount of excess oxygen the normal person needs at the end of exertion. It usually takes from one to two minutes to recover the oxygen necessary to relieve dyspnea. Decompensation of the gas-exchange mechanism is evident when the recovery period is longer than three minutes.

(*b*) *Oxygen Deficit.*—This is revealed by the excess intake of oxygen utilized when the patient shifts from air to oxygen inhalation. This is a sign of latent oxygen want, which may be present at rest. It is found, despite full oxygen saturation of the blood, in pulmonary diseases bordering on decompensation.

(*c*) *Oxygen Unsaturation.*—Oxygen unsaturation of the hemoglobin of the arterial blood, as determined by gas anaylsis, is found in disturbed gaseous exchange. Normally the oxygen saturation of arterial blood is about 95 per cent; saturation below 93 per cent indicates anoxemia, a sign of deficient oxygen supply.

Testing Pulmonary Circulation.—Pulmonary circulation is tested by the use of either dehydrochloric acid, sodium cyanide, or calcium, determining the circulation time from arm to arm, arm to tongue, or arm to lung. Intravenous infusion of 1000 cc. of fluid, followed by a

study of its effect on the venous pressure and vital capacity, reveals loss in reserve of the pulmonary vascular bed by the proportionate rise in the venous pressure and decrease of vital capacity.

Congestive heart failure was observed to be a common occurrence in cases of far-advanced anthracosilicosis by Giering and Charr[10] among the hard-coal miners of eastern Pennsylvania. They attempted to determine whether dyspnea in these cases was of cardiac or pulmonary origin by studying the vital capacity, the venous pressure, and the velocity of pulmonary circulation. Of twenty-five miners studied, two showed by roentgenograms evidence of heart enlargement, while secondary polycythemia and reduction of vital capacity were frequent. No appreciable variation between the normal venous pressure and pulmonary and complete circulation time was noted. Electrocardiograms in the majority of instances showed myocardial damage.

There is no question but that the procedures employed by Mayer, Giering and Charr, and others constitute an advance step in the attempt to better evaluate the disability of the silicotic, but the method is too elaborate and complicated for the average physician. A routine use of these methods by a special examining board might, after a period of time, produce some acceptable standards of measurement. Until then, the question remains largely a matter of clinical judgment. With or without the use of these tests, the examiner must rule out as a partial or complete cause of dyspnea, such conditions as unrelated respiratory disease, unrelated cardiac disease, or mechanical blockage to normal respiration, such as occluded nasal passages, nasal polyps, sinusitis, and the like.

Illustrative Cases:

Case I.—A thirty-six-year-old Mexican female presented herself to the community surgeon who did the first-aid surgery for a soap manufacturer. Her complaint was that about six months previously she first noticed shortness of breath, which progressed until at the time she was markedly dyspneic on the least effort.

Following examination, she was told that she had a bad heart. Digitalis was prescribed and bed rest ordered. Eight months later her employer referred her to this clinic. History taken here revealed that she had worked for fourteen years in abrasive-soap factories. Her x-ray films showed a bilateral involvement of the lungs with a widespread discrete nodulation of varying density and size, with fuzziness of the borders and areas of coalescence. In addition to shortness of breath, she complained of chest pains. There was no loss of weight or fever. Subsequently chest pain was increased and there was a noticeable weight loss. No tubercle bacilli were found.

This girl went for over eight months without an accurate diagnosis and, therefore, without compensation to which she was entitled. Her first physician failed to take an accurate occupational history or *x*-rays. The case was compensable, and she was placed on total disability.

Case II.—A man fifty-nine years of age, who had been in the employ of a monument company for twenty-six years, was discharged when a physical examination, required of all employees of the company by its new insurance carrier, showed that he had *silicosis*. The worker contended at that time, however, to the examining physician that he was all right and could continue to do his work. Subsequently he instituted proceedings under the Wisconsin Workmen's Compensation Act before the Industrial Commission. An examiner for the commission found that the man was suffering from silicosis in an advanced stage, caused by the exposure in his employment and that since as a result he was incapable of more than slight physical exertion and could be employed only in occupations which involved so-called "light" work, he was 50 per cent disabled. The Industrial Commission confirmed those findings and awarded compensation accordingly. From a judgment of the circuit court for the county, affirming the award, the employer and his insurance carrier appealed to the Supreme Court of Wisconsin.

In general, the Wisconsin Workmen's Compensation Act provides for compensation for such disability, whether resulting from accidental injury or from occupational disease, arising out of and in the course of employment, which results in a wage loss. The appellants seem to have contended that the man had suffered no wage loss. Wage, answered the Supreme Court of Wisconsin, is dependent on two factors, time and rate of compensation. In Zurich General Accident and Liability Insurance Company *vs.* the Industrial Commission, 203 Wis. 135, 233 N. W. 772, this court held that a worker who was transferred from a place of exposure to outside work at a diminished wage suffered a wage loss; that is, his rate of compensation per unit of time was diminished.

The real source of difficulty, the Supreme Court continued, in silicosis cases is that many men suffering from the ailment in some of the various stages are able to, and do continue to, work and receive full compensation therefor long after they have sustained what has been referred to as a medical disability, but what might be more properly referred to as a pathological disability, and therefore as a matter of fact sustain no wage loss. It is because the legislature has so far seen fit to withhold compensation for physical impairment which does not immediately result in the physical incapacity of the claimant to work that the difficulty in these cases springs. There is a feel-

ing that a person who has sustained loss of physical vigor and had his system invaded by foreign substances, which may and often do result in impairing his ability to work and not infrequently in death, should have compensation. If, however, the legislature had intended the term "disability" to embrace so-called "medical" or "pathological" disability as distinguished from actual physical incapacity to work, it would undoubtedly have said so.

"This court," continued the Supreme Court, "has previously held that the injury or wage loss to be compensable must be sustained at a time when the relation of employer and employee existed. An amendment to the Workmen's Compensation Act adopted thereafter (*Laws*, 1933, c. 314, Sec. 27) provides that the time of injury or the occurrence of disability shall be deemed to be the last day of work for the last employer whose employment caused the disability. This obviously refers the time of injury or disability back to a point in time when the employer and employee relationship existed. Even if a plant shuts down, and an employee is discharged and is not thereafter employed, if he is thereafter disabled, the time when his disability occurs is referred to as the last day of employment which caused his disability. In the case at bar when it is considered that admittedly Marsz is suffering from silicosis; that as a result of it he is incapable of more than light physical exertion; that by reason of his physical incapacity he can be employed only in occupations which involve so-called light work, this court cannot say that there is no evidence to sustain the finding of the Industrial Commission that Marsz has sustained a wage loss, which, measured by the rate of compensation he had theretofore received, amounts to 50 per cent."[11] The award in favor of the worker accordingly was affirmed.

This case is recited here to show the legal phase of silicosis and wage loss.

Case III.—The workman's employment subjected him to the dust of burnt clay and other vitrified products disseminated by machines used in the manufacture of china, and to silica dust raised by sweeping. In 1927, after several years of employment, he developed a cough. He became chronically tired and weak. The employer discontinued business in November, 1933, but at that time the workman did not consider his condition serious enough to require medical attention. The following February a physician diagnosed the workman's condition as due to a slight temporary inflammation of the lungs. In March, when the patient was treated for injuries from an automobile accident, the physician discovered a tuberculous condition. In May, following roentgen examinations, his condition was diagnosed as pulmonary tuberculosis activated by silicosis. Attributing the silicosis to his former employment, he instituted proceedings for compensation under

the California Workmen's Compensation Act. The Industrial Accident Commission dismissed his claim on the grounds that it had not been filed within six months after the date of injury, as required by the Workmen's Compensation Act. The worker then appealed to the District Court of Appeal, Second District, Division 2, California.

"In cases of the character under consideration," said the District Court of Appeal, "the date of injury, in computing the limitation period set forth in the Workmen's Compensation Act, is not the date of any particular exposure to the hazards of the employment, but the time when the accumulated effects culminate in a disability traceable to the latent disease as the primary cause, and by the exercise of reasonable care and diligence it is discoverable and apparent that a compensable injury was sustained in performance of the duties of the employment." [12]

In the case cited, the court disapproved the holding of the Commission that the statute of limitations began to run as of the date the employee first was disabled and indicated that the running of the prescribed period began when the presence of silicosis was, or should have been, diagnosed as the primary and efficient cause of the injury. "It appears quite obvious," said the court in the present case, "that the claimant, from the inception of his cough until he received the roentgen report in May 1934, had no reason to believe he was suffering from a compensable injury, silicosis, arising out of his employment. Even his visit to a physician in February 1934, disclosed nothing further than the given diagnosis of a slight inflammation of the lungs from which a speedy recovery was assured.

"An employee," continued the District Court of Appeal, "is not to be deprived of compensation because he incorrectly diagnoses his condition." The commission in this case found that the claimant should have discovered the character of his condition prior to the date of his final termination of work. "Such a finding," concluded the District Court of Appeal, "is without support in the evidence. It is wholly unreasonable to argue that the worker should, through the exercise of reasonable care and diligence, have known in November, 1933, that which his medical adviser did not discover in February, 1934." The court accordingly ordered that the award of the commission dismissing the claimant's claim be set aside and that the commission proceed with the determination of the claim.[13]

The above case from the *J.A.M.A.*[14] illustrates the legal viewpoint in one state regarding the period in which a claim for compensation due to silicosis may be filed. This court intimated that if the claimant had known or had been informed that he had silicosis and had failed to file for compensation within the six-month period, then the statute of limitations would have held.

Case IV.—A foundry company had to reinsure its employees. The prospective insurer demanded as a condition precedent to the execution of a policy that all employees be examined by a physician selected by it. Before the examination was made, all employees were discharged. Only those were reemployed who were believed to be physically fit for the duties to be imposed upon them. Five employees who were not reemployed, because they were not physically fit, were awarded compensation by the Industrial Commission, under the Wisconsin Workmen's Compensation Act. Thereupon their employer and its insurance carrier brought actions against the commission and the several claimants to set aside the awards. From a judgment modifying and affirming the award in each case, the Industrial Commission, the several claimants, the employer, and the employer's insurance carrier all appealed to the Supreme Court of Wisconsin.

The Supreme Court based its decision on the case of one claimant, as the same questions were involved in all cases. This man, before he was discharged, had worked steadily. He was not aware that he was suffering from any disease. The physical examination, however, showed that he had had silicosis for probably five years or more and was in the second stage of that disease and that tuberculosis had already developed. Although at the time of the examination he was able to perform his work, his chance of recovery would be diminished if he remained in the dusty employment in which he had been engaged. Reexamination three months later showed that his ability to do work requiring physical exertion was greatly diminished, if he could be said to be able to do it at all. Nevertheless, his employer and its insurance carrier contended that he had suffered no compensable injury in the course of his employment with his employer.

"Disability within the meaning of the statute," said the Supreme Court, "occurs when an employee is disabled from rendering further service; that is, when he no longer has physical ability to perform his work in the usual and customary way. Unless he is so disabled he sustains no compensable injury even though in the course of his employment he is subjected to conditions that contribute to disability as an end result. The right of an employee to compensation depends on whether or not, in the course of his employment and because of it, an occupational disease renders him incapable of performing his customary work. . . . It has been argued that if an employee is entitled to compensation, as he is when he voluntarily ceases work because of physical incapacity to continue, then an employee who is discharged by his employer because of potential physical disability is equally entitled to compensation. For the latter contingency, however, the present law, which was framed to cover injuries resulting from accident, rather than incapacity resulting from occupational disease, affords no relief. An employee who has been exposed to industrial hazards, who has suffered no *physical disability* that prevents his performing his

work in his usual and customary way, and who is discharged because an examination discloses that such disability may arise in the future, is not entitled to compensation for *medical disability*."

It has been argued that it is against public policy to permit an employer to discharge an employee who has been exposed to industrial hazards, in order to anticipate his employee's future disability. "That, however," said the Supreme Court, "is a problem for the legislature and not for the courts. The courts can do nothing more than interpret and administer the statute. The remedy lies solely with the legislature." Finding no evidence to show that the claimants suffered disability within the meaning of the law during the period of their employment with the foundry Company, nor until after the relation of employer and employee had been terminated, the judgments of the courts below in the several cases were reversed and remanded with instructions to enter judgments setting aside the awards of the Industrial Commission.[15]

Case V.—The plaintiff, a workman, in the course of his employment as a grinder in the defendant company's grinding department, operated emery wheels for a period of five years. Later he sued the company at common law, alleging that, because of the company's failure to fulfil its statutory duty of supplying adequate exhaust fans and sufficient ventilation, he had contracted ". . . the occupational disease of silicosis as a result of inhaling . . . a quantity of steel, ascoloy, and carborundum dust." From a verdict in favor of the plaintiff and a judgment denying the defendant's motion for judgment notwithstanding the verdict, the defendant appealed to the Supreme Court of Pennsylvania.

The Workmen's Compensation Act of Pennsylvania provides that it shall apply to industrial accidents within the commonwealth. It defines the terms "injury" and "personal injury" as meaning "only violence to the physical structure of the body, and such disease or infection as naturally results therefrom." The defendant company contended: (1) that the plaintiff had sustained an occupational disease, for which no compensation was provided in the Workmen's Compensation Act; and (2) that he was barred from bringing an action at common law, because silicosis causes "violence to the physical structure of the body," thus making the plaintiff's condition an "injury" within the meaning of that act for which the act provided no compensation.

"The Workmen's Compensation Act," said the Supreme Court, "must be interpreted as if to the phrase 'violence to the physical structure of the body' there was added the phrase 'when said violence was due to an accident'; the Act does not apply to all cases in which workmen receive injuries which do 'violence to the physical structure of the body.' " Every disease affecting human beings is pathologically an

alteration of the normal, healthy condition of the human body and as such involves some degree of "violence." The word "violence," however, in common usage connotes a vehement application of force and is not ordinarily used to characterize the progress of a disease. "Conceding," continued the court, "that diseases such as tuberculosis and silicosis may involve 'violence' in some degree to the 'physical structure of the body,' a workman who has contracted the disease in the course of his employment does not come under the Workmen's Compensation Act unless that disease had its origin in an injury by accident, and occupational diseases do not so arise. In the opinion of the court the plaintiff could not have maintained an action under the Workmen's Compensation Act because he had not sustained a compensable accidental injury."

The Supreme Court was unable to agree with the defendant's contention that in Pennsylvania no action lies at common law for an occupational disease. Even though the Workmen's Compensation Act provides no compensation for such a disease, it cannot be said that the employee must assume the risk and cannot resort to his common-law remedy to secure damages provided the disease, whether occupational or otherwise, arose from the employer's negligence. The court concluded that the workman had a common-law right of action against his employer for injuries caused by a disease contracted in the course of his employment and owing to the negligent conduct of his employer in violation of a statutory duty.

Accordingly, the Supreme Court affirmed the judgment in favor of the plaintiff.[16]

This is an example of the disputes which frequently occur in those states which have no special occupational-disease schedule regarding the term "injury." (Pennsylvania has since enacted an occupational-disease schedule.) In the above case no mention is made as to whether or not the patient actually had silicosis.

Case VI.—A forty-one-year-old male had been an electric welder for sixteen years. In June, 1937, he fell, striking his chest wall. *x*-Rays revealed no fractured ribs but did show unusual markings in the lung field. His physician informed him that he had silicosis, which the patient promptly reported to his employer. When seen by us he had no complaints; his past medical history was negative, and his physical findings the same. *x*-Rays of the chest showed nodular shadows which *resembled* modified silicosis.

The patient was informed that he did not have silicosis and that there existed no compensable condition. Examination of his employment condition revealed that he had never worked in a confined area and that he inhaled no appreciable amount of dust, but rather the

fumes only from the welding process. This patient had inhaled particles of iron oxide which were responsible for the markings noted by the *x*-ray. Iron deposits (free from silica) cause no functional impairment of the lungs, no symptoms, or susceptibility to complicating infections.

Case VII.—On July 3, 1940, I examined a sixty-one-year-old miner at the Mojave, California, Hospital, who was in the employ of a mining company. He had been a timberman for twenty-eight years, eighteen of which were spent in a gold mine, and ten in copper mining. His past medical history was not significant. He first noted some shortness of breath in January, 1940, which was not disabling. About June 1, this dyspnea increased to the point where he was forced to quit work. Three weeks later he entered the Mojave Hospital with marked dyspnea, ascites, and edema of the ankles.

When seen by the writer he was in the terminal stages of what appeared to be myocardial insufficiency. Propped up in bed, he was markedly cyanotic and dyspneic. Respirations were rapid, shallow, and with little movement of the chest cage, which was of the huge barrel type. Percussion of the chest was dull, and auscultation revealed many large mucous râles and what appeared to be terminal rhonchi. These excluded any other findings which might have been present except a friction rub in the left posterior base. The heart rate was 120, tones almost imperceptible; and the blood pressure was 110/90. (It was recorded at 160/100 on admission.) The abdomen was distended with fluid, the genitalia edematous, as were the ankles. He died ten hours after this examination. No *x*-rays or laboratory investigation had been done.

The details of the autopsy will be omitted. The anatomical diagnosis was: acute, purulent tracheitis and bronchitis; bilateral pneumonitis; moderately advanced silicosis; enlargement of the right ventricle of the heart; chronic passive congestion of the liver, spleen, and kidneys; and moderate generalized arteriosclerosis.

It was not possible to determine the extent of the silicosis roentgenologically since no films were taken. It appeared to this examiner that the course of events was a rapidly progressive myocardial failure, followed by a complicating pulmonary infection which could not be handled by the patient, owing to his heart condition and also to the existing deficient pulmonary aeration. To what degree the silicosis entered as a cause of death it would be impossible to state, but it was considered a factor in preventing recovery and, therefore, the death was held to be compensable.

Case VIII.—Two weeks after the death of the workman referred to in Case VII, a forty-three-year-old miner, employed by the same

mining company, reported to the clinic alleging silicosis. He was well acquainted with the details of the preceding case. He claimed that six months prior to the date of examination he had noted shortness of breath, cough, and lassitude. During these six months he thought he had lost twelve pounds. For the past two months the dyspnea had increased to the point where even slight exertion caused shortness of breath. His past medical history was essentially negative, and there was no family history of tuberculosis.

Occupational History.—The patient's mining experience consisted of about nineteen years of copper-mining in Arizona, four years in gold-mining, and about four years in gold-prospecting.

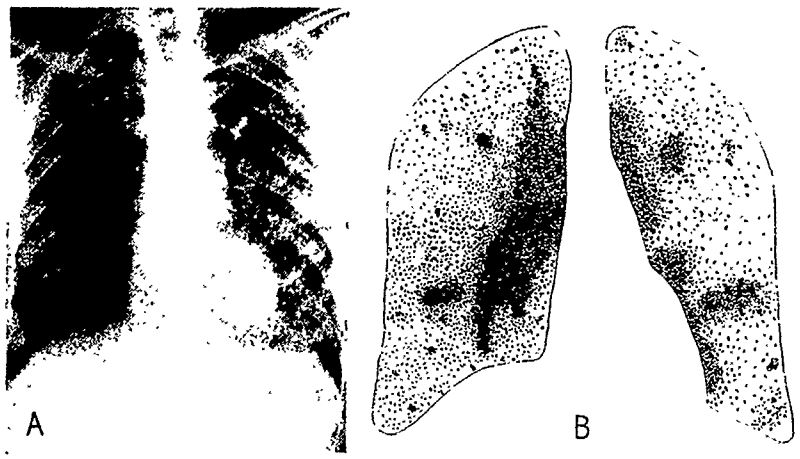


Fig. 52.—*A*, Advanced silicosis of a gold-miner referred to in Case VIII. While a co-existing infection appeared likely, tuberculosis was not proved to be present. *B*, A schematic drawing of third-stage silicosis or silicosis with conglomerate shadows and coalescent nodules. The medial aspect of each lung is especially heavy because of the hilus shadows and the extension of the bronchus in addition to the conglomeration. The first consequence of dust inhalation as seen by *x*-ray examination occurs in the hilus and adjacent portion. The markings proceed mostly downward and outward and have been purposely exaggerated by this drawing to emphasize this.

Physical Examination.—The heart and lung findings were not significant, and the entire examination was essentially negative. Laboratory investigation failed to disclose any evidence of tuberculosis. Electrocardiogram normal. Stereo *x*-ray films showed moderately advanced silicosis (Fig. 52).

This case is included owing to the interest provoked by the occupational exposure. The *x*-ray films disclosed silicosis to an extent not to be expected in one whose exposure was apparently for only about four years. Furthermore, this four-year exposure was in a mine where a wet process is maintained, and recent dust counts did not indicate a

harmful concentration: and also, the patient wore a respirator. In this regard, it is interesting to note what Dr. Porter Miller, of Los Angeles, wrote regarding this patient and his exposure. Dr. Miller[17] saw this patient two days after the completion of my examination, and commented as follows:

The exposure in this case is interesting. The applicant began mining in 1914 and has been at it ever since. There were periods when he would be off work and during the interim he would be prospecting. I feel that in the past, too little attention has been directed towards prospecting as a contributing factor in the development of silicosis. It is the general belief that if a man is above ground, or at least not deep within the ground, that the exposure is more or less minimum. In that I would take issue. In the first place, this is all dry mining and we know that Mojave and 29 Palms is a very dusty area, due to the winds which constantly keep the dust in circulation. If a man is constantly digging in the earth's crust which normally contains sixty per cent silica, is it not reasonable to assume that this is quite a factor? I would refer the reader to the United States Public Health reports 50, 1369-1385 (1935), by Brown, Gottlieb, and LaBroun, and their report upon dusty conditions in the dust bowl, which as you know, would be more or less parallel to the man who might be prospecting. It was their findings that during the dust storms silica was present in the air in from 67 to 93 per cent, and they also stated that this would provoke pathologic changes if the exposure was continued long enough, and particle size was fine enough. It would seem that the exposure while prospecting, assuming conditions to be dry, would be equally as great as in a mine where working conditions are regulated, and men have access to respirators, and wet methods and ventilation processes are used to a maximum degree.

I cannot refute, or concur in, what Dr. Miller observes. This is the first claim ever presented to me wherein prospecting might be considered a factor of exposure; nor have I ever read any comments elsewhere on this point. While it may be true and worthy of consideration, it appears to be a dangerous assumption without knowing the details of the prospecting, the type of dust encountered, the concentration, and a serial study of other prospectors' lungs.

An award was made in this case by the Industrial Accident Commission of California.

Case IX.—A twenty-four-year-old male entered a claim for disability after being told by his family physician that he had "dust tuberculosis." Upon being referred to our clinic he brought with him a letter from his physician which stated that he had "arrived at this

diagnosis after consideration of the patient's symptoms, occupation, and films of the lungs." Briefly, the history was as follows.

For the past six months the patient had noticed a loss of weight, increasing fatigue, shortness of breath, cough, and occasionally some blood in the sputum. When closely questioned, he recalled that just prior to this decline he had suffered an attack which he thought was "flu" but for which he did not consult a doctor. His past medical history revealed measles at the age of fourteen, complicated by rheumatic fever.

The patient had been engaged as a sprayer for a concrete emulsion company for the five preceding years. This work consisted of first applying an adhesive paint to the facing of buildings which was then quickly sprayed with a 25-mesh silica dust. Then following a repainting of adhesive substance, black mica of 80 mesh was rapidly sprayed upon the surface. The workmen wore respirators.

Physical Examination.—Temperature 99.4° F.; tonsils cryptic and red, anterior pillars injected. Lungs were normal except for evidence of congestion at both bases. Heart was slightly enlarged, with the apical impulse noted upon inspection. Auricular fibrillation was present, with a diastolic murmur at the apex. The blood pressure was low.

Laboratory Findings.—Blood count showed a mild anemia with a leukocytosis of 14,300 white cells. Sedimentation rate increased. Urine showed slight albumin with occasional red blood cells. Repeated sputum examinations revealed no tubercle bacilli. Electrocardiogram not done. Stereoscopic films of the lungs showed no evidence of tuberculosis or silicosis.

The *x*-ray film taken by the patient's family physician was a single anteroposterior view, which was greatly overdeveloped and valueless for interpretation. Subsequent stereo views denied the evidence of either tuberculosis or silicosis. The history of rheumatic fever when younger, and a recent exacerbating illness followed by typical findings, resulted in a diagnosis of rheumatic heart disease (mitral stenosis with auricular fibrillation). Foregoing any censorship of the family physician for assuming a "dust tuberculosis" to be present because of the symptoms and occupation, this case is cited in order that it may be emphasized that good roentgenological technique should be employed in the filming of suspected silicotic patients, and that these films be interpreted by a roentgenologist or someone experienced in silicosis. This case was not compensable.

Case X.—A sixty-four-year-old male reported to his physician because of shortness of breath. Although retired at this time, he had been engaged at intervals in hard-rock mining for a period of approximately twenty-four years. He was cognizant of the effects of silicosis

and stated honestly to the examining physician that he believed his difficulty was due to this disease. His family doctor had informed him that there was only one way to determine the presence of silicosis, that being by *x*-ray. Accordingly, the patient was referred to a roentgenologist who reported that the films showed first-stage silicosis. Upon the reception of this report, the patient was informed by his physician that the dyspnea was due to silicosis. The man, therefore, entered his claim for compensation.

Fig. 53.—Advanced first-stage or early second-stage silicosis. As mentioned in Case X, patient had arteriosclerosis with hypertension and mitral heart disease. His mild dyspnea on effort was thought to be cardiac in origin.

Past Medical History.—The past medical history was entirely negative other than for rheumatic fever at the age of twenty. The patient had maintained his weight for over fifteen years.

Physical Examination.—The physical examination of this patient revealed a long, thin chest, not barreled or emphysematous in type. There was no restriction of the respiratory excursion. Auscultation of the lungs revealed no râles or other adventitious sounds. The heart showed a marked mitral stenosis. Blood pressure, 180 100. Generalized

arteriosclerosis. Albumin and casts in the urine. Mild second-degree anemia. Negative blood Wassermann. The *x*-ray films confirmed the previous report of first-stage silicosis (Fig. 53).

The first physician consulted by this patient never examined him. He correctly stated that the *x*-ray was the best means of determining the presence or absence of silicosis. When he received the report that first-stage silicosis was present, he informed the patient that this was the cause of his dyspnea. He instituted no search for a cause of the dyspnea and falsely assumed that dyspnea can result from early silicosis. The diagnosis was mitral heart disease, with generalized arteriosclerosis and first-stage silicosis. The patient was denied compensation on the basis that his disability was due to his heart disease and that first-stage silicosis causes no disability.

Case XI.—Collins reviews a case reported by Lovelock[18] of a man who had mined steamcoal for twenty-two years in South Wales. He was admitted to the Brompton Hospital for chest diseases, with a diagnosis of tuberculosilicosis. No tubercle bacilli were found, and *x*-rays raised doubt as to the diagnosis. He was transferred to St. Mary's General Hospital. He lost ground and finally died, but before death, an enlarged liver had indicated the presence of carcinoma. Examination revealed anthracosilicosis of both upper lobes and apices, together with carcinoma of the right lower lobe bronchus. A guinea-pig injected died of generalized tuberculosis. A silica content of the lungs was between eight and nine times normal, amounting to 2.83 per cent in fresh lung and 12.43 per cent in dry lung. This case during life presented great difficulties in diagnosis, and after death raised legal difficulties regarding the claims of the dependents for compensation because of the industrial origin of the cause of death.

Case XII.—A twenty-nine-year-old male had been employed in a pottery works for six months, when in September, 1939, he developed a fever, cough, and pain in his chest. After the patient had been in bed for five days, his physician had the patient's chest *x*-rayed, and informed him that he had silicosis. The insurance carrier sent the patient to this clinic. When seen six weeks after his acute episode, he complained only of occasional pain in the chest. The history revealed that as an inspector he walked through rooms where there was an atmosphere containing 40 per cent silica. He wore no respirator. It was estimated that the total hours spent each day in this atmosphere were three and that the total number of hours during his entire employment were ninety. His physical examination, except for enlarged tonsils, was negative. The *x*-ray films were entirely negative for any type of lung pathology. By actual count there were five small, indistinct nodules scattered throughout the entire lung field. It was prob-

able that these findings led the physician to assume that silicosis was present.

This case has no value except to show the cost of faulty interpretation. The patient was sure he had silicosis because his "physician said so." He would not accept our diagnosis and took his case to the Industrial Accident Commission, where it was denied. The procedure

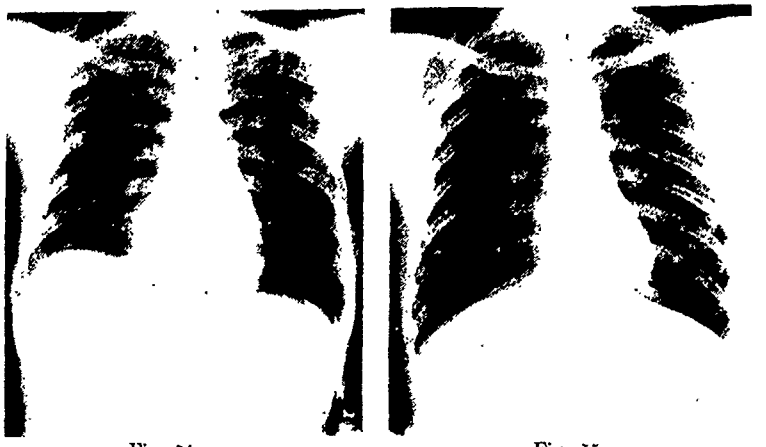


Fig. 54. Fig. 55.

Fig. 54.—This film reveals irregular linear and nodular infiltration involving the greater portion of the upper half of the left lung; also involvement of the right apex. There is a thin-walled cavity, 3 by ½ cm. in the greatest diameter, at the left apex. There is obliteration of the right costophrenic angle. The *x*-ray picture indicates moderately advanced pulmonary tuberculosis. Clinical investigation confirmed the diagnosis. This twenty-seven-year-old Mexican claimed compensation because he had been diagnosed as having pneumoconiosis as a result of operating at intervals, over a five-year period, a carpet-making machine in an atmosphere laden with heavy dust.

Fig. 55.—Very early silicosis with questionable apical tuberculosis. This twenty-nine-year-old male had helped his father prospect for gold at intervals during his youth. He had actually mined gold for a combined period of about one year. He sought compensation in California after being turned down by the Canadian government when he sought to become an aviator in their air force. He had no symptoms of tuberculosis or silicosis. Physical examination and laboratory investigation failed to reveal evidence of tuberculosis. The film is used here to demonstrate the earliest stage of silica deposition in the lung tissue.

cost time and money. An accurate diagnosis in the beginning would have prevented this wasteful expenditure.

Similar cases are rather frequent. Tuberculosis, cardiac decompensation, asthma, emphysema, pleurisy, and various forms of pneumonitis are attributed to silicosis in instances where no silicosis exists and where the occupation does not involve free silica. The cost to industry where large awards were made is well known, but it is impossible to estimate the aggregate cost of these frequent, unfounded

claims which are daily presented to the Industrial Accident Commission because of faulty medical opinion when the patient is first seen. Only when the basis for diagnosis, as previously outlined, is adhered to by the practicing physician will this cost be minimized (Figs. 54, 55, 56, 57).

Case XIII.—The details of the clinical aspects of this case will not be given since they are not of essential interest. Suffice it to state that an adult male, who had been employed for a number of years, entered a claim for a permanent disability due to a "dust disease of the lungs." The physical examination did not indicate any disability of pulmonary origin. The *x*-rays revealed an old, healed, left apical tuberculosis, but no evidence of pneumoconiosis.

Fig. 56.

Fig. 57.

Fig. 56.—Advanced silicosis in a retired fifty-nine-year-old male, who had spent twenty-eight years as a gold-miner. He presented himself at this clinic for treatment of arthritis. His only symptom referable to silicosis was that of mild dyspnea following effort.

Fig. 57.—Advanced silicosis in a fifty-seven-year-old male, who had spent thirty years in gold- and copper-mining. Patient complained of mild dyspnea with gradual loss of weight. Tuberculosis not proved to be present. Died of cancer of the bowel.

It was recognized that the process in which the claimant was engaged involved the grinding of materials which did contain silicates, but it was also believed that there existed no exposure to free silica. The operation consisted of grinding the barytes obtained from Battle Mountain, Nevada, and bentonite procured from a district east of Daggett, California.

To the writer's knowledge, these substances had never before been *on trial* as a cause of silicosis. In the years of its existence the defendant company had not been confronted with a similar claim.

Chemistry of Minerals.—A chemical analysis of each follows:

TABLE 14—TYPICAL ANALYSIS OF BARITE GROUND AT LOS ANGELES

	Percentage by Weigh
$BaSO_4$	91.50
$BaCO_3$	0.50
$Fe_2O_3+Al_2O_3$	0.75
SiO_2	6.50
CaO	Trace
Moisture	0.10
	99.35

TABLE 15—TYPICAL ANALYSIS OF HECTOR MINE-RUN BENTONITE

	Percentage by Weight
Silica (SiO_2)	31.50
Alumina (Al_2O_3)	7.21
Iron oxide (Fe_2O_3)	0.11
Titanium dioxide (TiO_2)	0.12
Calcium oxide (CaO)	21.42
Magnesium oxide (MgO)	9.05
Sodium oxide (Na_2O)	6.36
Potassium oxide (K_2O)	0.65
Moisture at 105° C.	2.93
Combined water and volatile	4.07
Carbon dioxide (CO_2)	16.42
Sulfuric anhydride (SO_3)	Trace
	99.84

Barytosis is chiefly mentioned in the foreign literature. Fifty cases of pneumoconiosis among workers in the barite mills in Italy were reported in which the pneumoconiosis presumably did not tend to cause tuberculosis, but rather acute forms of lung affection. The American literature is practically devoid of any mention of barytosis, although Lanza reproduces an *x*-ray film showing diffuse, discrete nodulation in a person who worked in barium rock. In view of this to seek all aid available. Dust counts had already established the fact negligible aid from the literature and because the defendant desired to have some concrete evidence against future claims, it was decided that the atmosphere in which the claimant worked was one in which the concentration of the dust was well within permissible limits.

x-Ray Diffraction Analysis.—It was decided to submit samples of the bentonite and barytes to Ernst A. Hauser, of the Massachusetts Institute of Technology, for *x*-ray diffraction analysis. These samples were: (1) composite Hector mill-run bentonite; (2) com-

posite El Portal mill-run barytes; (3) typical Battle Mountain, Oakland, grind barytes; and (for the purpose of comparison) (4) a quantity of Ottawa sand, which is quite pure quartz. To these, also for comparison, Dr. Hauser added a standard pure quartz from his own stock. At the end of his investigation, he reported no evidence of uncombined silica in the samples submitted except that the El Portal barytes "might be faintly contaminated with uncombined silica, but

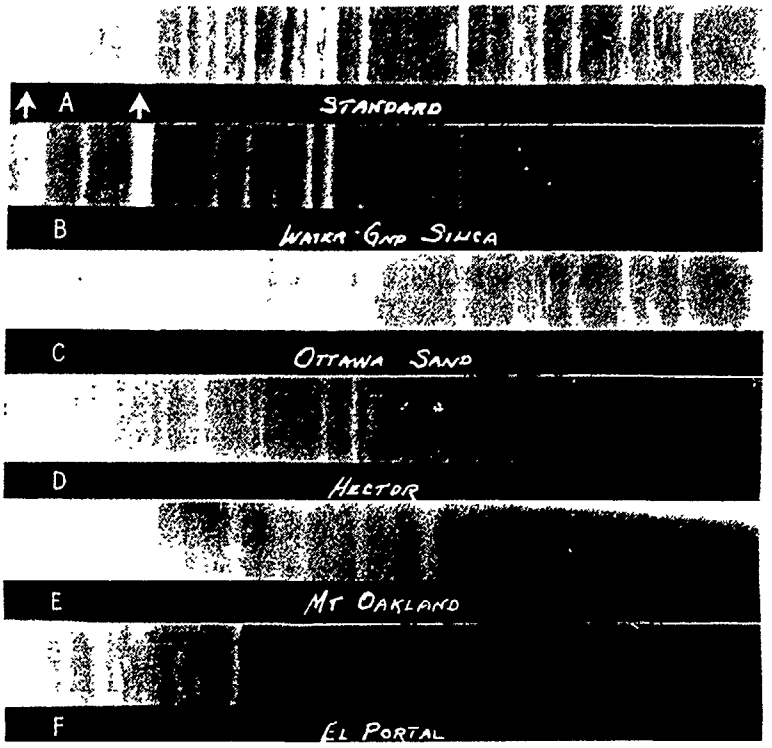


Fig. 58.—*A*, *x*-Ray diffraction of sample quartz used for a standard. Arrows at left of film indicate the heavy silica line. *B*, A ground silica, about 2 μ in diameter, also used as a standard. *C*, Ottawa sand, a silica of average 30 μ diameter. *D*, California magnesium type bentonite. *E*, Nevada Battle Mountain barytes (Oakland grind), —325 mesh. *F*, California barytes, —325 mesh.

according to the pattern, the amount of free silica was not over one per cent." This report of Dr. Hauser's offered substantial evidence of the negligible amount of silica present in this grinding process (Fig. 58).

A scientific description of this method of detecting the crystalline state of solids is not within the scope of this book, nor is the author capable of detailing such a process. If interested, the reader will find an adequate description of this method in *Applied X-Ray*.[19] However,

certain elementary facts deserve mentioning. Prior to the use of *x*-rays, a knowledge of the crystalline state of matter was limited to the observations gained from the appearance of crystals through the use of the microscope and optical goniometers. The external appearance revealed crystals to be either single entities, with pairs of parallel surfaces, disposed in definite geometric fashion at angles which could be measured, or aggregates of these single crystals. But the internal structure of these substances remained a mystery, although external arrangement suggested a similar, regular internal arrangement. *x*-Ray analysis now shows that such a regularity does exist in practically every solid substance. A knowledge of the planes is a prerequisite for the understanding of the structural arrangement of these solids—faces, angles, reflection planes, the unit cell, space groups, space lattices, and many other phases, including the crystal systems, triclinic, monoclinic, orthorhombic, tetragonal, hexagonal, and cubic. On first thought, the *x*-ray analysis of the structure of solids may appear as having extremely limited value, as most of us in medicine have considered the *x*-ray only as an adjunct to the study of pathological processes. Yet industry is using the *x*-ray to determine the strength of steel, the corrosion of alloys, the wearing properties of metals, the covering power of pigments, defects in material such as gas cavities, blowholes, cracks, shrinkage, porosity, and the like.

x-Ray Diffraction Principle.—The principle of the diffraction method is as follows[20]:

When a beam of monochromatic x-rays is directed toward a finely divided crystalline substance, some of these x-rays are diffracted. If these diffracted x-rays come into contact with a photographic film, a definite pattern is produced and a reference point is formed by the undiffracted and unabsorbed x-rays that pass through the material. The diffraction pattern produced is governed by the nature and arrangement of the atoms in the crystalline material; that is, by the crystalline structure.

All evidence so far obtained indicates that no two crystalline substances give the same pattern, which in a sense, is the "fingerprint" of the substance. The x-ray diffraction pattern, therefore, serves as a method of identifying crystalline substances.

According to the technique developed by Clark and Reynolds[21]:

A pure crystalline powder, known not to be present in the dust being examined, is added to the unknown in a definite ratio and the diffraction pattern registered. The ratio of the density of a line sought to that of a nearby line of the added standard is determined protometrically. The ratio thus obtained is proportional to the line intensity of the substance sought, which in turn is proportional to the amount of the substance in the mixture. By reference to a curve that

is prepared empirically from mixtures of known composition, the percentage of the constituent sought is obtainable at once. Fig. 59 shows the pattern for a typical dust sample compared with a standard pattern for ordinary quartz. This method, of course, is applicable to the determination of any selected mineral constituent.

It is to be appreciated by the reader that the problem in our case was to determine whether or not the claimant was subjected to a silicate-free silica mixture, and if free silica was present, to what extent. Some time after our investigation of this case, Louis H. Berkelhamer, of the Saranac Laboratory, reported additional applications of the *x*-ray diffraction method. He compares the values of the spectroscopic, chemical, petrographic, and *x*-ray diffraction methods of analysis in determining the presence of quartz in a given material.

According to Berkelhamer[22]:

Spectroscopy is of little value in this instance. Although it can be employed on very small samples, it demonstrates *only* the presence of the element silicon. By the unaided *chemical analysis* silicon dioxide

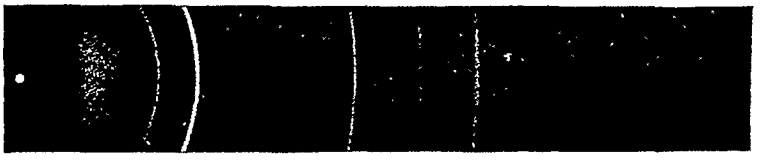


Fig. 59.—Powder diffraction pattern for quartz. (Clarke: *Applied X-ray*, McGraw-Hill Book Co., New York.)

is determined without the qualification as to the original state of the silica. Differential solution methods allow for an empirical estimate of quartz, but variations in particle size of the fine quartz fraction have so much influence upon its rate of solution that the results are not always reliable. A *petrographic* examination permits direct identification of quartz and when combined with a chemical procedure, offers a satisfactory means of determining quartz. Petrographic methods, however, are difficult to apply to particles less than 5 μ in diameter, and it is particles 3 μ and under that are of the greatest pathological significance. In this extremely small particle size range petrographic analysis must give way to *x-ray diffraction*.

Berkelhamer's report is of significant value in that it reveals the application of the various methods of analysis just mentioned to an individual case. A twenty-seven-year-old colored male, who had mined pyrophyllite in North Carolina for twelve years, died and the diagnosis was silicosis resulting from the inhalation of pyrophyllite dust. Petrographic study of hand samples was unsatisfactory since pyrophyllite resembles sericite and talc. (By *x*-ray diffraction, the identifi-

cation was relatively simple.) Chemical analysis of the dried lung was made, and the pulverized lung tissue was subjected to the *x*-ray diffraction test. But most interesting of all was the analysis of a 6-μ tissue section by *x*-ray diffraction, thus identifying the minerals present ". . . in the very section of tissue that the pathologist had studied."

This case is cited to call attention to a recently discovered type of investigation. It is not likely that the *x*-ray diffraction method will be used in the ordinary claim for silicosis, but on occasion the physician, investigator, or medicolegal consultant may wish to utilize it when other methods of investigation are inconclusive.

Treatment.—This follows "The Inert Dusts."

REFERENCES

1. Committee on Pneumoconiosis of the American Public Health Association: *Official Transcript of the Second Silicosis Symposium*, Saranac Lake, N. Y., 1935, p. 146.
2. McCord, C. P.: "Action of Silica as Modified by the Presence of Alkalies," *Indust. Med.*, *5:* 17 (Jan.), 1936.
3. Denny, J. J., Robson, W. D., and Irwin, D.: "Prevention of Silicosis by Metallic Aluminum," *Indust. Med.*, *8:* 134 (April), 1939.
4. Cole, L. G., and Cole, Wm. G.: "The Dyspnea of Silicosis: What Causes It?" *J.A.M.A.*, *113:* 1216–21 (Sept. 23), 1939.
5. Pancoast, H. K., and Pendergrass, E. P.: "The Roentgen Classification of Pneumoconiosis, Based upon Roentgen Appearances, with and without Coexisting Tuberculous Process, and Differential Diagnosis," *J. Indust. Hyg. and Toxicol.*, *16:* 327, 1934.
6. Lanza, A. J.: *Silicosis and Asbestosis*, Oxford University Press, New York, 1938.
7. Gardner, L. U.: "Anthraco-silicosis Simulating Pulmonary Carcinoma," *J. Indust. Hyg. and Toxicol.*, *21:* 193 (Oct.), 1939. (Abstract.)
8. Schnurer, L. J.: "Incidence of Pulmonary and Extra-pulmonary Tuberculosis in Anthracite Coal Miners," *J. Indust. Hyg. and Toxicol.*, *21:* 194, 1939. (Abstract.)
9. Mayer, E.: "Clinical Evaluation of Disability in Pulmonary Disease of Industry," *J.A.M.A.*, *116* (Jan. 11), 1941.
10. Giering, J. F., and Charr, R.: "The Heart in Anthraco-silicosis," *J.A.M.A.*, *113* (Aug. 12), 1939.
11. Shaefer and Company *vs.* Industrial Commission, Wis. 265 N. W. 390.
12. Marsh *vs.* Industrial Accident Commission, 217 Calif. 338, 18 P. (2d), 933.
13. Price *vs.* Industrial Accident Commission, Calif. 49 P. (2d), 294.
14. "Abstract Silicosis; Period of Claim within Which Claim for Compensation Must Be Filed," *J.A.M.A.*, *106:* 1122 (March 28), 1936.
15. North End Foundry Co. *vs.* Industrial Commission, Wis. 258 N. W. 439.
16. Billo *vs.* Allegheny Steel Co., Pa. 195 A. 110.
17. Miller, F. P.: "Examination and Report," Letter to Pacific Employers Insurance Company, Los Angeles, July 19, 1941.
18. Lovelock, J. E.: "Anthraco-silicosis and Bronchial Carcinoma with Quiescent Tuberculosis," *J. Indust. Hyg. and Toxicol.*, *21:* 191 (Oct.), 1939. (Abstract.)
19. Clark, G. L.: *Applied X-Ray*, McGraw-Hill Book Co., New York, 1940.
20. Ballard, J. W., Oshry, H. J., and Schenck, H. H.: *Report of Investigation No. 3520*, U. S. Bureau of Mines.
21. University of Toronto Studies, *Geol. Ser.*, *38:* 13, 1935; *Indust. and Engin. Chem.*, Anal. Ed., *8:* 36, 1936.
22. Berkelhamer, L. H.: "X-Ray Diffraction an Important Tool in Pneumoconiosis Research," *J. Indust. Hyg. and Toxicol.*, *23:* Plate 63 (May), 1941.

CHAPTER XXVII

ANTHRACOSILICOSIS

Most cases of silicosis result from exposure to an atmosphere which contains not pure silica, but rather an admixture with other dusts. Anthracosilicosis is an example of this, and in this country it constitutes a disease confined chiefly to the miners in the hard-coal district of eastern Pennsylvania.

Findings in Governmental Survey.—In May, 1933, at the request of the governor of that state, the United States Public Health Service began a survey of the nature and prevalence of chronic incapacitating miner's asthma. During this study the occupational and medical histories, physical examinations and roentgenological examinations were recorded on 2711 men. At the completion of their work, the following summary and findings were offered.[1]

Definition.—The term "anthracosilicosis" is a descriptive title for the form of pneumoconiosis commonly called "miner's asthma." It is a chronic disease due to breathing air containing dust generated in the various processes involved in the mining and preparation of anthracite coal. It is characterized anatomically by generalized fibrotic changes throughout both lungs with the presence of excessive amounts of carbonaceous and siliceous material, usually by compensating emphysema, and often by cardiac changes in the later stages of the disease.

Symptoms.—The chief subjective symptoms found in the early stages were shortness of breath, cough, and pain in the chest; later, weakness and hemoptysis were frequently mentioned. The most common objective symptoms were dyspnea, prolonged expiration, decreased chest expansion, clubbing of the fingers, change in breath sounds, altered fremitus, and impaired resonance. In the more advanced stages (or in cases complicated by infection), persistent râles, cardiac impairment, loss of weight, and cyanosis were often seen, and usually there was more pulmonary infection, and moderate or marked decreased capacity for work. Lung changes, general in distribution, often with more or less fixation of the diaphragm, were shown by fluoroscopic and *x*-ray-film examination.

Basis of Diagnosis.—Diagnosis was based on three important classes of findings: (*a*) histories, occupational and past medical; (*b*) clinical examination, symptoms, and physical findings; and (*c*) *x*-ray

examination, fluoroscopic and film. For a definite diagnosis of anthracosilicosis, information from all three sources is required. In determining the rôle played by specific infection, laboratory examination should supplement the knowledge gained from the three sources mentioned. In certain borderline cases, repeated examinations may be necessary. A postmortem diagnosis should be based on the results of pathological and chemical examination of the lungs. A complete diagnosis of anthracosilicosis includes a statement of any associated complications, and of the extent and cause of permanent decreased capacity for work, if such is shown.

Of the various contributory factors observed in the development, course, and final effect of the disease, *pulmonary infection* and *cardiac impairment* appeared to be the most important, especially in the advanced stages of the disease.

Groups Studied.—These conclusions in regard to the nature and development of the disease were based on the examination of 2711 men (about 96 per cent of the number on the payroll of three representative anthracite-coal-mining companies studied); on the findings for 135 disabled ex-miners who were hospitalized for observation; and on a smaller group examined in a state tuberculosis sanitarium.

The coal-mining employees were grouped occupationally, largely in accordance with the proportion of free silica found in the dust to which they were exposed.

No cases of anthracosilicosis were found in a control group composed of hard-coal-mining employees whose dust exposure averaged less than 5,000,000 particles per cubic foot of air.

Prevalence and Exposure.—The prevalence of anthracosilicosis among the entire group of employees was found to be about 23 per cent.

Among all except rock workers, less than 2 per cent of the men developed anthracosilicosis when the duration of employment was less than fifteen years, regardless of the amount of dust in the air.

Among men exposed from fifteen to twenty-four years to dust containing less than 5 per cent free silica, 14 per cent of those who had worked where the average dust count was from 100,000,000 to 199,000,000 particles per cubic foot, 29 per cent of those exposed to from 200,000,000 to 299,000,000 particles, and 58 per cent of the men who had worked for this period in more than 300,000,000 particles per cubic foot, developed anthracosilicosis.

Among men employed for more than twenty-five years in dust containing less than 5 per cent free silica, the proportion of persons found with anthracosilicosis under different concentrations of dust was as follows: from 5,000,000 to 99,000,000 particles, 7 per cent;

from 100,000,000 to 199,000,000 particles, 54 per cent; from 200,000,000 to 299,000,000 particles, 71 per cent; 300,000,000 or more particles per cubic foot, 89 per cent.

With the exception of miners, their helpers, and rock workers, about 25 per cent of all the men employed underground developed anthracosilicosis after a working period of more than twenty-five years. This group was exposed to dust having a quartz content of about 13 per cent.

The prevalence of anthracosilicosis among persons who had been exposed for more than two or three years to dust, of which about 35 per cent was free silica, varied from 10 per cent. among those who had worked in concentrations of less than 200,000,000 particles per cubic foot for less than fifteen years, to 92 per cent. among those who had been employed for more than twenty-five years in dust concentrations exceeding 200,000,000 particles per cubic foot, more than two or three years of which time was spent in rock work.

Age *per se* appeared to play a minor rôle in the development of anthracosilicosis.

Safe Limits of Exposure.—Analysis of the data for the purpose of determining safe limits of dust exposure indicated that employment in an atmosphere containing less than 50,000,000 dust particles per cubic foot would produce a negligible number of cases of anthracosilicosis when the quartz content of the dust was less than 5 per cent. In the gangways where the silica content of the dust was about 13 per cent, a safe limit appeared to be from 10,000,000 to 15,000,000 particles per cubic foot. The limit of toleration for rock workers was set tentatively at from 5,000,000 to 10,000,000 particles per cubic foot of air.

Respiratory Disease.—Pulmonary infection increased with length of service more rapidly among the men in the haulageways than in the control group, and much more rapidly among the regular miners. The highest rates of pulmonary infection, however, were found among the rock workers of more than fifteen years' service.

The prevalence of pulmonary tuberculosis among the hard-coal-mining employees at ages below thirty-five was slightly less than that found through studies of tuberculosis among male adults in the general population of the country. In the age-group thirty-five to forty-four, however, the prevalence of tuberculosis was about twice; at ages forty-five to fifty-four, about five times; and for the ages above fifty-five, it was about ten times the rate found in the general population.

The highest prevalence of clinical pulmonary tuberculosis occurred among the rock workers (men who had been employed in rock-loading

or rock-extraction for more than two or three years). After twenty years' service, of which more than two or three were in rock work, 37 per cent presented evidence of pulmonary tuberculosis.

In a group of 135 completely disabled former anthracite workers, which did not include any known cases of tuberculosis, 10 per cent proved positive for pulmonary tuberculosis.

Pulmonary infection (tuberculous and non-tuberculous) was found among 58 per cent of the employed men having early anthracosilicosis, and in 92 per cent of the workers in the more advanced stages.

Clinical pulmonary tuberculosis was diagnosed in 15 per cent of those with early anthracosilicosis, and in 43 per cent of those in the more advanced stages.

In all groups combined, with the exception of the control group, about 20 per cent of the non-tuberculous workers were diagnosed as having some respiratory disease other than tuberculosis. In the control group only 6 per cent had non-tuberculous respiratory disease.

In the control group less than 2 per cent were found with moderate or marked physical impairment causing decreased capacity for work as compared with about 10 per cent among the regular miners, and with approximately 13 per cent among the rock workers. With the exception of the rock workers, no group showed moderate or marked physical impairment in excess of that found among the controls when the period of employment was less than twenty years. However, an excess in the prevalence of slight impairment was found among the regular miners and among others exposed to dust containing less than 5 per cent free silica when they had worked from ten to twenty years in atmospheres containing more than 100,000,000 particles per cubic foot.

The correlations between exposure to dust and the evidence of constitutional changes left little doubt as to the etiological significance of the dust in the air breathed. Like correlations were found between the silica exposure and the extent of pulmonary changes.

Mortality from respiratory diseases was found to be much greater among anthracite workers than in the general adult male population of the country. The data indicated that underground work in the absence of dust did not predispose to fatal attacks of respiratory disease.

Signs and Symptoms.—As in silicosis the first symptom is dyspnea. Initially this dypnea does not require the patients to be in bed, but usually at the time they become short of breath on mild effort, their appearance takes on certain changes, there is a beginning loss of weight, the skin becomes pale or dusky, muscle weakness is noted, and the patients appear asthenic. A cough is common and productive

of a blackish sputum. Because of the pleural adhesions and the fibrous changes causing fixation of the diaphragm or distortion of the mediastinum, pain is a frequent companion to the cough. Pain appears to be more common and seems to occur earlier than in silicosis with infection. Percussion note is impaired, breath sounds may be decreased or show signs of consolidation depending upon underlying pathological conditions.

Pathology. *Gross Appearance.*—The lungs are dark gray, firm, and feel heavy. Black linear markings may be noted on the surfaces of the lungs. Adhesions between the parietal and visceral pleura are fre-

Fig. 60.

Fig. 61.

Fig. 60.—Simple pigmentation of uncomplicated anthracosis. The lungs appear almost normal. (Gardner.)

Fig. 61.—Details of an anthracosilicotic nodule. (Gardner.)

quent. When sectioned these black lines are seen to run through the lungs, and there are also noted the disseminated, black nodules. In severe states, the pleural thickening and adhesions, as well as the fibrosis of the lung proper, tend to distort the shape of the lung. Coalescence of the nodules takes place in this stage, and emphysematous blebs become larger. Consolidation of these areas occurs usually subapically replacing normal tissue (Figs. 60–65).

Microscopic Appearance.—According to Dreesen and Jones,[2] black particles are present in the macrophages, but they are rarely seen free in the alveolar epithelial cells. Collections of "dust cells" are

seen, however, in the perivascular and peribronchial lymphatics and adjacent interstitial tissue. Fibrous hyperplasia is observed quite

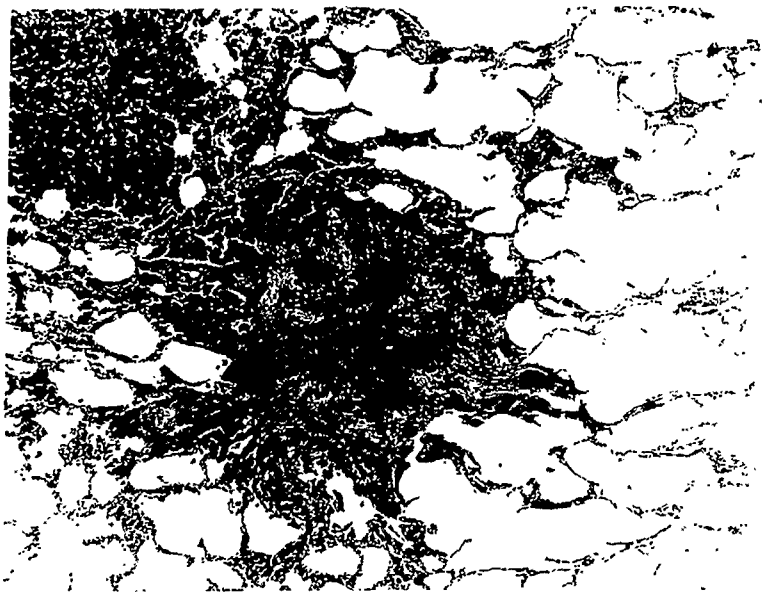


Fig. 62.—Details of an anthracosilicotic nodule with increased pigmentation. (Gardner.)

Fig. 63.

Fig. 64.

Fig. 63.—Conglomerate anthracosilicosis. (Gardner.)
Fig. 64.—Conglomerate anthracosilicosis. (Gardner.)

early along the lymph channels and increases in certain areas to the formation of nodules. In the center of the smaller nodules, evidence of

devascularization may be seen. The centers of larger nodules are for the most part well-formed, white, fibrous connective tissue and commonly reveal evidence of hyaline degeneration but are often free of appreciable amounts of carbon. In the periphery of the nodules, dust-laden macrophages are present. Free extracellular dust is noted in large quantities in the adjacent underlying dense connective tissue.

As confluence of the nodules occurs, the peripheral coal-dust deposits are enclosed in the larger nodule formed. Areas of central, amorphous, finely granular necrosis are often seen. Fibrous hyperplasia and dense depositions of coal dust are seen in the peritracheal and

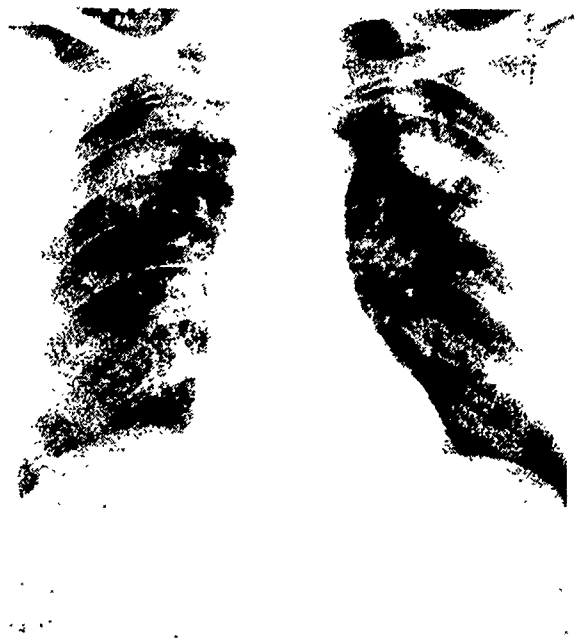


Fig. 65.—Anthracosilicosis proved by autopsy. (McCloskey.)

pulmonary lymph glands, often to such an extent that histology of the organ is completely obscured.

x-Ray Findings.—The first typical appearance is that of an exaggeration of the normal linear markings of the lungs. Following this there is some increase in the general fibrosis. With continued exposure the film takes on a "groundglass" appearance, the linear markings tend to disappear, and the generalized fibrosis is increased. At about this time very fine nodules may be scattered throughout the film. Further progress results in increase in number and size of the nodules. These nodules become larger than those seen in miliary tuberculosis.

and at this time also emphysematous blebs will be noted. Finally, conglomerate masses are much in evidence, the emphysema has increased, and the generalized fibrosis is marked.

Medicolegal Aspects.—The determination of disability and the basis for such a determination correspond to those principles outlined under "Silicosis."

Treatment.—This follows "The Inert Dusts."

REFERENCES

1. *U. S. Public Health Service Bulletin 221*, Dec., 1938.
2. Dreesen, W. C., and Jones, R. R.: "Anthracosilicosis," *J.A.M.A., 107:* 1179 (Oct. 10), 1936.

CHAPTER XXVIII

ASBESTOSIS

The health hazard of asbestos dust has been recognized only recently.

In England, over a number of years, up to 1934, there were only forty-one deaths from asbestosis. There were twenty-six deaths from asbestosis, with silicosis.

As late as February, 1927, the first claim for compensation due to a disability from this disease was filed in Massachusetts. In 1936 Lanza[1] reported that the files of the Metropolitan Life Insurance Company revealed only nineteen cases. He estimated that there were about 10,000 persons exposed to asbestos in this country. It can be seen that this is small in comparison to 500,000 workers exposed to harmful mineral dusts in mining and industry in the United States.

Occupational Hazards.—Asbestos is a hydrated magnesium silicate. The more prominent hazards are offered in mining (especially chrysolite in Canada), handling and crushing crude asbestos, making asbestos mattresses, manufacturing of insulation, the carding and weaving of fine asbestos, and in certain phases of the automobile industry.

Signs and Symptoms.—British investigators feel that the signs and symptoms are more severe in asbestosis than in silicosis, but American investigators believe they are milder. As in silicosis, the first sign is that of dyspnea, the cough is usually dry, and auscultation reveals few, if any, physical signs. Loss of weight is usually noticeable, the color is pale, and cyanosis is a fairly early sign. After some time, fine, crackling râles may be heard, and the chest appears emaciated and lacks the robust character noticed in silicosis. Early asbestosis must be differentiated from acute emphysema. If tuberculosis is present, the symptoms may take on the character of this disease, but this complication is far less frequent than in silicosis.

Pathology. *Pulmonary Changes.*—According to Stewart, as quoted by Auerbach,[2] the pulmonary fibrosis of asbestosis is diffuse in character, peribronchial and basal in location, in contrast to silicosis in which the fibrosis is nodular in character and present in the upper part of the lungs. As the process continues, the fibrosis extends into almost all portions of the lung tissue. Bronchiolectasis and bronchiectasis are present within the substance of the fibroid areas, and bronchopneumonia and acute tracheobronchitis often cause the death of these

patients. The pleurae, especially in the basal regions, are thickened and adherent. In some cases the pleural sac is completely obliterated; in others a fibrous or serous exudate may be present within it (Figs. 66–70).

Action of Asbestos and Silica.—The difference between chrysotile asbestos and quartz silica in their mode of action has caused con-

Fig. 66.—Photomicrographs of asbestosis bodies found in sputum. Enlarged 530 times except for *E*, which is enlarged 310 times. A scale ruled in units of 50 μ has been drawn beside each asbestosis body. (U. S. Public Health Service, *Bulletin 241.*)

siderable speculation as well as experimentation. Gardner[3] particularly, at the Saranac Laboratory, has carried on intensive study of these two materials. He feels that while the action of free silica is chemical in nature, the action of asbestos is mechanical. He states:

Fig. 67.—Right lung, showing markedly thickened pleura with fusion of interlobar pleura. Dense fibrous tags over entire surface. (U. S. Public Health Service, *Bulletin 241.*)

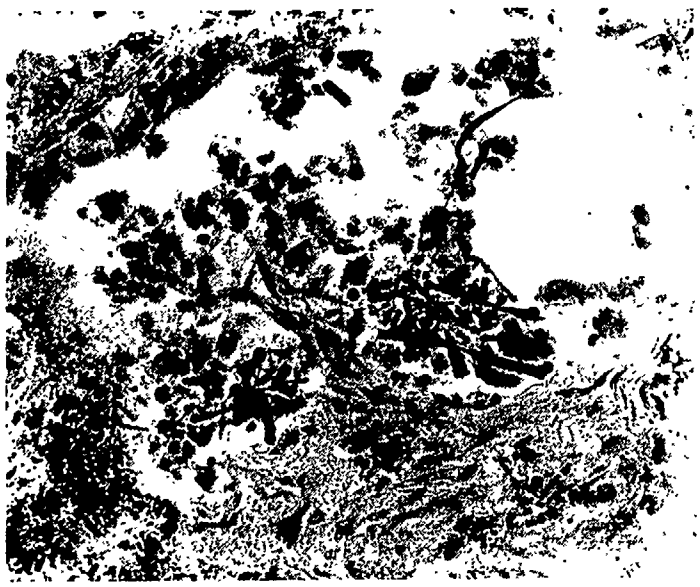


Fig. 68.—Asbestosis bodies accompanied by giant cells. (U. S. Army Medical Museum, Specimen 52255.)

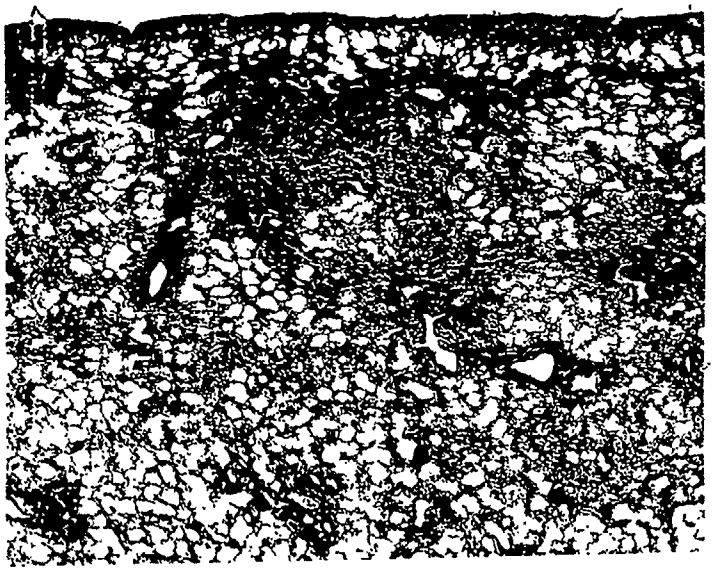


Fig. 69.—Section of a lung in asbestosis. (Gardner.)

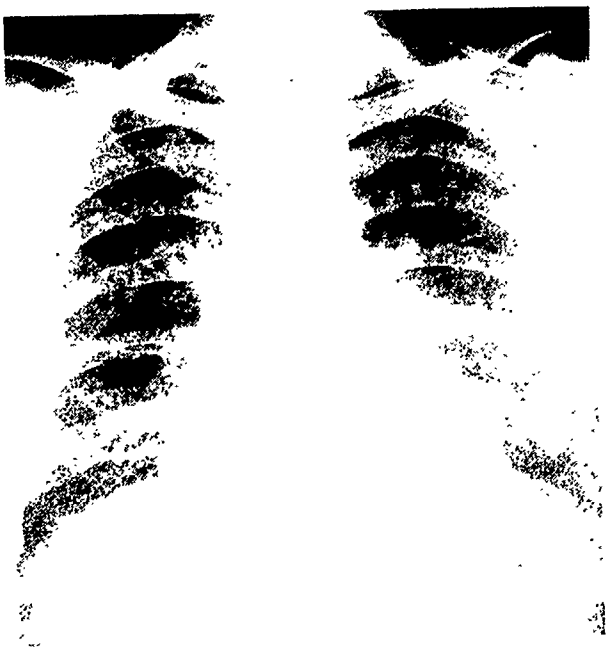


Fig. 70.—*x*-Ray film of a patient with asbestosis. (Pendergrass.)

For the last year or two we have been coming to the conclusion that inhaled asbestos fibres are irritating not because they are silicates but because they are stiff fibres which mechanically irritate the lungs. Unlike the free silicas, these minerals will not stimulate fibroblasts in any part of the body; only those in the lungs are affected. It was inferred that these organs were affected because the movements of respiration are so much more rapid and continuous than those of other viscera. Then it was discovered that if asbestos was ground very finely so that few of the fibres were longer than 2 μ in length the irritating property of the asbestos was practically destroyed. Inhalation experiments with such fine chrysotile asbestos have now been continued for three years. No fibrosis has developed in spite of the fact that an average atmospheric concentration of 125 million particles per cubic foot of air has been maintained. In contrast I would point out that in a previously reported experiment one-third this concentration of long fibre asbestos dust produced well marked fibrosis after about two years.

If the effect of asbestos were chemical, one would expect that a decrease in size would accelerate tissue response. With free silica large particles have little effect, but as their size decreases cellular reaction becomes more vigorous and even constitutional symptoms may ensue. With fibrous asbestos the reverse is true.

The histology of early asbestosis does not suggest a chemical injury. Even under the most extreme conditions that can be created by artificial injection there is no preliminary phase of tissue necrosis with infiltration of leucocytes as occurs with high concentrations of very fine quartz. The connective tissue cells merely multiply very slowly in areas where the asbestos fibres are caught in the bronchioles. As collagen forms and contracts, the air spaces are obliterated by scar tissue. In experimental animals, at least, this change is not a progressive one after cessation of exposure to the dust as is the case in the response to quartz. Perhaps the reason is the deposition of the peculiar iron-containing coating on the surface of the inhaled fibres giving rise to the characteristic "asbestosis bodies."

Finally it is most suggestive that among dozens of different silicate minerals only the five known as asbestos, which are unique because they are fibrous in structure, should be commonly recognized as pulmonary irritants. The variation in chemical composition within this group is greater than that between them and many other silicates. In fact chrysotile asbestos has the same chemical formula as a non-fibrous silicate, serpentine, which is physiologically inert. Obviously irritation would seem to be associated with the physical, rather than the chemical composition of these minerals.

One point of practical significance may be indicated by these observations; namely, that very finely ground asbestos is not dangerous.

This conclusion has support in clinical observation, for it has long been known that at the Thetford mills there was no clinical asbestosis even though in former years the atmosphere was very dusty and the dust was extremely fine. The fact that fabrication of fibres of the same mineral in American plants could produce disease was one of the puzzling features of this disease. But these experiments offer a plausible explanation. The fine dust in the mills is composed of serpentine and extremely short chrysotile fibres; that in the spinning and weaving mills contains many more long fibres.

Microscopic Appearance.—In the early phases of the disease there is a thickening of the alveolar septa as a result of fibroblastic proliferation. The alveolar spaces contain numerous alveolar phagocytes. With progression of the disease, fibrosis becomes more marked, the alveolar structure gradually disappears, and in its place there is now dense fibrous tissue. The few remaining alveoli which lie in the area of fibrous tissue are lined by a low cuboidal epithelium and have a glandular appearance.

Scattered throughout the lung in both the diseased and healthy parts are spindle-shaped structures (first described by McDonald) known as asbestosis bodies. These bodies are slender in their center and bulbous at each extremity. Many are arranged like strings of graduated beads with the largest at the end of the chain.

x-Ray Findings.—Certain distinct peculiarities exist here as contrasted to silicosis. In asbestosis, the lesions may be bilateral or largely unilateral. According to the experience of Pendergrass,[4] the roentgenological findings in asbestosis are largely limited to the lower half or two thirds of the lung fields, while in silicosis the upper portions of the lungs are also involved. In moderately advanced asbestosis, *x*-rays will reveal lessened ventilation of the lung fields, the parietal pleura is thickened, and there is a "groundglass" appearance to the picture. The vascular shadows lose their identity. Nodulation is absent. Pendergrass feels that a roentgenological diagnosis of early asbestosis is unreliable and that the condition must be moderately or markedly advanced to make a differential diagnosis from the roentgenograms.

Medicolegal Aspects.—It is felt that the time necessary to develop asbestosis is on the average from seven to nine years in a fairly high concentration. With a less severe concentration, from fifteen to twenty years. The allowable concentration is 10,000,000 particles per cubic foot of air, of a size between 0.5 and 5 μ.

Once established, the disease is progressive, even after the cessation of exposure. The basis for diagnosis is similar to that stated under "Silicosis."

Treatment.—This follows "The Inert Dusts."

REFERENCES

1. Lanza, A. J.: "Asbestosis," *J.A.M.A.*, *106*: 368 (Feb.), 1936.
2. Auerbach, O.: "Pathology of Asbestosis," *Indust. Med.*, *6*: 318 (Jan.), 1937.
3. Gardner, L. U.: "Recent Developments in Relation to Silicosis," *Indust. Med.*, *9*: 45 (Feb.), 1940.
4. Pendergrass, E. P.: *Silicosis and Asbestosis*, Oxford University Press, New York, 1938.

CHAPTER XXIX

THE INERT DUSTS

How deleterious are the various industrial dusts? Ill health as a result of working in a dusty atmosphere is claimed by a sufficient number of workers to make this a problem of frequent incidence in any industrial medical practice. Usually such claims arise from trades where the dust particles are large and plainly visible. It is not difficult to understand that men laboring in such circumstances easily imagine all types of harm to the lungs from breathing such dust. Nor has the pathological picture of the inert dusts been sufficiently clarified in the various textbooks to permit the average physician an adequate understanding of their status.

Effects of Inert Dusts.—To date there is no evidence that the inert dusts are relatively harmful. This does not mean that breathing heavy concentrations of any dust may not have an irritating effect upon the upper respiratory passages. Nor is it to be implied that continuous exposure to a moderately severe or severe concentration of dust may not produce a mild fibrosis. But this reaction is very slow, noticeable by *x*-ray only after a number of years of continuous exposure and rarely productive of any symptoms. After many years of exposure, dyspnea may be present to mild degrees, but this is not often disabling. Furthermore, to attribute this dyspnea to an existing fibrosis, one would have to rule out unrelated cardiac pathology.

Relation to Tuberculosis.—It has long been recognized that the tuberculosis death rate among certain industrial groups exceeds that of the general population. To evaluate this increase, many factors must be considered, such as overcrowding of workers in the shops and plants, thus favoring the spread of tuberculosis; poor ventilation at work as well as in overcrowded homes; faulty diet; ignorance of general hygiene and the like. Tuberculosis is not to be considered a sequela of exposure to inert dusts *per se*. This point cannot be too strongly emphasized.

Supportive evidence of the above statements may be found in several investigations. Clark[1] studied men subjected to the effects of inhalation of abrasive substances for a period of twenty-five years. He found that in factories which provide for proper dust removal, the continuous inhalation of abrasive dust (chiefly aluminum oxide) over a period of years did not produce symptoms or *x*-ray findings of

crippling lung fibrosis, that tuberculosis in these workers was not greater than in the community, and that the risk was slight.

Some of the common inert dusts will be mentioned briefly with enlargement upon several others:

Talc.—Talc powder creates the impression that it is so dense that it must of necessity cause disturbance within those who breathe it. While talc is a mineral often occurring with asbestos or near it in nature, there is no evidence that it causes any undue fibrosis. This observation is based upon the facts that exposure to talc powder is not a new hazard and that there has been sufficient evidence gathered to confirm the above statement.

Calcium and Magnesium Carbonates.—From these compounds come limestone rock, marble, chalk, calcined magnesium used for insulations, furnace linings, and the like. Men have been exposed to these dusts for years, but there is no evidence that they are harmful to the lungs.

Carbon Dust.—This dust is a common constituent of the air in any industrial district where anthracite or bituminous coal is used. Exposure causes black pigmentation of the lungs, noted in city-dwellers even though they have no specific or intense exposure. Miners reveal a fibrosis greater than that of the non-exposed, but it is a non-disabling fibrosis, unless the carbonaceous substance is combined with siliceous material.

Iron Dust.—Exposure to excessive amounts of iron dust is confined chiefly to the mining of iron ore. Inhalation of this substance may cause a mottling or staining of the lungs, simulating silicosis to a degree. This condition has been called "siderosis." This is a non-disabling condition.

Tobacco Dust.—Tobacco dust has been referred to in the literature of the past as a cause of pulmonary disease. In reviewing this literature it is interesting to note that practically all, if not all, of these reports were made without the benefit of *x*-ray. Some of them were purely assumption, such as the observation that tuberculosis was greater in tobacco workers of Baden than in the general population, and it was, therefore, concluded by Stephani that the tobacco dust was responsible. The probability of faulty hygiene, poor working conditions, long hours of labor, etc., was overlooked. Thus there crept into the literature a condemnation of a dust not proved to be an actual offender. Long,[2] after studying a group of tobacco workers over a period of ten years, did not once see the occurrence of this disease. He studied 2254 persons, of whom 1246 had been exposed to tobacco dust while working. By comparison of the findings of these two groups it

was concluded that pneumoconiosis (tabacosis) does not develop in tobacco workers. The value in his observation lies in the *x*-ray and fluoroscopic revelations as compared to the absence of these in other reports.

REFERENCES

1. Clark, W. L.: "Dust Hazard in the Abrasive Industries," *J. Indust. Hyg.*, *7:* 345, 1925; *11:* 92, 1939; *13:* 343, 1931.
2. Long, C. F.: "Tobacco Dust and the Human Lung," *Indust. Med.*, *8:* 365 (Sept.), 1939.

CEMENT DUST

Probably the most informative and extensive survey and one which should, for the present time, present a picture of the actual status of inert dusts is the one conducted by the Saranac Laboratory at the request of the Portland Cement Company. This study was begun in 1935, but was not completely reported until September, 1939.[1]

Table 16 and the following comments are based upon that survey.

Chemistry.—The essential ingredients of Portland cement are CaO, SiO_2, Al_2O_3, and Fe_2O_3. The chief raw materials which supply these constituents are limestone to furnish the CaO, and shale to supply the other ingredients. An analysis of Portland cement reveals that it contains 19.86 per cent of SiO_2, yet almost none of this silica exists in a free state. Complete studies were made in eleven plants involving 2278 employees. Of these 299 worked in a dust-free atmosphere.

Clinical Findings.—Complete physical examinations of 2278 employees in eleven different plants situated in widely scattered parts of the country have demonstrated nothing even suggestive of a pneumoconiosis, nor has any other condition common to a majority of cement workers been disclosed. Stereoroentgenograms of the same group have revealed certain variations of the usual pulmonary shadows. The same abnormalities also occur in the films of other industrial groups and are in no sense specific for these.

Pulmonary Roentgenograms. *Classification.*—The roentgenographic interpretations have been classified in two categories: linear exaggerations and nodulation.

(*a*) P_1 *Shadows.*—In the first, the normal shadows, cast largely by the pulmonary blood vessels, are unusually prominent. The slightest degree of exaggeration designated as P_1 is so ill-defined that had not all the films been read by one observer there would be little point in discussing them. They will be considered here, not because they have any clinical significance, but because of a tendency among certain roentgenologists to attribute all possible variations from the usual

pulmonary pattern to a dusty occupation. It should be clearly understood that changes of this intensity are not indicative of a disease process and that they may be produced by a variety of causes whose number increases with advancing age.

(*b*) *P_2 Shadows.*—A more marked accentuation of the linear shadows in the pulmonary roentgenogram, designated for purposes of classification by the symbol P_2, is sufficiently unusual that all observers recognize it, although there are borderline cases, difficult to differentiate from exaggeration of lesser intensity. The pathological reaction responsible for the heavier vascular shadows may again be due to different causes, but in groups of men employed in dusty atmospheres, the inhaled material is much more likely to have produced the change. As a general rule, second-degree linear exaggeration (P_2) is not associated with clinical symptoms. Exceptions have been reported by some observers in patients exposed for prolonged periods to certain types of dust, particularly those containing some free silica, but there is always a question of the causal relationship between this type of pulmonary reaction and the clinical findings. In the experience of the Saranac Laboratory, however, symptoms in men whose *x*-rays show this reaction should be attributed to associated conditions like heart disease or obvious bronchial infection.

(*c*) *P_3 Shadows.*—A still more marked exaggeration of the usual branching shadow pattern has been provisionally designated as P_3. The condition is rare, and its cause is not definitely known. The shadows of the peripheral branches of the vascular tree are so heavy that they lose definition and tend to fuse. It is suspected that complicating infection may play a part in producing such appearances.

Roentgenographic Findings.—For purposes of analysis the employees were separated into two groups—the first comprising 1979 exposed to more or less dust in occupation (designated "exposed"), and the second, 299 office workers, laboratory employees, machinists, watchmen, and others not often in contact with cement-plant dusts (designated "control").

The roentgenographic findings in the larger group exposed to dusts of various kinds in all parts of the plant are shown in Table 16.

Perhaps the most striking feature of this table is the fact that 82.06 per cent of the films showed no abnormality of any kind (Figs. 71, 72). The changes that were noted in this group include 17.45 per cent classified as linear exaggeration of one degree or another, and 0.40 per cent read as silicotic nodulation. The eight cases in the latter category were apparently due to exposures before entering this industry. They are of little interest in this study.

TABLE 16—SUMMARY OF ROENTGENOGRAPHIC FINDINGS BY LENGTH OF SERVICE—EXPOSED GROUP

Years	Number	0		P_1*		P_2		P_3		S_1†		S_2		S_3	
		Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
Under 5	503	454	90 26	42	8 32	6	1 19			1	0 20				
5–9	379	316	83 38	53	14 25	6	1 58			1	0 26	1	0 26	1	0 26
10–14	469	380	82 31	73	15 57	6	1 28	1	0 21	1	0 21	2	0 43		
15–19	271	217	80 07	44	16 23	9	3 32	1	0 37						
20–24	141	97	68 79	34	24 11	10	7 09								
25–29	106	74	69 81	24	22 64	8	7 55								
30–34	56	39	69 64	15	26 79	2	3 57								
35–39	26	19	73 08	5	19 23	1	3 85			1	3 85				
40–44	10	7	70 00	2	20 00	1	10 00								
45–49	9	9	100 00												
50–54	6	4	66 67	2	33 33										
55–59	3	2	66 67			1	33 33								
Total	1979	1624	82 06	295	14 91	50	2 53	2	0 10	3	0 15	4	0 20	1	0 05

* P_1, P_2, P_3 indicate the degree of linear exaggerations.
† S_1, S_2, S_3 indicate the degree of nodulation exaggerations.

Subdividing the cases of linear exaggeration according to the intensity of the reaction, it was noted that 14.91 per cent were read as P_1 and 2.53 per cent as P_2. There were also two cases (0.10 per cent) of the rare P_3 type of change. These figures present a well-marked contrast to those for the group of hard-rock miners, whose films were interpreted by the same observer. In them the total incidence of linear exaggeration was nearly three times as great: P_1 readings occurred in 32.33 per cent of the films. Many of these miners had been exposed to considerable amounts of free silica, whose capacity to produce pulmonary reaction needs no comment.

Since the amount of well-marked linear exaggeration in the cement workers was so very small, there was a reasonable doubt as to whether any of it was produced by the dust that they had inhaled in this industry. Many of them had also worked in other places where they were exposed to dusts of different kinds. To throw more light on this question a group of 274 plant employees was selected whose histories stated, or whose age in relation to length of service indicated, that they had never worked outside the cement industry, even on farms. None but persons likely to have been exposed to cement dust or its components were included.

Of this group, 70 per cent had been employed for periods varying from ten to fifty-five years. The films of 19 (6.93 per cent) were read as P_1, and 5 (1.82 per cent) as P_2. In other words, among those exposed only to cement-plant dusts the percentage of first-degree linear exaggeration was about half that for the group as a whole; and the percentage of second-degree reactions was three-fourths as great as

in the entire group. This comparison emphasizes the non-specific nature of the linear type of change and indicates that, in the group as a whole, causes other than inhaled cement-plant dusts contributed to its production.

The data have been analyzed further to demonstrate the incidence of different types of roentgenographic change in relation to length of employment, to age, to geographic distribution of plants, and to concentration and composition of dust.

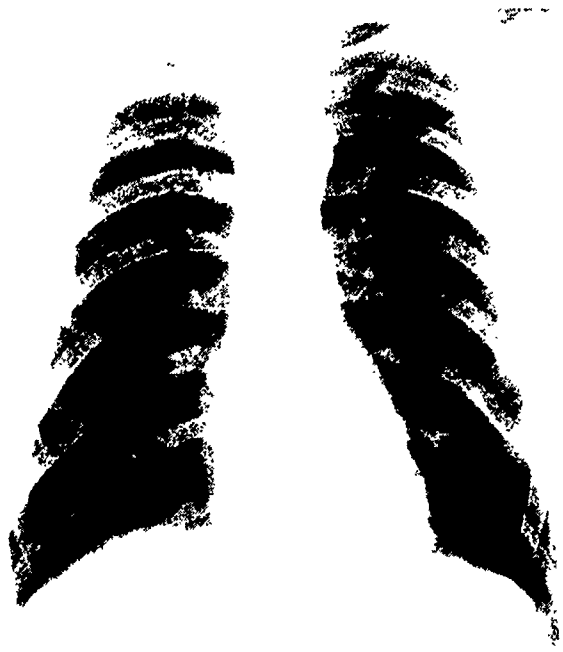


Fig. 71.—Film of a thirty-five-year-old male who worked in cement dust for twenty years. (A. J. R. Curtis, of Portland Cement Co., and Drs. Gardner and Sampson, of Trudeau Sanatorium.)

In this report several of the few cases of nodulation were found in southern California. During a visit of Mr. Curtis, of the Portland Cement Company, to Los Angeles, he described these cases and showed the *x*-ray films. It is rather surprising that, in the long period these southern California plants have operated, more cases of nodulation have not been acquired from outside. Cement rock is often so located in southern California that it has to be taken out by methods familiar to the mining industry rather than the typical quarrying

processes used in the East. Consequently such help is drawn to a considerable extent from the hard-rock-mining industries of the state, in which there is in many instances a pronounced silica exposure. Yet no case exhibited other than moderate nodulation disclosed by the roentgenogram.

(*a*) *Length of Exposure.*—The table reveals a steady increase in the percentage of P_1 readings in each successive five-year period of

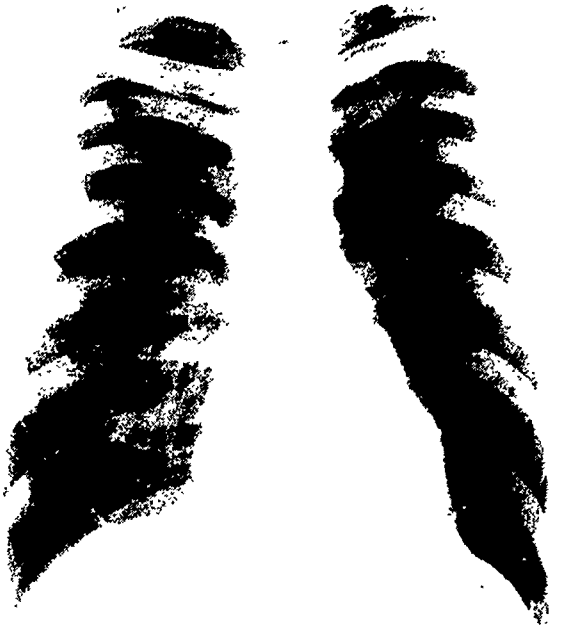


Fig. 72.—Film of a fifty-five-year-old male who worked for over thirty years in cement dust. The linear markings are comparable to one of this age not engaged in a dusty trade. This and Fig. 71 serve to illustrate the relatively minor extent of fibrosis resulting from exposure to the inert dusts. (A. J. R. Curtis, of Portland Cement Co., and Drs. Gardner and Sampson, of Trudeau Sanatorium.)

employment, starting at 8.35 per cent and reaching a value of 26.79 per cent for men working from thirty to thirty-four years. Then the figures become irregular, but probably not altogether because of the small number of men in the groups. There were 54 employed for from thirty-five to fifty-nine years, inclusive, of whom 9 (16.66 per cent) showed first-degree linear exaggeration (P_1).

The less frequent second-degree linear exaggeration (P_2) also increases with duration of employment, but it reaches its greatest fre-

quency five years earlier than the P_1 reaction and then occurs occasionally. It is manifested in only 4.5 per cent of the entire group of 110 working for more than thirty years. This lack of correlation between length of exposure and the degree of reaction again suggests an influence of factors other than dust.

(*b*) *Dust Exposure and Respiratory Infection.*—This group quite thoroughly investigated the relationship between dust exposure and tuberculosis, bronchitis, and pneumonia, and their study indicated that the incidence of tuberculosis and other pulmonary infections is as low as or lower than that in the general population. There is nothing to suggest that prolonged inhalation of cement dust has altered natural susceptibility to tuberculosis. If this is true of cement dust, it is probably true of other types of inert materials. They likewise found no significant effect of this inert dust on old tuberculosis. It was also noted that the incidence of bronchitis and pneumonia in those working in a cement dust varied, as did the figures for the general community where the various plants were located. The reports of a low incidence of bronchitis in the dry sections of Texas or Kansas, as contrasted with the high incidence in one of the dampest parts of the state of New York, suggested a marked climatic influence. Absenteeism due to upper respiratory infections such as coryza was somewhat greater than that in the general community.

It is the belief of the author that the above findings regarding a well-known dusty trade should aid in the prevention of too hasty and ill-founded conclusions regarding the action of inert dusts. Details have been given because much of this information is not found in the general literature available to the physician. Cotton and mill dusts are likewise subsequently presented for this same reason.

Illustrative Cases:

Case I.—A man was employed as a still-cleaner by a petroleum corporation. The work of a cleaner was to rake down coke, burning or smouldering, in stills that escaped the mechanical rake, and sweep it out. The breaking up of these coke deposits gave off dust and a trace of gases. The man worked steadily in that capacity for about ten years, at the expiration of which time he became ill and died. His physician diagnosed his condition as pneumoconiosis. The widow of the workman, as the administratrix of his estate, brought a common-law action against the petroleum corporation for the workman's death, alleging that the corporation failed to provide him with a reasonably safe place in which to work and reasonably safe appliances with which to work, and that it failed to warn or to instruct him with respect to the dangers incident to the work. The trial court, at the close of the plaintiff's evidence, directed a verdict for the defendant, and the

plaintiff appealed to the United States Circuit Court of Appeals, Tenth Circuit.

Among other reasons for directing a verdict, the trial court was of the opinion that there was no substantial proof that death resulted from the workman's work. The proof was most unsatisfactory, said the Circuit Court of Appeals. Conceding that death resulted from pneumoconiosis, the attending physician's opinion that it resulted from the work was formed, he testified, because he could find no other reason for it. However, said the court, pneumoconiosis concededly results from deposits of foreign material, and the man did work in a dust-laden atmosphere. That the physicians who testified never knew of another "still worker" becoming so affected, and that the medical books contain reference to no other case, merely goes to the weight of the evidence. In the opinion of the appellate court, the evidence was sufficient to justify a submission of the question to the jury. If the man died of pneumoconiosis, and if his occupation had anything to do with it, it was because of the dust in which he worked. He knew the dust was present when he took the job, and he kept it year after year. He knew it was harmful to the lungs, for he was furnished with a respirator and told to wear it. While assuming the ordinary, known risks attendant on inhaling dust-laden air, he did not assume the risk of any concealed dangers lurking in the dust. The plaintiff contended that the presence of carbon monoxide and hydrogen sulfide in the stills contributed to the pneumoconiosis. Any finding by a jury, said the court, that hydrogen sulfide or carbon monoxide had anything to do with the pneumoconiosis would rest on speculation. There was nothing in the record to warrant a finding that, after the forced ventilation, there remained in the stills a sufficient amount of the gases to do injury. There was, furthermore, no evidence of acute or chronic poisoning of still workers due to either gas.

The petroleum corporation, continued the court, was engaged in the refining of oil, a business attended with some danger as are nearly all constructive undertakings. Its equipment was standard and not defective. It followed the approved practices. It did all that was then known to minimize the risk to its employees. It operated year after year with no occupational disease resulting from the work. It had no reason to suspect any such. "Tested by the principles of fault which underlie recovery for injuries at common law," concluded the court, "we do not believe that the plaintiff has established her case." The judgment of the trial court was consequently affirmed.[2]

In this case three contentions are made but not verified; (1) there was failure to provide a reasonably safe place to work; (2) death resulted from pneumoconiosis; and (3) carbon monoxide and hydrogen sulfide gas contributed to the death.

The first contention requires the decision of an industrial hygienist; the second and third, an opinion from a physician based upon knowledge and proof—not assumption. This case illustrates the need for a better understanding of the effects of dust upon the lung, since the dusty trades comprise the largest group exposing the workman to any one material.

Case II.—Two longshoremen from the Los Angeles Harbor presented themselves to this clinic within an hour of each other with the allegation that recent handling of borax had produced severe chest pain, cough, shortness of breath, and weakness. The histories obtained from each were practically identical and were as follows:

On May 15, 1940, together with other men, they were engaged in loading bags of borax to be shipped to England. Patient A stated that on the evening of May 16 he developed shortness of breath, a feeling of oppression in his chest and a cough. He was so "all in" that he was unable to report to the company physician until May 18. Patient B gave an identical history but did not report to the same physician until May 19.

Physical examination of A revealed obvious dyspnea upon inspection. The chest was barrel-shaped and inspiratory excursion bilaterally limited. Auscultation revealed widely scattered musical râles at the beginning of expiration. The rest of the examination was entirely negative. The patient denied ever having had asthma, to his knowledge. The examination of B was entirely negative. The lungs were clear throughout, temperature normal, and no evidence of inflammatory reaction of the nose and throat.

These two claims puzzled the writer. For years this clinic has handled the workmen of the borax company involved. All of these cases have been injuries—the traumata arising out of employment. No claims had ever been presented, at least during the writer's tenure with this clinic, alleging a disturbance of the lungs from occupation. Did this particular shipment of borax, owing to war exigencies, contain an added ingredient that might have caused an allergic response? I immediately contacted Mr. G. A. Connell, Technical Director for the Pacific Coast Borax Company. His reply was as follows:

Dear Dr. Johnstone:

I am in receipt of your letter of May 22nd, regarding the complaints of men loading Borax on a shipment made May 15th. I have examined our records and find that on May 15th we shipped to England a parcel of bags containing what we term Calcined Rasorite, at Wharf 90. I have examined our analysis of this parcel and find it to be almost exactly similar to all other shipments of this nature. We manufacture at our Mines at Boron, California, several hundred tons of this material

a week and to date know of no complaints from our men working with this material or handling the same after it has been bagged. As a matter of fact, investigations made by the Insurance Commission and the Mining Bureau of the State have given us a clean bill of health in regard to our products, although the dust condition obviously should be worse than that in loading ships with this material after it has been bagged. We have also had occasion to investigate the effects of borax from an allergic standpoint and tests conducted under a very competent physician indicate that while some people may be allergic to borax, the evidence indicated that this allergy amounted to only about one out of one thousand people.

This information indicated the unlikelihood of borax as the cause of the alleged complaints, but it was felt that investigation should not stop at that point. It must be appreciated that one of these patients presented a condition of bronchial asthma. The possibility of a claim for frequent temporary disability or an eventful permanent disability due to the initial onset following the exposure to this dust presented itself. An investigation in the patient's community brought out the following facts. Patient A had been under treatment with a family physician for asthma prior to this contact with borax. A longshoreman strike was impending, and it was thought that a layoff was due. So, if these two could be on compensation during that time, they would benefit financially. A drugstore clerk testified that A had met B at the fountain and told him he had another attack of asthma and was going to "fool the doctor" by blaming it on this dust. B thought that was a good idea and asked A just how he felt. The conclusions finally reached by this examiner were that A was suffering from an attack of asthma, which he had experienced before, and that his attack was coincidental with, but not incidental to, the handling of borax. The negative examination of B and subsequently determined facts led us to believe that he was faking.

This illustration offers certain lessons that should be emphasized. In the first place, before a claim is denied where doubt exists, it should be investigated thoroughly. Any doubt in compensation cases is to be given in favor of the workman. If a doubt exists, thorough search to substantiate or deny the claim should be instituted. Snap judgment is unfair to all parties. Time and effort must be expended to obtain facts. When the general practitioner is faced with doubt regarding the cause of his patient's illness, he resorts to his experience and to his textbooks. But the industrial physician is required to go beyond that, to seek into conditions of employment, to examine the plant by personal inspection or by submitting material for analysis. The willingness to do this qualifies the industrial physician as just and fair in his deci-

sions. Both patients in the above illustration were denied compensation.

REFERENCES

1. Gardner, L. U., Durkan, T. M., Brumfiel, D. M., and Sampson, H. L.: "Survey of Twenty-Two Hundred Cement Workers," *Indust. Hyg. and Toxicol.*, 21: 7 (Sept.), 1939.
2. Grammer vs. Mid-Continent Petroleum Corporation, Texas 71 F (2d) 38.

COTTON DUST

Occupational Hazard.—The cotton industry is an important field of employment in this country. But if this work provides any risk to the workers' health it is not indicated in the American medical literature. One case which presented itself aroused the author's interest, and he believes that his study of this case might be of interest to others who may, on occasion, be confronted with the problem of determining the effect of cotton dust upon the lungs of those exposed. The bulk of comment is to be found in the English literature. In America it is classified as an inert substance, and little thought has been given to its effect, if any, upon the lung. In a report from the *English Encyclopedia of Hygiene, Pathology, and Social Welfare* (1930), cotton workers' pneumoconiosis (byssinosis) is classified as one of the chief affections of the lungs among the workmen. Possibly their method of sorting, carding, and handling this substance differs from ours, and so in our system there may exist little or no hazard. Nevertheless, there are bound to be occasions when this question will present itself to the practitioner in our cotton-growing states.

Signs and Symptoms. *Respiratory Effects.*—According to the English investigators, the effects produced by breathing an atmosphere heavily impregnated with cotton dust are characteristic and indicate a severe irritation of the air passages. Dyspnea is a prominent symptom, but it is dyspnea of an undoubtedly asthmatic type. It is different from the dyspnea of silicosis, which involves the abdominal efforts and not the marked thoracic efforts of those affected by cotton dust. The cough is dry with little expectoration. Whatever sputum is produced is of an extremely tenacious character. The attacks tend to disappear over the week-end or during a vacation, but reappear with the resumption of work. Chronic asthma eventually results in loss of weight, increased dyspnea, emphysema, and the development of the barrel-type chest. From reading the English literature it can be assumed that it takes from twenty to twenty-five years for the development of severe symptoms. It is also noted that the coarser grades of cotton contain more dust than the finer varieties and, therefore, constitute a greater hazard. In addition to the asthma and bronchitis, catarrh of the upper respiratory passages is common.

Allergic Phenomena.—These have been reported by the English investigators as well as by Teleky in Vienna, and Schmidt in Germany. Weavers do not appear to be affected by this allergic reaction as much as those who prepare the material. In the latter group irritation of the upper respiratory passages, a sensation of oppression of the chest, and attacks of coughing with scanty sputum are the common complaints.

Fibrosis.—Autopsies on cotton-strippers and grinders fail to reveal pulmonary fibrosis. It is thought by most investigators that any fibrosis developed by such cotton workers is the result of a combination of bronchitis and asthma and not of the cotton dust itself. In England the Home Office Departmental Committee defined byssinosis as a chronic bronchitis complicated by emphysema in its later stages and by asthma in some cases. It is felt by these investigators that the combination of asthma and chronic bronchitis is due to the action of the protein fraction of the cotton dust inhaled.

Non-Pulmonary Conditions.—Certain non-pulmonary conditions may exist in the industry. Workers in the processing rooms may develop conjunctivitis or blepharitis (from the burned products of the gassing of double yarn), headaches, lassitude, and loss of appetite (from the high temperatures), in addition to the possible effects of carbon monoxide.

Molds and Fungi as Factors.—A comment by Collis[1] particularly interested the author in view of the *x*-ray findings in the case he is about to report. After investigating the cotton mills of England, Collis concluded that the occasional severe epidemics of coughing among these workers were due to mildew and molds and that these molds were Penicillium glaucum or Aspergillus glaucus. Others have indicated a relationship between the chest conditions and fungus. The British statistics show a high incidence of upper respiratory diseases in persons engaged in the preliminary operations such as balers, carders, or sorters. Our patient was a sorter for twenty-eight years.

Roentgenographic Studies.—It was disappointing in searching the American and British literature to find no report of *x*-ray studies of cotton workers. Dr. R. R. Sayers, Senior Surgeon of the United States Public Health Service, Division of Industrial Hygiene, was appealed to, and he graciously aided in supplying references to the foreign literature but could suggest no source of help in this country. Our films were submitted to certain authorities in America on pneumoconiosis and various interpretations were received.

Illustrative Case.—It was because of the history of exposure, symptoms, and the peculiar *x*-ray findings that this case was of interest.

Case I.—In May, 1939, a forty-year-old Negro male presented himself to the writer with a complaint of attacks of nocturnal dyspnea.

History.—Since the age of twelve this patient had worked as a cotton-sampler. He had not worked in the cotton mills or been connected with any other processing phase of the cotton industry; his activities had been confined to the handling of cotton for inspection. The only time he was not exposed to this was during a brief sojourn in the army during the first World War. Until 1918 his work was done in the South, and since that time he had been engaged as a cotton-sampler in Los Angeles.

About one year previous to this examination he noticed a sensation which he described as a dull weight or oppression between his shoulder blades. He claimed that this was not a pain and never had felt like a pain, but was simply a heavy sensation in the upper posterior chest.

Six months after noticing this heavy feeling he began having nocturnal attacks of shortness of breath. He described these attacks as follows:

I do not have any trouble during the day or while I am at work. I am all right until after I have been asleep for a while. I usually fall asleep without difficulty, but about an hour later or sometimes less, I am awakened by a wheezing sensation in my chest. I then start coughing and get very short of breath. I feel as if my air is being cut off, and I have to get up. When I get up I usually spit up a heavy, thick, sticky substance, and then I start to feel better. I never notice any of this whistling or wheezing in my chest during the day and I rarely cough during the day. I do not have any shortness of breath except if I walk about three blocks of if I climb a flight of stairs in a hurry.

Symptoms Referable to the Various Systems.—Essentially negative.

Past Medical History.—Entirely negative with the exception of gonorrhea contracted while in the army, at which time he also developed a sore at the end of his penis to which he gave the familiar term of "hair cut."

Physical Examination.—Patient is an adult Negro male; height, 5 feet 8 inches; weight, 191 pounds. He is forty years of age. His intelligence is above the normal, for his race.

The pupils reacted to light and accommodation; the eyegrounds were normal. Throat normal, tonsils atrophied. Teeth appear to be in a good state of hygiene. Neck short, bull-neck type.

Chest barrel-shaped and measured 41 inches. Following full inspiration it measured 42 inches. There was a moderate limitation of the normal excursion rate bilaterally. There was no noticeable lag on one side over that of the other. Percussion note was slightly duller than normal. Auscultation of the lungs revealed the breath sounds

over the entire chest to be quite distant, almost imperceptible. In spite of the history of wheezing and whistling sounds, we were unable to detect any râles during three separate examinations of this patient's chest. Heart rate of 84, sounds distant, no murmurs heard. Blood pressure: right arm, 128/90; left arm, 130/90.

Abdomen obese; no organs or masses palpated. Genitalia and rectal examination negative.

Neurological Examination.—Pupils reacted as noted. Knee jerks, 2 plus. Achilles reflexes, positive bilaterally. Romberg sign, negative. Finger-to-nose test, negative. Babinski test, negative.

Laboratory Examination.—Urinalysis, negative. Blood count, normal. Blood Wassermann, 4 plus. Kahn, 4 plus. Repeated sputum specimen, moderate number of white blood cells; ordinary bacterial flora; acid-fast bacilli not found. No fungi found. Guinea-pig, negative for tuberculosis.

This case presented the following similarities to the English cases:

1. Length of exposure. Twenty to twenty-five years is the time interval they have observed before the pronouncement of severe symptoms. Our patient had an exposure of twenty-eight years.
2. The symptoms. Cough with scanty but tenacious sputum. (The laboratory technician had to cut specimens in this instance with scissors for material for slides.) Oppression in the chest; this was one of the first things noted by our patient. Dyspnea at night with the presence of râles. Symptoms are more marked in those who handle the raw cotton. (Our patient was a cotton-sorter.)
3. The development of emphysema and barrel-type chest.
4. The possible infestation of fungus. This question had to be considered in our case.

Roentgenographic Interpretation.—The chief problem was to evaluate the chest plates. These were so strikingly unusual and outside the experience of several clinicians and roentgenologists who viewed them that they were mailed along with the history to several nationally known authorities for their comment. In the base of both lung fields, especially on the left, there were concentric whirlpools as if made by interlacing white threads. These films in stereo were interpreted by our roentgenologist as follows:

x-Ray examination; stereo of the chest (Fig. 73). There is a definite area of increased radiability along the upper and lateral wall of the left lung field, consistent with a partial pneumothorax or coalition of several large emphysematous blebs. On the right side there is also noted near the apex and along the upper lateral wall an area of

increased radiability suggestive of a similar condition. The low, flat diaphragm, together with a rather enlarged chest, bears out the picture of general emphysema. There is increased fibrosis around both lung fields near the base of the heart on the left side, but the cause of this is rather obscure. It is very definitely not suggestive of pulmonary tuberculosis nor does it have the markings of pneumoconiosis.

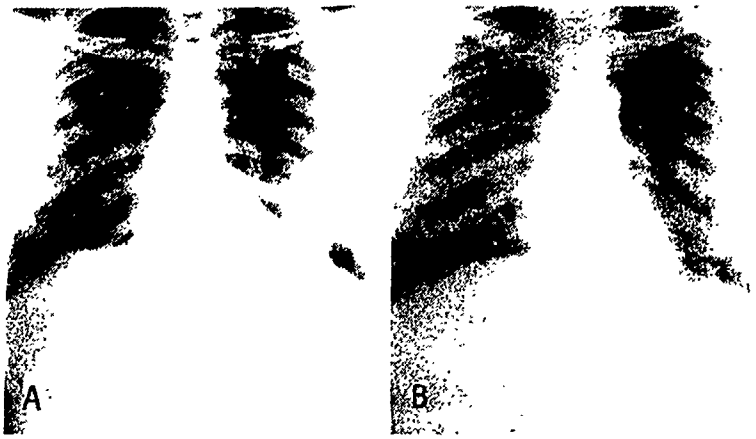


Fig. 73.—*A*, These films are of the Negro cotton worker referred to under "Cotton Dust." For their interpretation, the reader is referred to the comments in the text. *B*, Film of the same patient, taken almost one year later, showing no perceptible change.

This fibrosis is most likely due to a chronic bronchitis with a coexistent pneumonitis. It is conceivable that these rounded areas of increased density might represent small areas of atelectasis developed on a basis of emphysema plus a partial pneumothorax.

Dr. Leroy Gardner, at Saranac, commented:

I am most grateful to you for letting us see the films of your negro cotton sampler. Both Dr. Sampson and I have studied them with greatest interest. I have been looking for a long time for such a case, as I have never had opportunity to study any material after long exposure to dust of this kind.

We quite agree with you that the anatomic changes are probably not due directly to exposure to cotton dust; that there is a complicating infection, but whether this is directly attributable to the dust exposure or not cannot be established.

Dr. Sampson saw the films before he read your letter and because they were marked California he naturally thought of coccidioidal granuloma as a possible etiologic agent of infection. This I would assume you have excluded.

I certainly feel that the sputum should be examined for all kinds of fungus and unusual bacteria. I quite agree with you that lues can probably be excluded as a cause of pulmonary change.

I am sorry that I cannot help you. If you should arrive at any definite conclusions about the case I would be interested to learn about them.

Dr. H. L. Sampson, at Saranac, stated:

Definite pathological process involving both lungs, more particularly at the base, and more particularly the left base, with some emphysema in the top of each lung. The character of the shadows suggests this to be non-tuberculous, but it does not have the nodulation characteristic of silicosis. About the best that can be said for the process is that it does not appear to be tuberculous. The possibility of coccidioidal granuloma would have to be considered.

After viewing these films, Dr. A. J. Lanza wrote:

Both Dr. McConnell, my colleague, and I have viewed these films and see nothing in them to suggest an occupational condition, nor have we at any time seen anything in the films of cotton mill workers that would indicate any specific pulmonary reaction as a result of exposure to cotton dust.

It did seem to us that the appearance in the left base was somewhat suggestive of gumma. Speaking from my own experience, in a healthy, colored male, with a 4 plus Wassermann, presenting symptoms not readily accounted for, I would be very much inclined to try anti-syphilitic treatment. It would be interesting and well worth while to see what changes there might be in the appearance on the *x*-ray films following a few doses of specific therapy.

I will be interested to know what the outcome of this case is and would appreciate your informing me.

Dr. C. O. Sappington had this to say:

Taking all the information into consideration and making a differential diagnosis, it is my opinion that all of the following things must be taken into consideration: Pulmonary tuberculosis; asbestosis; some form of mixed infection; and some form of fungus infestation.

I do not believe that there is any reliance to be placed on a diagnosis of byssinosis, because I believe that the symptoms, the clinical course, and the *x*-ray findings are all out of proportion to any effect that could be produced by cotton dust alone.

While it is not entirely possible to rule out tuberculosis, it is unusual that the predominating lesions in pulmonary tuberculosis should be basal in type as they are here.

The question of asbestosis is raised merely because of the char-

acter of the lung changes, and also because of the suggestion of emphysema in the upper lobes; however, there is also emphysema at both bases, which is against asbestosis; furthermore, at the left base there are large "snowball-like" changes which certainly do not suggest asbestosis.

There is, of course, no suggestion of silicosis in this film.

It is my belief that the greatest possibilities in this case lie between a so-called mixed infection and some sort of fungus infestation.

Summary.—From the various opinions expressed regarding the *x*-ray films, it is evident that the markings indicate (1) a pronounced emphysema and increased fibrosis; (2) the presence of snowball-like or whirlpool configurations of unusual and undetermined character; (3) the possibility of fungous infection; (4) the possibility of gumma.

The maintenance of weight, the absence of afternoon temperature, the negative sputa, the negative guinea-pig inoculation, and the character of the films argue against the possibility of *tuberculosis*. *Syphilis of the lung* can definitely be ruled out. This patient was first seen in May, 1939. At that time the positive Wassermann was noted, and the patient immediately underwent antiluetic treatment. Examination nearly one year later shows the treatmert has changed the complexion of the Wassermann reaction, but the chest films remain unchanged and, if anything, seem further accentuated. According to Sante,[2] the lungs are structures relatively, if not entirely, immune from syphilis. Our present knowledge of the action of inert dusts dispels any likelihood that the markings of the lung as revealed by *x*-ray are the result of cotton dust. It is conceivable that the repeated attacks of allergic asthma, in conjunction with continuous inhalation of dust over many years, may finally set up pulmonary changes consistent with a chronic bronchitis and produce a loss of elasticity of the pulmonary tissue.

In view of the English observations of certain *molds* in the material used, the writer questions the possibility of some unrecognized *fungous infection* in the case at hand. One case of this type is no basis upon which to form an opinion, yet medical history is replete with instances where an isolated finding or suspicion has instigated research into undeveloped fields. The case is not presented as an illustration of pneumoconiosis, but rather one of unrecognized pathology, suggesting the presence of an unrecognized concomitant factor in cotton-dust inhalation.

REFERENCES

1. Collis, E. L.: *English Encyclopedia of Hygiene, Pathology, and Social Welfare*, 1930.
2. Sante, L. R.: "The Chest," *Annals of Roentgenology*, Paul B. Hoeber, New York, 1930, Vol. XI, p. 386.

MILL DUST

Occupational Hazards.—The inert dusts should not be passed over without a word regarding a rather neglected problem in certain mills and grain elevators. While these dusts have no direct effect upon the tissues of the respiratory tract, they do carry certain allergens. In various mills, factories, and plants, the concentration of inert dusts may be very great and yet not affect the majority of workmen employed there. A few of these, however, may develop a respiratory or cutaneous allergy, the cause of which may be unrecognized by themselves or their physicians. Particularly is this apt to be true in those who work in or around flour or grain mills. Attention to this problem has been called by the report of Wittich.[1]

Discharge of Dust into Air.—Wittich points out that in the milling of grains the problem becomes one which concerns not only the workmen in these mills but the community, for the dust discharged into the air from "cyclones" atop these mills causes high contamination of the air over an area of many blocks. In fact, the dust hazard for the workmen is often controlled while the general community remains unprotected. This investigator has pointed out that slides exposed for twenty-four hours inside offices within six blocks of these mills showed numerous grain smuts, rusts, and molds. It appears that the worst offenders are the grass and grain smuts.

Smuts.—These are minute, threadlike, parasitic plants of mycelium, which enter the growing portion of the seedling and grow up with the host, remaining invisible until the heads of grain appear when they partially or wholly destroy the heads, appearing at that stage as masses of dark substance. These masses contain spores which further propagate by getting on or into the seeds, where they reenact their life after the seed is planted. Due to these smut spores, contact dermatitis, atopic forms of skin allergy, perennial rhinitis, and asthma may occur. These conditions may be fairly abundant wherever conditions favor the infestation of smut. They are most pronounced in the Middle West, the Pacific Northwest, the South, and the Southwest.

Grain or Malt Fever.—There exists another illness among grain workers which, when described by them, sounds fantastic. This condition, referred to as "grain fever" or "malt fever" came to the writer's attention just after the printing of this book had begun, so that there was not sufficient time for thorough laboratory investigation of this type of case.

A weighman at the grain elevator of a malt company in Los Angeles, was hospitalized suffering from a penetrating wound of the chest. In relating his past medical history he described recurrent attacks of an odd malaise which he attributed to exposure to malt dust.

The other men at this mill were subsequently reached, and the patient's story was substantiated.

Symptoms.—A description of this illness follows: Several hours after leaving work, or while asleep, the workman becomes aware of a headache, weakness, fever, chills, and cold sweats, as well as nausea, cough, and often vomiting. By morning these symptoms have all subsided, the victim feels normal, and reports for work. Such nocturnal attacks may continue for several weeks. This strange illness occurs most frequently among men never before exposed to grain dust, or among experienced men at the beginning of a new season or upon returning to work after a short absence. In this respect it is not unlike metal-fume fever, an illness to which only those constantly exposed appear to develop an immunity. No satisfactory explanation can be offered for this reaction to malt dust, except that it is probably due to a foreign protein.

Report of Investigation.—To check our observations, a search of the literature revealed only one reference to "grain fever." In a survey of the hazards to which the grain-handlers at Buffalo are exposed, the Division of Industrial Hygiene of New York State reported the occurrence of the aforementioned syndrome.

The Buffalo workers empty boats bringing grain from Canada and the western states. The description of "grain fever" occurring in these workers is exactly like that found in our group. While the condition is not disabling and there appear to be no chronic ill effects, it is worthy of recording in the literature.

Roentgenographic Findings.—This report of the New York investigation not only confirmed our observations but in addition revealed the *x*-ray findings of 395 grain-handlers. It is as follows:

	Men x-Rayed	
x-Ray diagnosis:	*Number*	*Percentage*
Negative	323	81.7
Old healed tuberculosis	49	12.4
Clinically significant tuberculosis	7	1.8
Fibrosis	9	2.3
Non-specific lung infection	7	1.8
Total	395	100.0

The *x*-ray finding of greatest interest in this group of grain handlers was that designated as "non-specific lung infection." Such cases showed irregular patchy areas of increased density in the bases or mid-lung fields associated with an increase in linear markings. The appearance sometimes resembled a clearing pneumonitis, sometimes a bronchiectasis.[2]

The report concluded that the significant hazard to men in this occupational group is that of an *increased liability to respiratory disease*, particularly an irritation to the upper respiratory tract. This occupational cause coexists with, and is probably enhanced by, the hazard found in the inclement weather so prevalent along Lake Erie.

Medicolegal Aspects.—Florists, gardeners, field workers, and others in similar occupations present themselves with cutaneous manifestations of fungus infection or reactions to certain allergens in plants, weeds, etc. But it is not common to have a workman allege his asthma or hay fever is due to the dust in the mill or plant where he works. Undoubtedly more of these cases occur than are reported, probably because the workman is not cognizant of the possible etiology. These cases call for intensive search for the actual cause in order to establish the compensability of the claim.

REFERENCES

1. Wittich, F. W., and Stackman, E. C.: "Case of Respiratory Allergy Due to Inhalation of Grain Smuts," *J. Allergy*, *8*: 189–193 (Jan.), 1937.
2. *Industrial Hygiene Bulletin*, Division of Industrial Hygiene, New York State Department of Labor, Vol. 20, No. 1 (Jan.), 1941.

TREATMENT OF DUST DISEASES

There is no specific treatment for silicosis, asbestosis, or anthracosilicosis other than removal from exposure. Treatment used, then, will be symptomatic or preventive.

Symptomatic Treatment.—Under symptomatic care, general tonics, vitamin preparations, an adequate diet, cough mixtures, and mild narcotics, such as codeine phosphate or sulfate, may be listed. If the patient has marked dyspnea or other disabling factors, a regimen of limited activity should be outlined. However, not all patients showing marked involvement must necessarily join the great class of unemployed. Many of them are better off occupied. Cardiac insufficiency, usually of the right-heart-failure type, and emphysema may require special attention, but their treatment will often be very unsatisfactory in view of the irreversible structural changes which have already taken place in the lungs. Particular care should be taken to guard against respiratory infections, such as colds, acute bronchitis, bronchopneumonia, and lobar pneumonia.

Bronchodilator Vapors.—The use of a continuous-spray technique for the administration of vaporized bronchodilator solutions has been described as giving relief of dyspnea, asthma, and cough in patients with chronic fibrosis and emphysema who are confined to bed or who are on a limited ambulatory regimen. This might be found useful in some of the more advanced of these cases. The solutions used are

epinephrine, 1:100 solution, or when undesirable side-reactions occur with the former, neosynephrin, 1:100. This method as described by Richards, Barach, and Cromwell[1] is as follows: An oxygen pressure tank, with the usual reducing valve to regulate flow, is used. The outflow tube is attached to the vaporizer, and oxygen is run in at a flow sufficient to produce adequate vaporization. A small motor blower unit can be used instead of a pressure tank, but these units usually convey a slight undesirable odor to the vapor. With a flow of from 4 to 7 liters per minute, 1 cc. of the solution is vaporized in from three to ten minutes. The patient holds the vaporizer nozzle in his oropharynx and breathes quietly. As a vaporizer, Vapco and Vaponefrin models are satisfactory.

A convenient schedule suggested by these authors is the inhalation of the vapor of from 0.5 to 1.5 cc. of the solution for from five to ten minutes: (1) on waking in the morning, and before getting out of bed; (2) in the middle of the day, *e.g.*, after lunch; and (3) just before retiring at night. With this method significant increases in vital capacity were noted in most cases of chronic fibrosis and emphysema.

Measures in Tuberculosis.—If tuberculosis in active form is present, sanatorium treatment is indicated, with the various therapeutic approaches usually directed toward that disease being used. Special care to guard against spread of tuberculosis, if it occurs, is needed. *Spontaneous pneumothorax* with collapse of a lung from rupture of emphysematous blebs has been described, although its incidence is not great because pleural adhesions usually prevent the occurrence of such collapse even though a bleb may rupture. If such an incident occurs, removal of the compressing air, administration of oxygen, and treatment of circulatory failure may be required.

Prophylaxis.—Preventive measures are of extreme importance, especially as they apply to silicosis and asbestosis.

Aluminum Dust in Prevention of Silicosis.—Denny, Robson, and Irwin,[2] of the University of Toronto, have recently made a discovery which may shortly be of much practical importance in the prevention of silicosis. Their experimental results showed that a very small proportion of metallic aluminum dust would protect experimental animals from silicosis. In these animals the inhalation of aluminum dust in large quantities over long periods of time showed no effect on the general health of the animals and no evidence of toxicity or damage to tissues. The concentration of the aluminum necessary to prevent silicosis was hundreds of times below the explosive concentration of aluminum powder. At the McIntyre mine, by using small quantities of aluminum powders, varying from 3 to 20 gm. dispersed in a dust cloud, reductions in solubility of 90 per cent or better were obtained from

dust samples taken from 150 to 300 feet from the face, following a drift round blast having a volume of 8000 cubic feet.

Other Measures in Silicosis Prevention.—Lanza[3] listed the following as factors in the prevention of silicosis:

(*a*) *Abandonment of Process Producing Dust.*—An example of this is the cleaning of metal castings with adherent sand by a wet method instead of by sand- or shot-blasting.

(*b*) *Substitutes for Dangerous Materials.*—Examples include the use of artificial abrasive wheels for sandstone in metal-grinding; metal

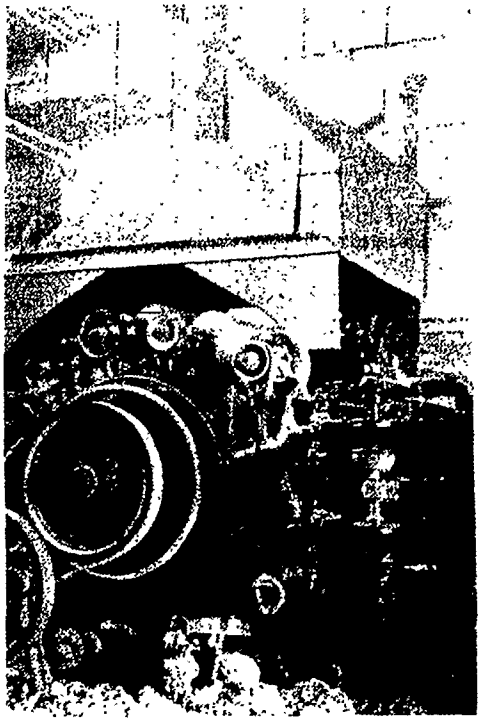


Fig. 74.—Exhaust ventilation applied to an unenclosed primary carding machine for asbestos. (U. S. Public Health Service, *Bulletin 241.*)

grit or aluminous abrasive for sand or flint in sand-blasting; alumina for placing china biscuitware and for polishing and grinding in the pottery industry.

(*c*) *Suppression of Dust Immediately on Its Production.*—An example of this is the improved outlook in mining and quarrying in sandstone, granite, and other siliceous rocks and deposits by the use of water alone or combined with foam-producers with suitably modified drills.

(*d*) *Removal of Dust Near Point of Origin.*—This is to prevent its reaching the breathing level. Localized exhaust ventilation by mechanical means has been widely used (Figs. 74, 75).

(*e*) *Exclusion of Persons.*—No one should be allowed in the room where a dusty process is being carried on unless he is actually and necessarily employed in it.

(*f*) *Protection by Individual Respirators or Breathing Apparatus.*—These should have a high filtering efficiency against fine dust, fit

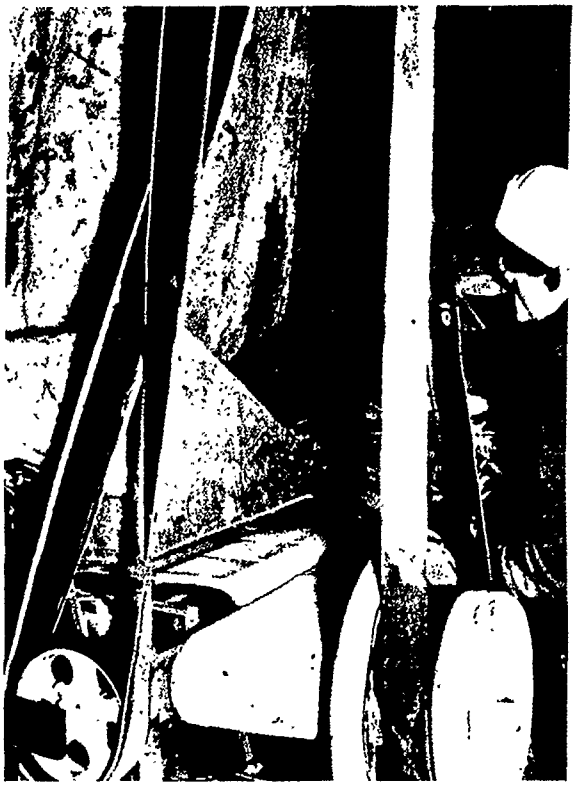


Fig. 75.—Large exhaust outlet to remove silica dust. (U. S. Public Health Service, *Bulletin 244.*)

accurately, have low resistance, and be reasonably comfortable and cheap. These are not substitutes for dust suppression or removal.

(*g*) *Adequate Medical Supervision.*—Preemployment examinations should be of an exacting type, with special attention given to the family history for tuberculosis, history of a tendency to respiratory infections, the past medical history, previous exposure to silica, the present physical condition, and *x*-ray examination of the lungs. Successful

applicants should be picked men in the best of health from every standpoint. Periodic reexaminations should be made, the frequency depending upon the severity of exposure, type of work done, etc. The first onset of pulmonary symptoms in those engaged in dusty occupations should be the signal to abandon the work, since, although the damage already done cannot be remedied, it may be checked to some extent by avoiding further exposure to the dust.

(*h*) *Ventilation.*—There should be rigid adherence to proper ventilation, and inspection of the hazard should be periodic to insure the minimal concentration of dust. For those working in the inert dusts, similar but less exacting requirements need to be enforced.

Asbestosis.—The control of asbestosis follows very closely the steps outlined above. In asbestosis the dust-control methods are very practicable and have been vigorously carried out by asbestos-manufacturing companies with successful results.

Mill Dust.—If alleged symptoms in a workman were, after thorough investigation, found to be due to grain smuts, rusts, or molds, the obvious treatment would seem to be his removal from exposure. Some striking results in patients with respiratory allergy due to rusts and smuts following hyposensitization with rust and smut extracts have been described.[4] The symptoms in these patients subsided after two or three injections.

REFERENCES

1. Richards, D. W., Jr.: "Use of Vaporized Bronchodilator Solutions in Asthma and Emphysema," *Am. J. M. Sc.*, *199:* 225–232 (Feb.), 1940.
2. Denny, J. J., Robson, W. D., and Irwin, D. A.: "The Prevention of Silicosis by Metallic Aluminum," *Canad. M. A. J.*, *40:* 213 (March), 1939.
3. Lanza, A. J.: *Silicosis and Asbestosis*, Oxford University Press, New York, 1938.
4. Waldbott, G. L., and Ascher, M. S.: "Rust and Smut, Major Causes of Respiratory Allergy," *Ann. Int. Med.*, *14:* 215 (Aug.), 1940.

PART V

CHAPTER XXX

THE INDUSTRIAL BACK*

WHILE backache has not been considered primarily an occupational disease, its prevalence among workmen and the frequency of claims for compensation for this disability render a discussion of this condition obligatory.

Increase in Number of Claims.—During 1939 every tenth patient presenting himself at this clinic alleged a back sprain. Each year there has been an increase in this percentage over the previous year. What is the reason for this increase? Certainly man's work is not more arduous. The jack, crane, hoist, derrick, steam shovel, truck, and other devices spare him much that was formerly done by purely human effort. Possibly these aids have changed the environmental conditions to the extent of altering the potentialities that man has for work. Be that as it may, there is no question but that the attitude the workman and the physician have toward the benefits of compensation has added to the number of claims. The patient, being unable to evaluate the relationship of his pain to his work, naturally thinks it is the result of effort. The average physician doesn't attempt to make any differentiation. Because of the existing situation, this relationship was subjected to a prolonged study, the results of which, it is hoped, will influence an adoption of a saner attitude toward the so-called "industrial back."

Relation of Pain to Work.—In complete disregard of the multiple causes of backache, the tendency in industrial medicine is to "mass-group" all these cases under the diagnosis of back sprain. This error seems to be predicated upon the "locale" of the onset of pain. If it ensues while a man is cutting his own lawn, the term "lumbago" is invariably applied and the condition attributed to causes within the man. But pain arising while stooping, bending, or lifting at the plant is called "back sprain" and considered the result of motion while working for someone else. The situation assumes added import when it is appreciated that this disability is becoming one of the most frequent causes of claims for compensation.

* This chapter represents a slight enlargement of the author's paper published in the *American Journal of Surgery*, *48* (July), 1940.

DIAGNOSIS

Sprain in Presence of Normal Motion.—In the series offered for consideration, an inadequate or negligible history of injury occurred in over 70 per cent of the cases. Obviously, then, the history should determine the basis for investigation before accepting or denying a case as being occupational in origin. The patient brought in disabled because of a fall from a ladder or platform requires such an examination as will determine the extent of his injury. But the man who becomes disabled while engaged in a simple motion requires an investigation upon more divergent lines. *It is imperative that criteria of normal and abnormal motion, or movement, be established in the examiner's mind.* If the origin of pain is to be evaluated and its responsibility allocated, the above statement must become indelibly fixed in the minds of all industrial physicians. Despite the contention that in the evolution of man instability of the lower back occurred when he assumed the orthograde position, there does exist a normal physiological range of motion. Sprain is defined as an overstrain, the result of excessive motion. Accepting the last two statements as a premise, how can a motion to which one is accustomed, one executed daily in the same manner, one within the normal range and lacking excessive, strenuous, or unusual characteristics, constitute a sprain? Yet, beyond any question of doubt, from mild to severe, disabling pain does occur following a normal motion or a trivial act. Why? It should be apparent that it would not have occurred had there not been an added factor, an unrealized factor, present. It must be this added factor, this added "straw" that breaks the patient's back. The failure to search for it in industrial medicine has resulted in the preponderant diagnosis of "back sprain." Multiple causes and various theories represent this "straw," which will be reviewed briefly.

Sacro-iliac Slip.—The sacro-iliac slip or subluxation will receive no mention here other than to state that despite its wide refutation by reputable investigators it still finds wide acceptance as a common cause of backache. Invariably this diagnosis is made without benefit of *x*-ray study. There was not a single instance of such a condition in our large series of cases. Indiscriminate use of this term should cease.

Sources of Back Pain.—At one time congenital or developmental defects were emphasized as a cause of backache. Then a lack of emphasis followed, and the importance of Bertolotti's syndrome (sacralization, sciatica, and scoliosis), along with other abnormalities, was minimized. Recently certain of these defects have been receiving more consideration as the cause of back pain.

Muscles and Ligaments.—Why is it that certain persons with these defects evidence no back pain, while others have a chronic discomfort

or disability? It is believed that the reason is based upon the condition of the muscles and ligaments. As long as they maintain their tone, the defect remains an innocent anomaly. If, however, the tone and strength of the muscles and ligaments supporting the lumbosacral area are deficient, then the structural defect becomes a participating factor. Persons with structural defects and mechanical weakness have a lessened capacity to engage in occupations requiring the use of the lower back. In fact, in the presence of this combined weakness, disability may occur without direct trauma or excessive strain.

Injuries and Toxemias.—Goodwyn[1] expressed the belief that certain anomalies are a definite source of pain when aggravated by injury or toxemias. He further stated that in the presence of such anomalies, disability can result without injury.

Facet Irregularities.—Putti[2] particularly has renewed the profession's interest in the abnormal placement of the articular facets. Their variation in the lower portion of the back is important when it is realized that the intervertebral canals between the fourth and fifth lumbars and the fifth lumbar and sacrum are the smallest, whereas the fourth and fifth nerve roots emerging from them are the largest. Facet irregularities can only be detected by employing the oblique view of the *x*-ray. How frequently this irregularity may be passed up by routine *x*-ray study is obvious. Willis once thought the separate neural arch an important defect, but later expressed agreement with Badgley in doubting its significance. Spondylolisthesis and the horizontal sacrum are unquestionably definite factors in low back pain.

Lumbosacral Contracture.—One of the commonest findings in the routine examination of the back by *x*-ray is a narrowing of the lumbosacral junction. Hodges[3] found it in 57 per cent of his cases with backache, Williams[4] in even a greater percentage. Its importance has been slighted until recently, and Williams in particular has awakened interest in this possible source of pain. He describes this lesion as a chronic, traumatic, or postural destruction of the lumbosacral intervertebral disc. Certain muscles weaken through disuse while others become stronger, resulting in several alterations of weight-bearing and a constantly increasing force brought to bear upon the posterior margins of the intervertebral discs of the lumbar and lumbosacral areas. This disc becomes wedge-shaped, and there is constriction of the foramina with subluxation of the facets. Its high incidence in patients with back pain indicates consideration in the eventual diagnosis.

Rupture or Herniation of Intervertebral Disc.—Another phase of intervertebral disc pathology receiving increasing attention is its rupture or herniation. To appreciate the pathology of disc protrusion or herniation, it is necessary to recall that the body of the vertebra is

composed of cancellous tissue, the major portion of which is uncovered and not protected by compact bone.

Disc Anatomy.—The disc between the vertebrae is made up of three parts:

(*a*) *The Central Portion (Nucleus Pulposus).*—It is firm, elastic, of fluid consistence and is retained in its central position by considerable tension. It functions as a cushion which serves to absorb and distribute evenly pressure applied along the axis of the column. Because of its fluid consistence, it swells or bulges out if extreme pressure is applied, or if the pressure of the surrounding tissue is removed (degenerative processes).

(*b*) *The Peripheral Portion (Annulus Fibrosus).*—This structure lacks the elasticity of the nucleus pulposus for which it forms a capsule and serves to fix the disc.

(*c*) *The Cartilaginous Plates.*—These are two thin layers of hyaline cartilage situated between the disc and the body of the vertebra and are found only over the central portion of the disc. They likewise serve to withstand the pressure of the nucleus.

Expansion of Disc.—With the above picture in mind, it is not difficult to conceive of the susceptibility of the disc to pressure influence, whether it is from a single act of trauma, from repeated trauma, or from degenerative changes. The spine is subjected to constant functional activity. Just being about our daily business imposes an element of wear and tear. The daily functional imposition placed upon the intervertebral disc is comparable to that placed upon the vascular system. Expansion of the disc is seen repeatedly in *x*-ray films of persons who present no evidence of cord symptoms. That is, the normal contour of the intervertebral space is altered, indicating an expansion into the body of the vertebra. It was found to be present in 32 per cent of our patients. Whether the incidence is less in the nonlaboring group as compared to workmen in heavy industries, the writer is unable to say. This nuclear expansion into the body of the vertebra is commonly referred to as "Schmorl's node" (Fig. 76). Just what effect these have in the production of a back disability is as yet not fully known. It is thought by some that reactive changes follow the protrusion of the disc substance into the spongiosa, such as proliferation of cartilage and bone. It is held that in youth the loss of the disc substance, together with the alteration in shape and size, gives rise to spinal deformities.

Retropulsion of Disc.—Probably a more important phase of disc disturbance—certainly one which is receiving widespread consideration in the present-day literature—is the retropulsion of the disc into the canal space (Figs. 77, 78). It has been definitely established that a

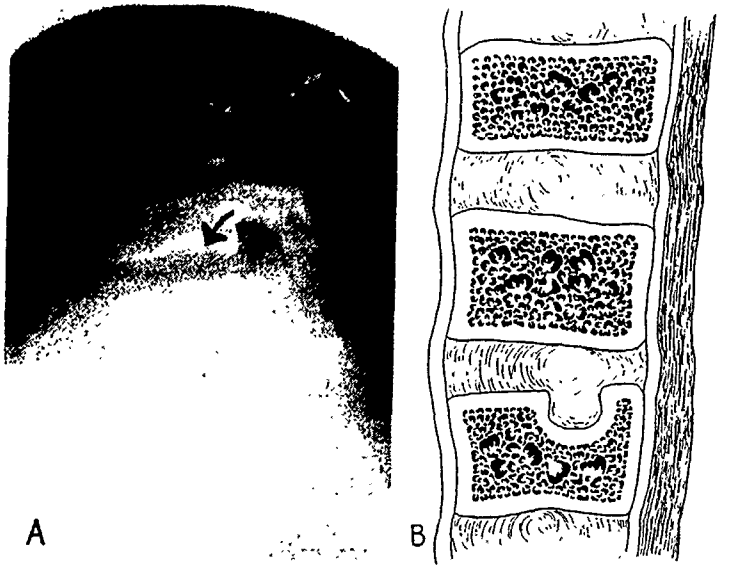


Fig. 76.—*A*, Schmörling of the intervertebral disc between the third and fourth vertebrae and to a less extent between the fourth and fifth. No history of serious injury to the back. *B*, A protrusion of the disc substance into the body of the vertebra (Schmorl's node). *x*-Ray films do not reveal the marked degree of indentation indicated by the drawing.

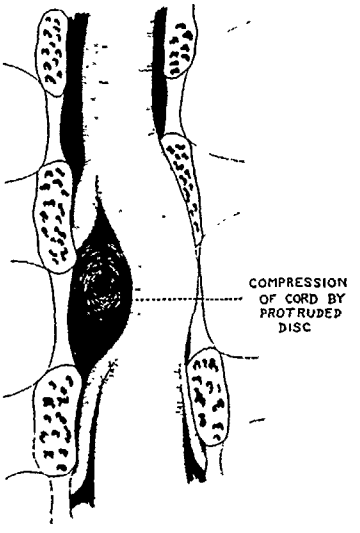


Fig. 77.

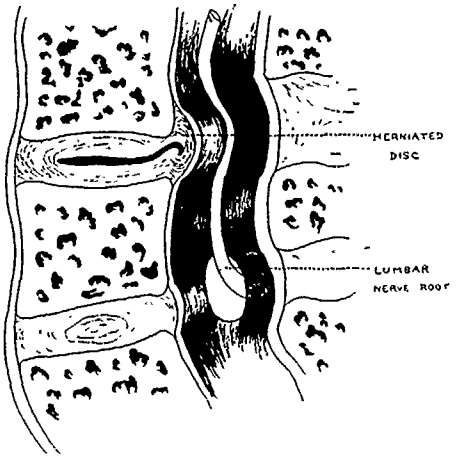


Fig. 78.

Fig. 77.—Retropulsion of the disc (herniated intervertebral disc) showing pressure upon the cord—dorsal view.

Fig. 78.—Retropulsion of the disc into the spinal cord—lateral view.

protrusion of this nature does cause compression of the spinal cord or of one or more of the nerve roots, resulting in a fairly typical syndrome of low back and sciatic pain. In the wake of this discovery there has been a tendency to operate on too many patients suspected of nucleus pulposus without a justifiable diagnostic basis. The symptoms are invariably pain in the lower back extending into the buttock and down the affected leg posteriorly, with a tendency to involve the lateral and anterior surface of the lower leg. The pain is usually constant but may be intermittent with acute attacks accompanied by sciatic scoliosis. The pain is accentuated usually by coughing, sneezing, or sudden increase in the intra-abdominal pressure. The pain may also be increased by hyperextension of the spine, spasm of the lumbar muscles, loss of normal lumbar lordosis, reaction to jugular compression, and positive Lasègue's and Kernig's signs are helpful diagnostic findings, as are also the diminution or loss of the achilles tendon reflex and frequently atrophy of the lower leg.

Fig. 79.—This film is of a patient who had clinical evidence of a ruptured nucleus pulposus. At operation no herniation of the disc was found, but hypertrophy of the ligamentum flavum was present. The shadow is that of lipiodol in the spinal canal.

These findings strongly indicate the presence of a nucleus pulposus, but the diagnosis should be further substantiated by the elevation of the total protein content of the spinal fluid (fluid should be withdrawn from the lower part of the lumbar spine), and by finding *x*-ray evidence of spinal blockage by using a radiopaque oil (Fig. 79).

Some investigators object to lipiodol on the belief that if the oil reveals no blockage and is permitted to remain in the canal, since no operation is performed in such a case, it is a source of irritation. These objectors prefer to use air. However, among others, Barr, Hampton, and Mixter[7] noted no serious disturbance from lipiodol in twenty patients who were not operated upon. Unquestionably the patient with chronic backache will receive in the future an investigation along the lines just outlined and undoubtedly many a suspected malingerer will have to be reclassified. At the time of operation, a search for a nucleus pulposus has revealed not infrequently the presence of a hypertrophy of the ligamentum flavum, causing cord pressure.

Contracture of Iliotibial Band.—To continue with other conditions considered to be the cause of back pain, Ober[6] calls attention to the contracture of the iliotibial band and suggests cutting of this band for the purpose of securing relief.

Involvement of Dorsolumbar Junction.—A recent study and report of Judovich and Bates[7] detracts from the lumbosacral area the importance that has been ascribed to it and centers attention on the dorsolumbar junction. They have been impressed with the frequency of a syndrome involving the twelfth dorsal and first lumbar nerves, resulting in a neuralgia due to the susceptibility of the dorsolumbar spine to injury and strain. Among some of their contentions are these: That the dorsolumbar area represents a combination of fixed and movable parts which are more susceptible to injury than the fixed segments found above and below this area; that this area bears almost as much weight as the lumbar spine, yet these segments are smaller and weaker; that because this site is nearly in the middle of the spine it bears more leverage; that the lumbar enlargement of the spinal cord in this region makes vertebral housing relatively smaller. Judovich and Bates' contention, in the main, is similar to Steindler's[8] in that there is an involvement in a large percentage of these cases of the posterior division of the spinal nerves. However, they locate the original site higher than Steindler, and disagree with the commonly accepted textbook distribution of the twelfth dorsal and first lumbar nerves (posterior division). They emphasize that tenderness should not be confused with pain, and that while pain may be confined to any one point, tenderness exists over the entire distribution of the nerve. Their illustrations reveal distribution extending anteriorly and involving the lower anterior wall adjacent to and below Poupart's ligament, ending in the midline. According to their theory, tenderness should exist over this portion of the abdomen as well as over the back. This is not borne out in our study.

Circulatory Disorders.—Makaroff[9] attributes much back pain to

a vasospastic disorder, believing in many instances it should be treated as a circulatory condition rather than an orthopedic entity. He states that an occlusion of the blood vessels can take place as a result of trauma or focal infection, producing a pain comparable to that in the leg following an ischemia. Ridlon and Berkheiser[10] called attention to the calcareous degeneration of the abdominal aorta as a cause of backache (Fig. 80). The author has one such unquestionable case in this series and eight others where this apparent circulatory deficiency was considered contributory.

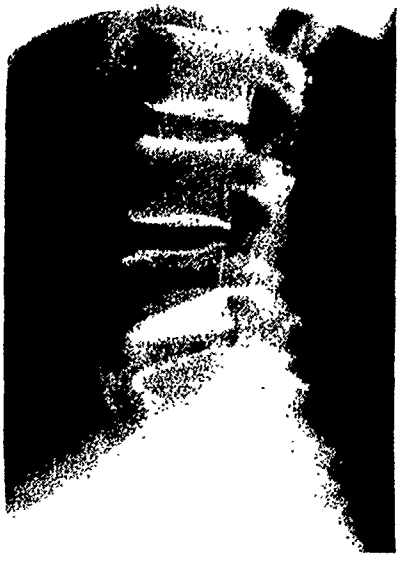


Fig. 80.—Calcification of the abdominal aorta. (Anteroposterior view shows marked calcification of the iliacs.) Patient complained of pain across the lower back and in both legs after climbing stairs or walking a short distance, which was almost immediately relieved by resting.

Fascial Adhesions.—Gratz[11] contends that, "Fascial planes function as joints, synchronizing motion between muscles, groups of muscles, nerves and blood vessels. Traumatic and inflammatory lesions may involve these planes resulting in myosynovitis or fascial adhesions. These are believed to be competent producing causes of muscular pain by involvement of the nerves and the limitation of the normal range of motion in the joint by retardation of the gliding mechanism of the muscles." Using air insufflation followed by pneumofasciograms he demonstrated these adhesions. If his viewpoint is accepted, manipulation is of value not only in the stretching of muscles but in the freeing of agglutinated surfaces and the breaking of adhesions.

Posture, Nutrition, and Exercise.—Faulty posture, poor nutrition, insufficient exercise, and ignorance of how to execute motion are definite sources of back pain. These are daily and frequently exemplified in the workman who presents himself with back pain following a simple act. He finds his contrast in the trained pole-vaulter whose spine and muscles coordinate as he twists, squirms, and turns to ascend to almost unbelievable heights. Consider the contact, impact, and pretzel-like positions imposed upon the football player. Yet the painful back in the college athlete is zero compared to that in the industrial worker. Proper food, guided exercise, and knowledge of how to engage in movements involving stress account for the former's seeming immunity. Industry of the future might well consider the physical director as essential as the guardrail or respirator in the prevention of disability among its workers.

Miscellaneous Sources.—This passing parade of possibilities must cite allergy and endocrine imbalance. Referred pain from adjacent organs is common. Finally, but most important, are the secondary effects from *systemic or focal infection*. Why this source is accorded such negligible recognition by the industrial physician is beyond understanding. Pain occurring in the knee, elbow, or shoulder, with or without motion, should invariably instigate a search for infection elsewhere in the body. Are the fascial planes and vertebral joints not just as susceptible?

Classification of Sources of Back Pain.—Consideration of the varied etiology outlined in the review just completed stimulated a study conducted over a three-year period. That the term "back sprain" could be applied to approximately 80 per cent of these disabilities appeared unreasonable and unscientific. It was felt that an adequate answer could be obtained only by studying a sufficiently large series of cases and only those cases where the onset of pain allegedly occurred while the patient *was working*. The series represents all ages, races, various social levels, and types of industry.

Procedure.—In the clinic where this material was gathered the following procedure is observed. Excluding the recently traumatized back (severe falls, crushes, and blows) every patient alleging back pain is referred to the Special Examination Department. Following a detailed, exacting history and a complete physical examination, *x*-rays, urinalyses, and blood counts are done routinely. If indicated, and when necessary to establish the diagnosis, further *x*-ray examinations of teeth, sinuses, lungs, genito-urinary tract, etc., are made; also blood Wassermann, blood chemistry, blood sedimentation, smears, and cultures. The data gathered from this three-year study are presented in the following tables:

TABLE 17 PATIENTS IN SURVEY

Number of cases studied from April 1, 1936, to February 28, 1939	3018
Incident to total clinic admissions, 1936	7 6
Incident to total clinic admissions, 1938	10 4
Males	2799
Females	219
Average age	36.2
Patients referred to clinic diagnosed as back sprain	74 per cent
Patients referred to clinic diagnosed as sacro-iliac disease	5 per cent

Attention is called in this table to an increase of 2.8 per cent of back claims in 1938 over those of 1936. Of the 219 females represented, over 60 per cent of these had received falls or contusions as a result of their heels catching, or their slipping on a smooth surface.

Special attention is called to the group of cases in Table 18. These patients had been diagnosed and treated elsewhere before being referred to this clinic. The condition in practically 80 per cent of them had been diagnosed as "back strain." Table 18 represents our opinion of their compensability after study.

TABLE 18—STATUS OF COMPENSABILITY

	No. of Cases	Accepted	Rejected	Percentage of Rejections
1936 (April 1 to Dec. 31)	722	433	289	40
1937	1092	564	528	49
1938	1014	315	699	69
1939 (Jan. 1 to Feb. 28)	190	56	134	71
Totals	2018	1368	1650	57

The reason for this sharp rise in the percentage of rejections found in Table 18 is that in the summer of 1937 a policy of unlimited laboratory investigation was adopted. This resulted in finding pathological conditions which, when considered with a negligible history of injury, formed a basis of rejection. While the entire average rejection over a period of three years was only 57 per cent, the rejections for the years 1938 and 1939 averaged 70 per cent.

TABLE 19—BASIS OF REJECTION

Of the accepted cases, a contusion occurred in	423 or 31 per cent
Of the accepted cases, a leukocytosis occurred in	3 per cent
Of the rejected cases, a leukocytosis occurred in	37 per cent
Of the rejected cases, abnormal urine, smears, serology, blood chemistry, etc., occurred in	13 per cent
Of the rejected cases, a history of previous back disability occurred in	42 per cent
Of the rejected cases, fever occurred in	11 per cent

The pathological conditions which were present in the rejected cases are broken down and outlined in Table 20.

TABLE 20—MORBID ANATOMY IN REJECTED CASES

Throat infection	301
Defective oral hygiene, significant degree	696
Coryza	127
Pleurisy	31
Pneumonia; pain in dorsal area first symptom	2
Cholecystitis	5
Gallstones	3
Aneurism	1
Aortitis, with positive serology	6
Coronary heart disease, suspected	12
Appendicitis, with chief complaint of back pain	3
Kidney stones	16
Nephritis, with urinary evidence	63
Pyelitis	3
Cystitis	1
Hydronephrosis	7
Pyonephrosis	1
Acute gonorrhea, with back pain the reason for examination	42
History of gonorrhea	512
Enlarged uterus, tumor	2
Abortion; called industrial doctor because of backache	1
Abnormal prostate	208
Pylonidal cysts, alleging their presence as resulting from contusion or strain	26
Pernicious anemia; presented himself because of "sciatica" of legs from heavy lifting	1
Tabes	3
Lead poisoning; back pain first symptom	1
Bronchiogenic carcinoma; presented himself because of dorsal pain	1
Cancer of prostate	3
Obesity, marked, with recurrent back pain, with congenital defective spine	9
Adiposogenital dystrophy, age 13, back pain after one day of lifting grocery baskets.	1

Tables 19 and 20 comprise Exhibit A in the indictment of the universal usage of the term "back sprain" as a diagnosis of the accepted cases. Of the accepted cases, one third of them revealed a history of actual contusion, leaving only two thirds or 945 out of 3018 cases to be accounted for by unusual stress or strain or extraordinary circumstances. One cannot pass lightly over the fact that of the rejected 51 per cent had abnormal laboratory findings. Nor can one observe the extents of morbid anatomy listed in Table 20 without realizing that the workman is subjected to the same ills all man is heir to and, therefore, the origin of his pain should not be stereotyped.

TABLE 21—ROENTGENOGRAPHIC FINDINGS

Patients x-rayed in this series	2414	
Lumbosacral pictures	1929	
Cervical pictures	305	
Dorsal pictures	180	
Osteo-arthritis	24 6	per cent
Cancer of vertebral bodies	6	
Tuberculosis of vertebral bodies	0	
Myeloma with involvement of spine	1	
Kümmell's disease	5	
Nuclear expansions (Schmorl's cartilaginous nodules)	32	per cent

The incidence of 24.6 per cent of osteo-arthritis recorded here is conservative. It represents only well-defined changes. Its place in the etiology of back pain is questionable. Hodges and Peck found it in 23 per cent of their cases of back pain but also present in 28 per cent of their control series with no back pain. They concluded it had no significance. This conclusion may be applicable to the average group, but the presence of advanced arthritis of the spine in the elderly laborer cannot be ignored. To our clinic come men from the Goodwill Industries, men older than their years, men who have faced life with

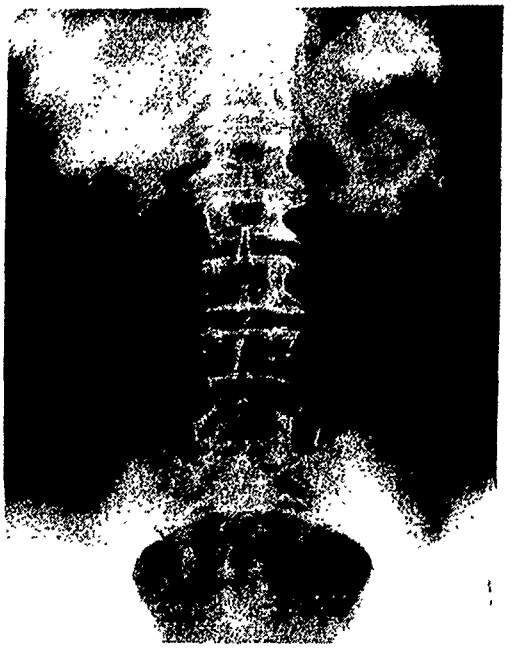


Fig. 81.—Marked osteo-arthritis of a sixty-four-year-old Goodwill Industry worker who complained of back pain after lifting bundles of paper (repeated claims of back sprain).

the wind of fortune not always at their backs. Invariably they present no sufficient history of injury, but on *x*-ray examination marked arthritis is observed (Fig. 81). One notes the absence of tuberculosis in this large series of films. So-called "Kümmell's disease" was present in two patients who recalled a previous history of injury, and in three who had no history of injury. Invasion of the vertebral bodies by nuclear expansions in this series has already been referred to. Coupled with narrowing of the intervertebral spaces, it is finding increasing emphasis in recent orthopedic literature.

TABLE 22.—VARIATIONS FOUND IN LUMBOSACRAL AREA
1929 Cases

Sacralization: bilateral, 5.2 per cent; unilateral, 9.4 per cent	14.6 per cent
Failure of fusion (2 found in dorsal area)	18.4 per cent
Horizontal sacrum	3.4 per cent
Narrowed lumbosacral disc	52 per cent
Spondylolisthesis	2.8 per cent
Posterior displacement of 5th lumbar	21 cases
Articular facet variations (incompletely studied)	1.6 per cent
Nucleus pulposus herniation into canal: 3 patients injected; 3 revealed blockage	

A narrowed lumbosacral disc found in 52 per cent of the patients is evidently smaller than that found by Williams and others. Our figure represents patients who showed not only a wedging of this disc but a diminution of the foramina. Efforts to determine cord pressure or blockage by posterior displacement of the nucleus pulposus have been but recently begun in the clinic, so that we have no knowledge of how often it might have been present in this series. Posterior displacement of the fifth lumbar is found in twenty-one patients in our series. We believe that these are not optical illusions, as Willis[12] warns they are apt to be.

TABLE 23—BACK FINDINGS

Tenderness:	Percentage
Lumbosacral: bilateral, 10 per cent; unilateral, 18 per cent	28
Sacro-iliac: bilateral, 6 per cent; unilateral, 13 per cent	19
Sciatic notch: unilateral	28
No tenderness	25
Limitation of motion, mild	63
Limitation of motion, marked	20
No limitation of motion	17
Limitation of straight leg raising:	
Unilateral	41
Bilateral	21
Pain, without motion; constant but aggravated by motion	56
Pain, on motion only	24
Pain, unclassified, indefinite as to motion or rest	20

Examination of Patient. *Normal Motion versus Abnormal.*—It is not to be construed from the foregoing discussion that a case of actual strain should be ruled as non-compensable simply because infection or morbid anatomy is found to coexist. But it is to be reiterated that the physician must carefully evaluate the history of the incident and determine if the factors constitute an abnormal, excessive, overt, or unusual stress; or whether the movement or motion was a normal act and one to which the patient was accustomed.

Details of Investigation.—Following the history, and especially if the act which allegedly induced the pain appears to be normal, the patient should be carefully searched for other causes. The vogue of ushering the patient into the physiotherapy room as soon as he has finished his preliminary statement of, "Doctor, I wrenched my back," is to be condemned. The examiner should have the patient describe

and illustrate, if possible, the exact mechanism of the alleged act. He should inquire into the weight, size, and type of object being handled, how soon pain ensued after this act, the type of pain and its location, and whether it remained in the same area or moved about. It is constantly present or only on motion? How frequently does the patient have backache and has he ever had a severe fall or blow to the back? These and many other details must be ascertained.

Physical Examination.—This should be done with all clothing removed. It is a good rule to disregard the back until the end of the physical examination, starting and going through the routine that would be carried out in any complete physical examination. Special search for focal infection in teeth, throat, or prostate must be made as well as a search for possible disease in the various organs. This advice may appear redundant, but there exists ample evidence that it is not.

Laboratory Investigation.—This should be made as indicated. Most insurance carriers or self-insured employers appreciate the value of, and willingly permit, routine *x*-ray examination of every case of alleged back strain. Most of them will give consent to special laboratory procedure if they are contacted and informed of the reason for such an examination. (This clinic does the bulk of the work for one large insurance carrier which has agreed to a flat-rate fee for *x*-ray examination, urine analysis, and blood count of every patient with back pain. While it might seem that such a procedure is costly and useless in many instances, in the end it has proved profitable in the accurate and just dispensation of the claim.)

Error of Attributing Back Pain to Occupational Activity Alone.—The investigation of over 3000 cases by the author reemphasizes what has long been recognized—that backache has a multiple and varied etiology. But the amazing revelation to be garnered from this study is that in spite of the recognized causes, and irrespective of the possible pathological conditions which may exist to explain backache, a diagnosis of assumption occurs. The study indicates that the industrial physician used this blanket diagnosis in 80 per cent of the cases which were subsequently studied by us and which were reclassified because of discovered accountable etiology, so that actually only 30 per cent were considered as having an occupational origin. From this study, *it should be appreciated that the disabling back is not an industrial entity.* The practice of according it the same occupational earmarks concerning its origin as are attributed to silicosis or lead poisoning, for instance, is a fallacy. The so-called "industrial back" is not primarily an orthopedic problem. It is one for differential diagnosis. From this study it is obvious that disease and infection comprise a considerable portion of the etiology.

MEDICOLEGAL ASPECTS

Figures from this clinic indicate that the average length of temporary disability in cases of actual strain or sprain was 24.3 days. Prolongation beyond that time should arouse suspicion of other coexisting causes. Permanent disability rarely exists from strain in the absence of direct trauma.

Illustrative Cases.—It is not necessary to cite instances of actual back strain. Such cases invariably present a history of sufficiently unusual stress, strain, or overexertion. Nevertheless, these patients *should receive just as thorough an examination as those without an* adequate history of strain. But the coexistence of an unrelated infection or disease should not prevent the case from being accepted as compensable. To reiterate a thought which runs as a theme throughout this book: the physician should remember that he is not making an examination to disprove the patient's claim or the employer's contention. In the acute back disability, the patient is not fooling about his pain. The duty of the doctor is to determine the cause of the pain, let the chips fall where they may.

On the other hand, the practice of accepting and treating a *pain in the back* as a back sprain when the cause of the pain is an infection or disease process is to be condemned. The numerous instances of back pain due to focal infection have been emphasized by our statistical study, and individual cases need not be related. The following few cases have been selected, not because they represent the infrequent error in assuming that a back sprain exists, but because such mistakes are common. The few illustrations selected serve to warn against: (1) the acceptance of the patient's statement about the cause of his pain; (2) the widespread acceptance of the opinion of the quasi-quack or the increasing lay opinion that a sacro-iliac slip has occurred; and (3) treatment without complete physical and laboratory investigation.

Case I.—A sixty-four-year-old janitor received a sudden, severe pain in the back as he stooped to roll a rubber mat. Without benefit of *x*-ray he was accorded six weeks of physiotherapy with no improvement resulting and with a steady downhill course on his part. Subsequent history revealed a marked loss of weight over a period of months, generalized weakness and increasing constipation, approaching obstipation. Examination revealed evidences of loss of weight, anemia, a sharp, angular kyphosis at the twelfth dorsal, first lumbar, which the patient maintained was not present prior to "his accident," and the *x*-rays revealed a cancer of the twelfth dorsal vertebra secondary to the prostate which had not previously been examined (Fig. 82).

Fig. 82.—*A* and *B*, Films show the presence of a destructive process in the eleventh thoracic vertebra with a complete collapse of the vertebral body so that only a part of the dorsal half remains. Despite the collapse, the intervertebral discs on each side show a space which is relatively well preserved. The anteroposterior film shows the presence of a paravertebral abscess, as well as numerous small chips of bone. Infiltration in the head and neck of the left eleventh rib is indicated by the spotty demineralization in this area. The ventral and cranial corner of the twelfth thoracic vertebra is apparently similarly infiltrated. Malignancy had to be differentiated from tuberculosis, but in view of the presence of a prostatic malignancy, metastatic carcinoma of the spine appeared more probable.

C, Schematic drawing to show the collapse of the body of the eleventh thoracic vertebra, the relatively clear disc spaces, and the fragments of bone overshadowing the lower border of the tenth and almost the entire body of the twelfth thoracic vertebrae. This patient (referred to in the text) was treated for back sprain.

Case II.—A patient—having a large palpable mass in the right abdominal quadrant; temperature of 101.2° F.; urine loaded with pus; and stones in the right kidney, as noted on *x*-ray examination—was treated for a sprained back because two days after slipping while carrying a radio he developed pain. A diagnosis of a pyonephrosis with stones was confirmed by an operation (Fig. 83).

Case III.—A man who cleaned out boilers for years developed a pain in the upper back and was given physiotherapy for occupational

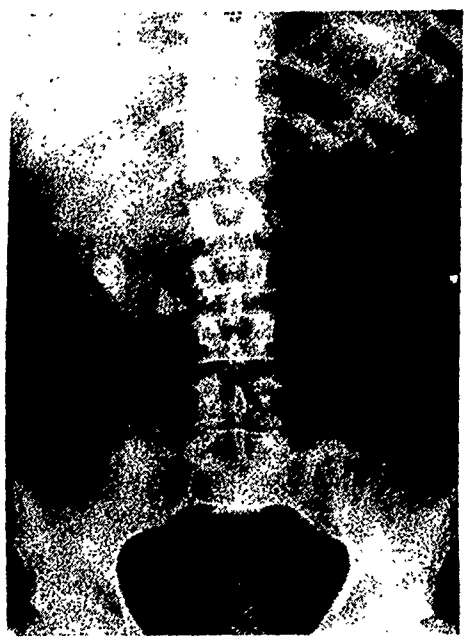


Fig. 83.—This film is of a forty-two-year-old janitor who noted back pain some hours after slipping while holding a small radio in his hands. Because of this history he had been treated for two weeks for "back sprain." Examination revealed a large palpable mass in the right quadrant of the abdomen, a temperature of 102° F., the urine loaded with pus cells, red blood cells, and albumin, and stones as shown by *x*-ray film.

or positional strain. Subsequent examination after six weeks of treatment and the payment of compensation revealed a bronchiogenic carcinoma (Fig. 84).

Case IV.—A thirty-two-year-old man developed pain at the medial border of the right scapula. He had been treated for: (*a*) back sprain, (*b*) subluxation of a vertebra, (*c*) intercostal neuritis, and (*d*) pleurisy, by regular practitioners, chiropractors, and osteopaths. Six months after the onset of symptoms he was seen by the author, at which time the following complaints were elicited: deep-seated pain

at about the middle of the medial border of the right scapula; extension of this pain (about four months after onset of the original pain) to the right shoulder and down the right arm; increasing weakness of grip in the right hand, to the point where he was attempting to educate his left hand to perform the duties ordinarily done by the right, such as using pliers, screwdriver, and the like; inability to sleep because of excruciating pain and failure of ordinary analgesics, such as anacin, to stop the pain; a sensation of swelling around the right eye; and mild, but constant, loss of weight. Cough, afternoon lag, and shortness of breath were denied.

Physical Examination.—This revealed no spasm of the muscles of the dorsal area, no limitation of motion, no increase of pain on

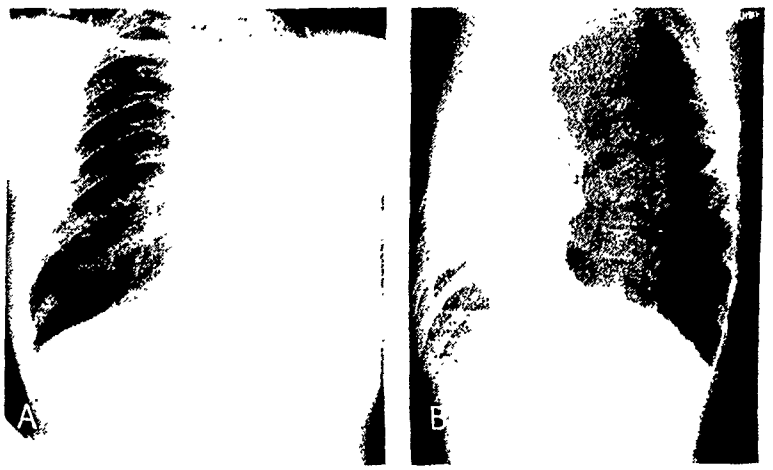


Fig. 84.—*A*, Anteroposterior view of the lungs of a forty-six-year-old boiler-cleaner, showing bronchiogenic carcinoma. Patient was treated with diathermy and massage for six weeks for "back sprain" of the dorsal area. *B*, Right oblique view of same lung as *A*. Autopsy confirmed *x*-ray diagnosis.

motion, and no tenderness to pressure over the original site of pain. Inspection of the face showed ptosis of the right eye, contraction of the right pupil and anhidrosis, constituting a typical Horner's syndrome. There was moderate atrophy of the muscles of the right shoulder girdle, with marked atrophy of the muscles of the right hand. The grip of the right hand was greatly diminished as compared to the left. Palpation of the arm showed marked loss of tone of the muscles of the forearm. The right palm was very dry (anhidrosis), while the left was moist. There was a tremor to the right hand. The lung findings were negative clinically except for a mild increase of the whispered voice sounds.

Laboratory and Roentgenographic Examination.—The routine laboratory investigations, such as urine analysis, complete blood count,

and blood Wassermann, were all negative. The *x*-ray film of the lungs revealed a clouding of the right apex, which was first considered to be a tuberculous process. At this point the neurological surgeon of our staff saw this patient in consultation. He suspected the syndrome to be a pulmonary sulcus tumor, as originally described by Pancoast, and ordered *x*-ray study of the cervical spine and ribs. *x*-Rays showed:

"In the anterior-posterior view thickening of the apex of the right lung, with a dense shadow extending over the apex and with evidence of a small amount of infiltration below it (Fig. 85).

"*x*-Ray of the cervical spine reveals no alteration of the vertebral bodies. It does show a destructive lesion of the vertebral end of the third right rib."

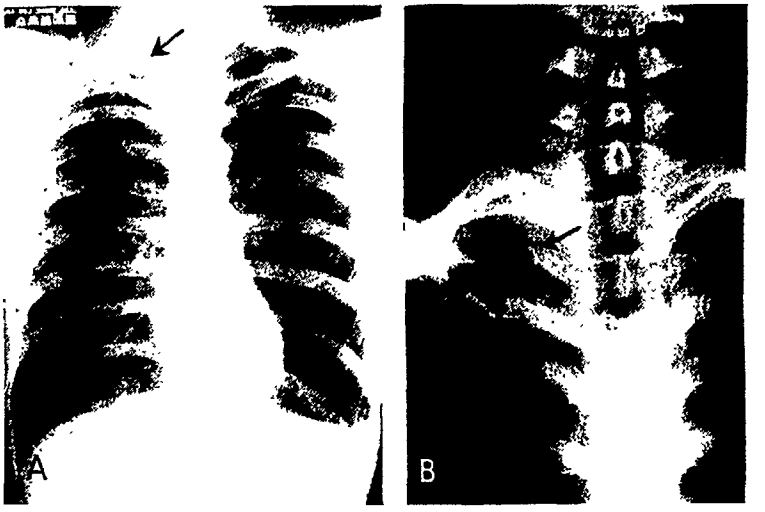


Fig. 85.—*A* and *B*, Involvement of the right apex and invasion of the right third rib at the vertebral end. Together with the clinical findings, the case represented a tumor of the pulmonary sulcus.

This case occurred just prior to the publication of this book and, therefore, no biopsy reports can be made. Clinically it conforms in every respect to the "superior pulmonary sulcus tumor" of Pancoast. In 1924 Pancoast[13] reported three cases of what appeared to him to be a new entity among intrathoracic tumors. He noted that "the tumors in question seemed to occur at the thoracic inlet, were characterized clinically by pain around the shoulder and down the arm, Horner's syndrome, atrophy of the muscles of the hand and roentgenographic evidences of a small, homogeneous shadow at the extreme apex of the lung, with always more or less local rib destruction and often vertebral infiltration."

Pancoast believed that the tumor had its origin from an embryonic remnant of the fifth branchial pouch and represented an epithelioid carcinoma of extrapulmonary origin. In 1932 he reported four additional cases. Since then the clinical entity has been fairly well established, but concerning the pathological origin of the tumor there has existed much controversy.[14-21]

Until further study is made, our case cannot be reported pathologically as a Pancoast tumor, but clinically it conforms to the classical picture.

EXPLANATION OF SPINAL ANOMALIES

From the *x*-ray files of our clinic, a number of films have been selected which depict the various congenital anomalies described below. In some instances these defects have been amplified by drawings. It is hoped that these will present an adequate roentgenological and graphic study of spinal anomalies. The drawings used to illustrate the various movements involving the lower back have been done after the manner of those used by Brahdy.[22]

It has been pointed out that in view of the extension of the Workmen's Compensation Act to include the occupational diseases, the general practitioner is confronted with an obligation not heretofore experienced. The so-called "industrial back" is an example, as has been emphasized in the present chapter. It is not unfair to state that in general the office treatment of back pain has consisted of strapping the back, without investigating the cause. When, however, a back disability has a medicolegal aspect and investigation reveals certain anomalies which could account for the disability, the general practitioner is expected to interpret their meaning despite his lack of special training or experience. An appreciation of such irregularities as asymmetry of facets, sacralization, and spondylolisthesis has been limited largely to the orthopedist. Now, owing to the expansion of industrial medicine, the general practitioner also must understand their significance. To the writer's knowledge, no textbook in general medicine or surgery attempts to define these congenital defects. Therefore, this chapter is augmented by an explanation of these conditions.

Asymmetry of Facets (Facet Irregularities, Anomalies of the Articular Processes).—Reverting to *Gray's Anatomy*, a typical vertebra is described as consisting of two essential parts, an anterior segment, or the body, and the posterior part, which is the vertebral or neural arch. These enclose a foramen, the vertebral foramen. The vertebral arch consists of a pair of pedicles and a pair of laminae, and supports seven processes; namely, the four articular, two transverse, and one spinous. The cervical, thoracic, and lumbar vertebrae differ

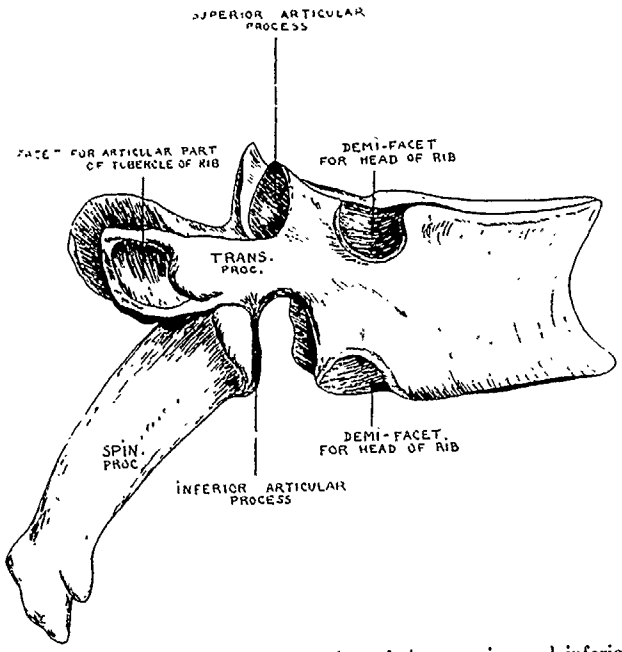


Fig. 86.—A thoracic vertebra. Note the direction of the superior and inferior articular facets.

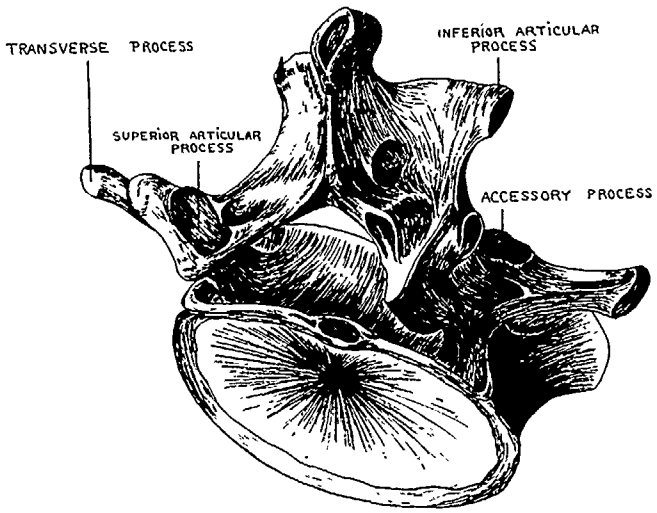


Fig. 87.—A lumbar vertebra as seen from above, showing the superior and posterior aspects. Note the plane of the superior and inferior articular facets.

in their arrangement of these parts. While the cervical vertebrae present an occasional anomaly, such as the cervical rib or failure of

fusion of the spinous process, the majority of anomalies are to be found in the thoracic and lumbar vertebrae, especially the lumbar. Normally in the thoracic (dorsal) vertebrae the superior articular processes are thin plates of bone projecting upward from the junction of the pedicles and laminae; their articular facets are practically flat, and are directed backward and a little lateralward and upward. The inferior articular processes are fused to a considerable extent with the laminae, and project but slightly beyond their lower borders; the facets are directed forward, a little medialward and downward (Fig. 86). The superior and inferior articular processes of the lumbar verte-

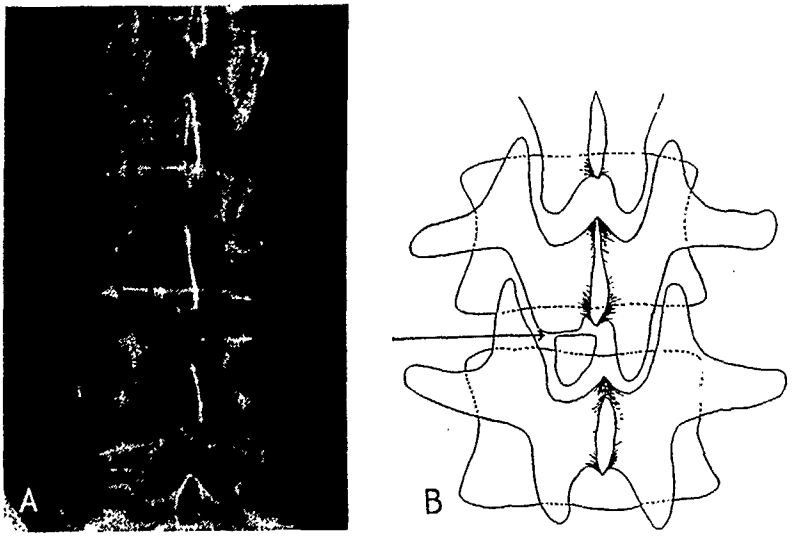


Fig. 88.—*A*, Failure of fusion—a thin fissure line—of the left inferior articular process of the third lumbar vertebra. This defect on the *x*-ray film is invariably faint and hard to detect. *B*, The arrow indicates the failure of fusion of the inferior process with a fissure line which is purposely exaggerated. On *x*-ray film this line is never so distinct, but on the contrary rather faint and therefore often overlooked.

brae are well defined, projecting respectively upward and downward from the junction of the pedicles and laminae. The facets of the superior processes are concave and face backward and medialward; those on the inferior are convex and are directed forward and lateralward (Fig. 87). When the facets do not face in a normal manner as just described, they are said to be asymmetrical, or the condition is referred to in the literature as anomalies of the articular processes. The surfaces of the articular facets are covered with cartilage, and the articular capsules of these joints are lined with synovial membrane. Hence they are susceptible to changes resulting from infection.

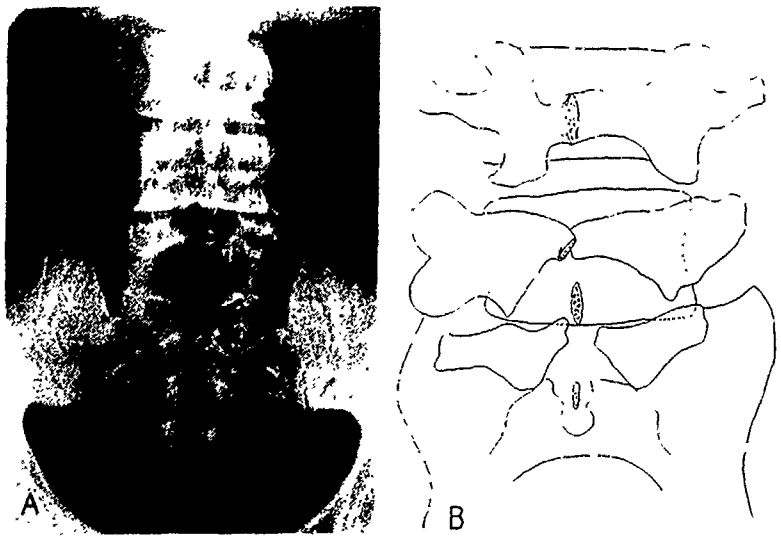


Fig. 89.—*A*, Spina bifida occulta. The defect includes the last lumbar and the first and second sacral segments. *B*, Spina bifida occulta as shown in *A*.

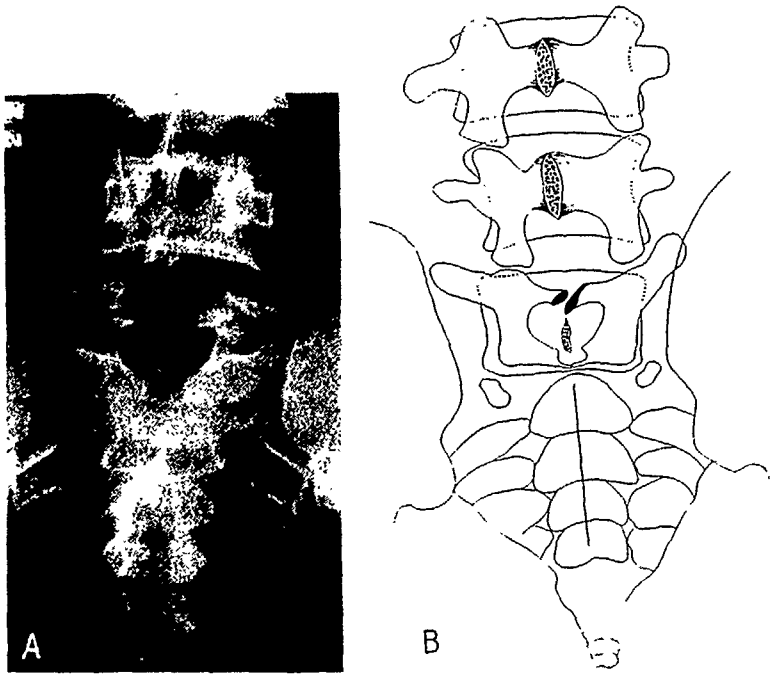


Fig. 90.—*A*, Failure of fusion of the spinous process of the fifth lumbar vertebra, as well as the first sacral segment. The congenital defect of the fifth lumbar had been interpreted as a fracture, the patient being placed in a body cast. *B*, To show the failure of fusion, the drawing of the film purposely overemphasizes the defect.

degenerative processes and trauma, especially if the primary irritation has been induced by alteration of their normal plane of direction. A further anomalous condition of the articular processes is the fissure line which is occasionally seen (Fig. 88). This line is so fine that it often escapes detection and when seen is sometimes mistaken for a fracture line.

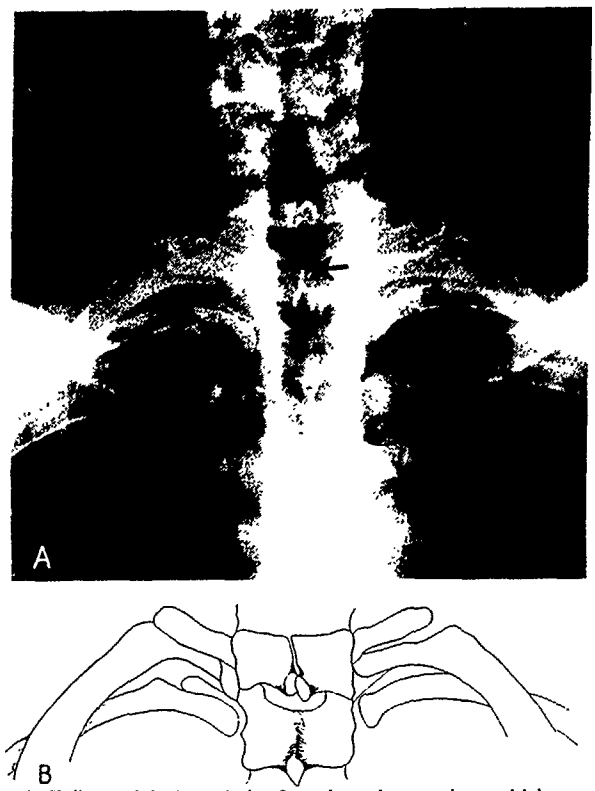


Fig. 91.—A, Failure of fusion of the first thoracic vertebra, which was interpreted as a fracture line. A body cast was applied, following which the patient developed a neurosis characterized by marked tremor of the head and right arm. *B*, Congenital defect of first thoracic vertebra as shown in *A*.

Spina Bifida Occulta.—This term refers to a failure of fusion of the spinous processes of the fifth lumbar and the first sacral segment. Occasionally more than the first sacral segment has failed to fuse. Inspection of the back reveals no evidence of this defect, although in rare instances there may be a small lipoma, tuft of hair, or a dimple in the lumbosacral region to indicate its presence. It is usually noted only by *x*-ray and rarely is of significance, although it may constitute an instability of the lower back (Fig. 89).

Failure of Fusion.—This congenital defect occurs in various parts of the posterior portion of the vertebra (the neural arch). It may be seen in one or more of the articular processes and more commonly in the spinous process. It is most commonly seen in the lumbar area, although occasionally elsewhere (Figs. 90, 91). In itself and without other accompanying congenital defects it is not a cause of back pain, but it is sometimes mistaken for a fracture line.

Spondylolisthesis.—This term is applied to that deformity of the lumbosacral region which consists of a *gradual* slipping forward of the fifth lumbar vertebra on the sacrum (Figs. 92–94). It was first

Fig. 92.—*A*, Spondylolisthesis of the fifth lumbar vertebra. *B*, The broken line indicates the extent of slipping forward of the fifth lumbar upon the sacrum. Note the narrowed foramen and the complete failure of fusion of the neural arch.

noted by obstetricians and considered a disorder among women, but subsequent study reveals it to be more prevalent in men. It is the result of a variation in the ossification of the fifth lumbar vertebra where, instead of one primary center for each half of the vertebral arch, there exist two primary centers in each half, the two parts being united by cartilage extending between the superior and inferior articular processes, the union being effected by hyaline cartilage only. Normally the apposition of the inferior articular processes of the fifth lumbar vertebra with the articular processes of the sacrum prevents any forward displacement of the fifth vertebra. But if this

continuity does not exist, displacement is apt to occur owing to the strain of the superimposed body weight, which in time overcomes the resistance of the muscular and ligamentous structures. From a medico-legal aspect it is important to appreciate that this slipping takes place gradually and over a period of years. Claims that a sudden strain or lift produced the condition are to be discounted. There is no set age when this condition progresses to the point that disability ensues, but it is almost always after the muscles and ligaments lose the tone of youth.

Fig. 93.

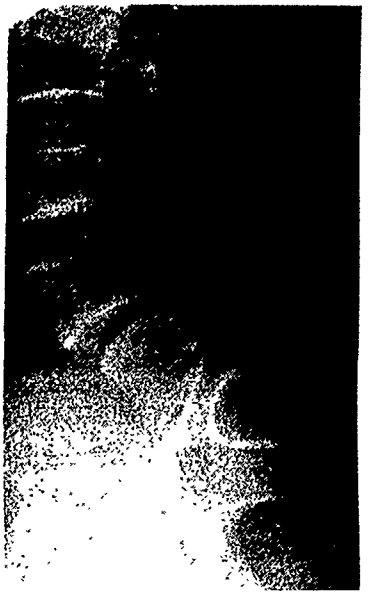


Fig. 94.

Fig. 93.—Spondylolisthesis of the fifth lumbar vertebra. Note the horizontal sacrum.

Fig. 94.—Spondylolisthesis of the fourth lumbar vertebra. Note failure of fusion of the neural arch as well as narrowed intervertebral space between the fifth lumbar and sacrum. No history of injury. Pain first experienced while pulling out a cabinet file drawer.

Spondylolysis.—This is a term used frequently to indicate a congenital failure of fusion in the neural arch, predisposing to a spondylolisthesis but before any slipping has occurred (Fig. 95).

Sacralization.—This term refers to an elongation of the transverse process of the fifth lumbar vertebra to the point where it articulates with or is in close connection with the sacrum or sometimes the ilium. It is most often unilateral, but frequently bilateral. It is often referred to in the literature as a "butterfly wing" (Figs. 96, 97, 98). Its pres-

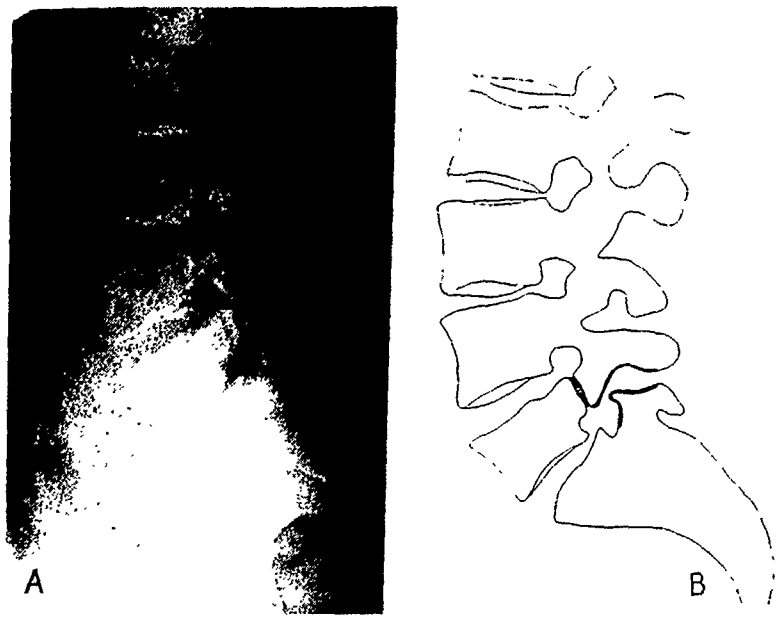


Fig. 95.—*A*, Spondylolysis. There is a complete failure of fusion of the neural arch between the fifth lumbar vertebra and the sacrum. It will be noted that no slipping has occurred. (Male, aged eighteen.) *B*, Drawing of spondylolysis. This defect prior to slipping is not frequently seen.

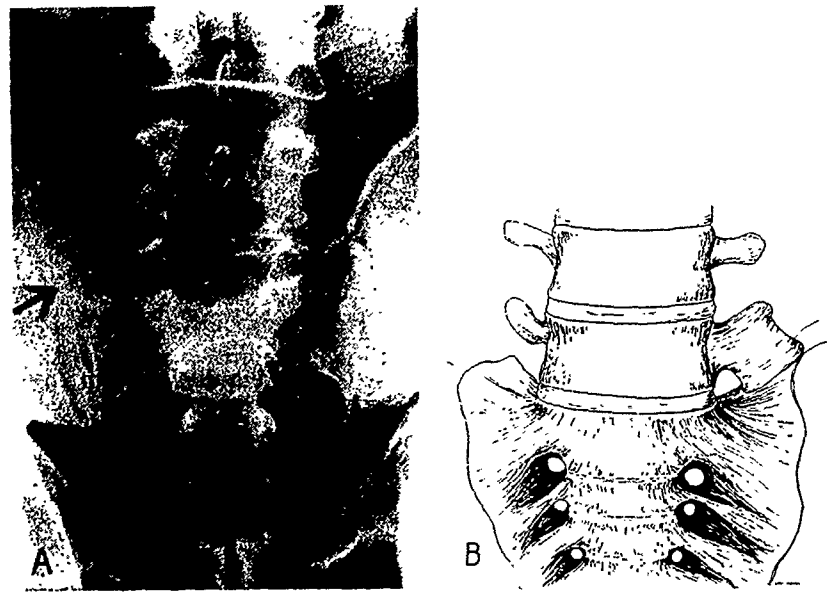


Fig. 96.—*A*, Unilateral sacralization (right) with sclerosis of the articulation with the sacrum. Note the tilting of the fifth lumbar vertebra. *B*, Unilateral sacralization. The left transverse process of the fifth lumbar vertebra is enlarged and has extended downward to fuse with the sacrum.

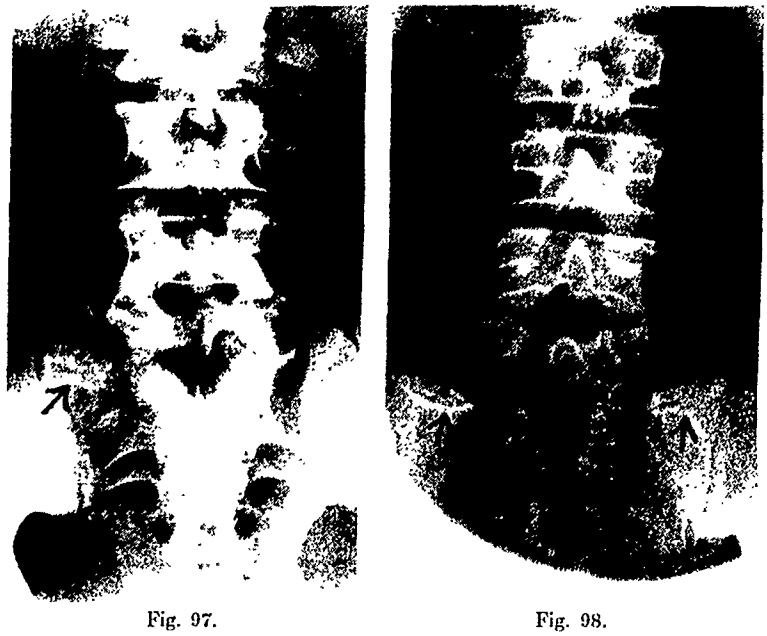


Fig. 97. Fig. 98.

Fig. 97.—Unilateral sacralization (right) with spina bifida occulta.
Fig. 98.—Bilateral sacralization, with marked sclerosis, in a thirty-eight-year-old auto mechanic who claimed frequent "back sprains" after minor effort.

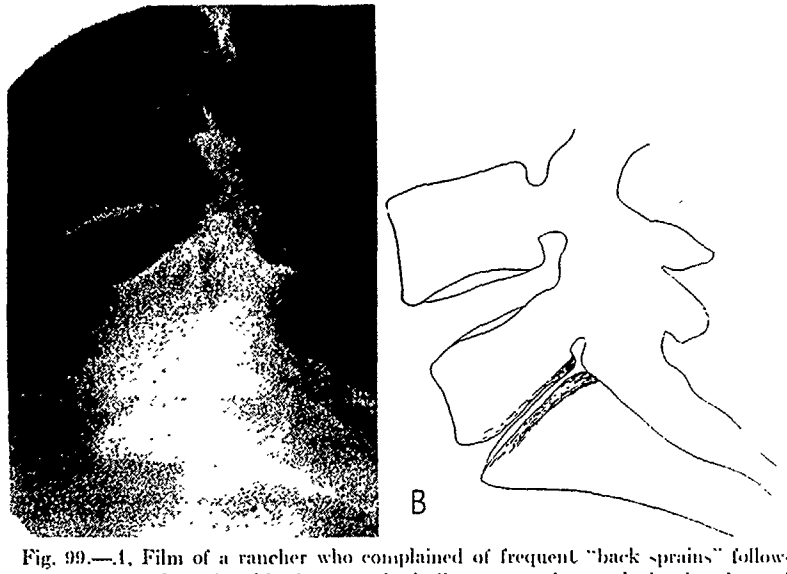


Fig. 99.—A, Film of a rancher who complained of frequent "back sprains" following minor effort. Note the thin intervertebral disc space, the marked sclerosis, and the obliteration of the foramen. *B*, Marked narrowing of the intervertebral space with encroachment upon the foramen. Sclerosis of the lower surface of the fifth lumbar vertebra and the upper surface of the sacrum indicative of chronic irritation.

ence may be symptomless, but frequently it may be the sole cause of back pain, especially if it distorts the position of the fifth lumbar vertebra.

The Lumbosacral Angle.—With but slight alteration of the angle, the sacrum should continue downward from the vertebral column without much alteration. If the sacrum is elevated, it approaches the horizontal position. When this occurs, there is encroachment upon the posterior angle of the intervertebral space and thus narrowing of the associated foramen. Narrowing of the posterior angle of the intervertebral space between the fifth lumbar vertebra and sacrum

Fig. 100.—Hemivertebra at the fifth lumbar joint, which is a congenital condition due to the absence of one ossification center. Such an anomaly is accompanied by rotation of the bodies and scoliosis as noted here.

is not always the result of alteration of the angle of the sacrum. Static changes and the wear and tear of normal activity may so alter the intervertebral space that encroachment of the foramen takes place (Fig. 99). Of all the conditions not associated with trauma, the narrowing of the lumbosacral disc space constitutes the most frequent cause of chronic back pain in the author's opinion (Figs. 100–105).

Diagnostic Signs by Manipulation.—Unfortunately, most of these diagnostic signs bear the names of their designers. Their indications are not finite, for they are based upon the theory which each individual author of these signs accepts. Broadly, the straight-leg-raising

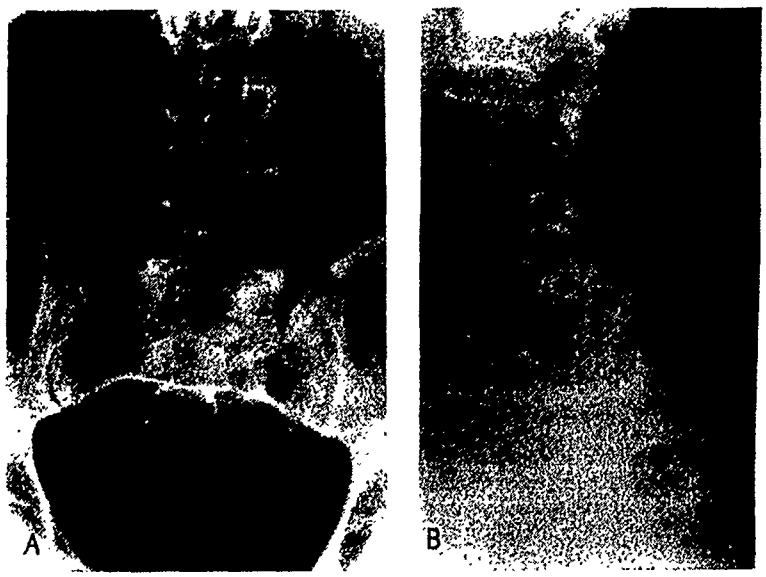


Fig. 101.—*A*, Anteroposterior view. The spinous process of the fourth lumbar vertebra extends upward, almost to touch that of the third to form what appears to be a line of cleavage. The laminae of the last lumbar vertebra have failed to fuse. *B*, Lateral view. The sacrum is horizontal, increasing the lumbosacral angle.

Fig. 102.—Spina bifida occulta of the first two sacral segments, asymmetry of development of the last lumbar body with sacralization on the right. The fissure line produced by the encroachment of the right fourth articular process upon the lateral process tends to form a joint.

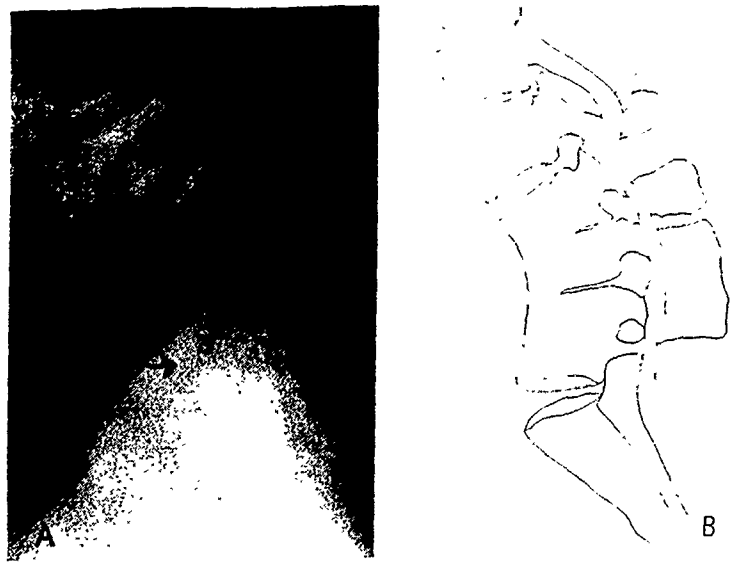


Fig. 103.—*A*, Fusion of the anterior portion of the bodies of the third, fourth, and fifth lumbar vertebrae. *B*, Drawing shows this fusion and the incomplete intervertebral spaces.

Fig. 104.

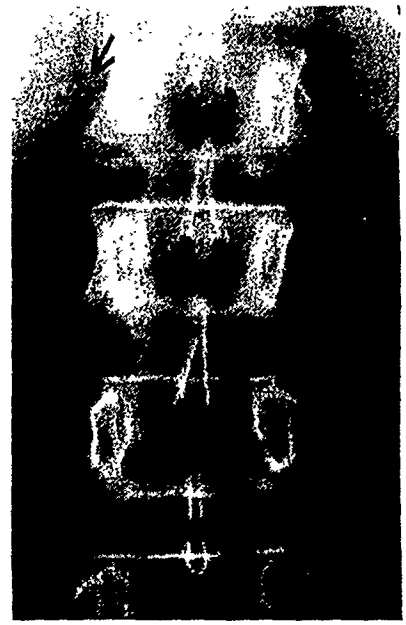


Fig. 105.

Fig. 104.—Narrowing of the space between the fourth and fifth lumbar bodies with posterior fusion.

Fig. 105.—Congenital first lumbar ribs. Patient developed a neurosis after being told he had fractures of the transverse processes which would require an operation.

test and its variations concern the lumbar muscles and the posterior sacral attachments, while the extension-abduction-rotation signs point

Fig. 106. Fig. 107. Fig. 108.

Fig. 106.—Normal forward flexion from the erect position, accompanied by movement of both spine and pelvis.

Fig. 107.—Forward flexion in which the pelvis has rotated to some extent but not accompanied by flexion of the spine.

Fig. 108.—Forward flexion of the spine but not accompanied by any pelvic rotation.

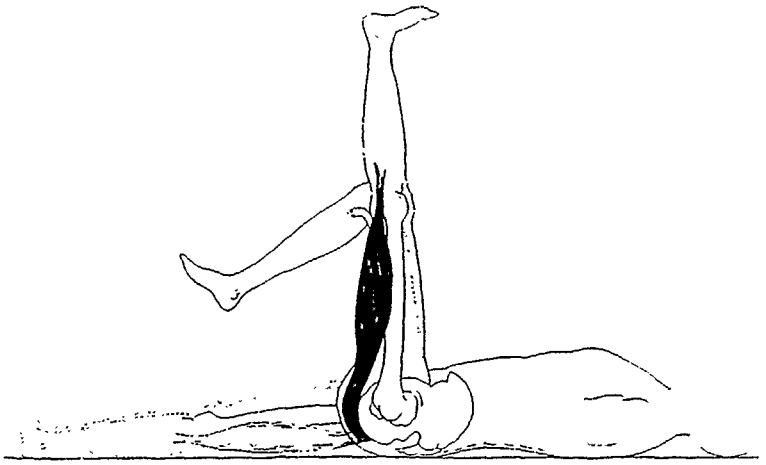


Fig. 109.—The straight-leg-raising test is illustrated by this drawing. It shows the decrease in the distance from the origin of the hamstring muscle to its point of insertion when the knee is flexed as compared to the distance when the knee is extended.

to involvement of the hip. The following is a description of the more commonly mentioned signs (Figs. 106–113).

Lasègue's Sign.—This test was designed to distinguish between sciatica and hip-joint disease. The patient lies on his back on the

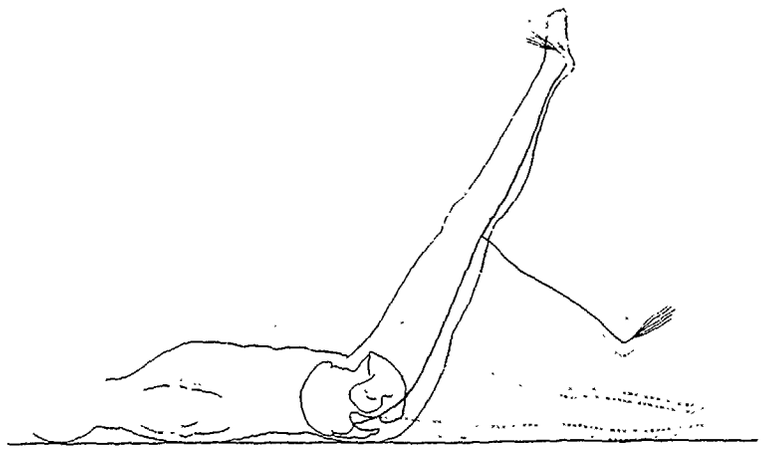


Fig. 110.—This drawing is designed to show the pull on the s iatic nerve when flexion of the hip is engaged in with the knee extended. Dorsiflexion of the foot produces a similar effect. Tension on the nerve is relieved when the knee is flexed.

Fig. 111.

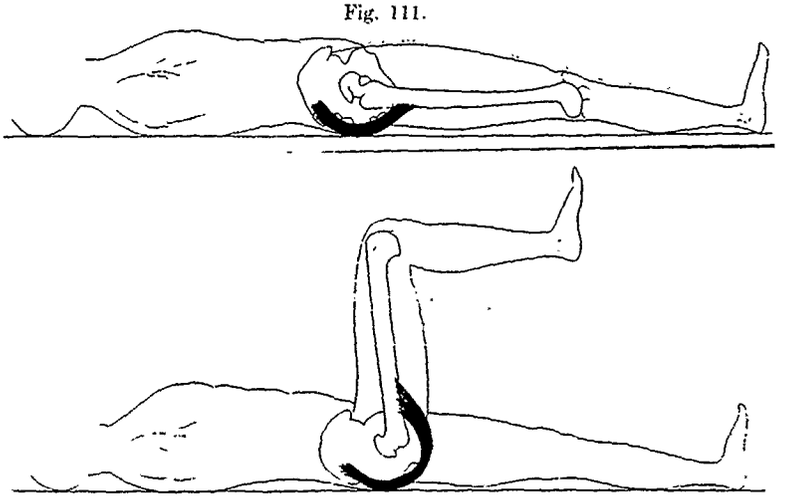


Fig. 112.

Fig. 111.—The specimen is in the supine position showing the gluteus maximus muscle. Just anterior to the muscle lies the ischiogluteal bursa.

Fig. 112.—Flexion of the thigh with stretching of the gluteus maximus muscle.

examining table and raises the leg with the lower leg straight (extended). The normal person should be able to flex the hip through a range of 90 degrees. In sciatica, pain is produced if the knee is ex-

tended when the leg is raised, but no pain occurs in the same motion if the knee is flexed. In hip-joint disease flexion of the hip is painful whether the knee is flexed or extended.

Bechterew's Sign.—In sciatica, straight-leg-raising on the opposite side may cause pain on the affected side to a less degree, since the stretched nerve on the unaffected side pulls upon the nerve roots of the opposite side by its attachment in the cord.

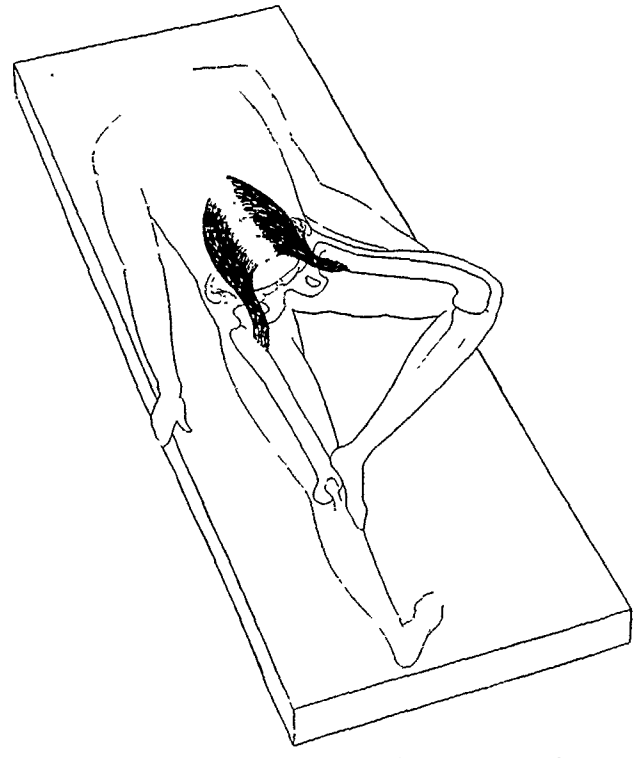


Fig. 113.—Lesions of the hip joint may produce spasm of the psoas magnus muscle. This drawing shows the left heel placed on the patella of the opposite leg. Pushing downward on the left knee produces outward rotation and abduction of the thigh which stretches the psoas magnus. If this muscle is in spasm due to hip-joint disease, the movement is painful and limited.

Demianoff's Sign.—The following test is to differentiate lumbago from sciatica. The straight-leg test is first done. If it is positive, it is repeated with the examiner's one hand holding the pelvis of the patient firmly to the table. In sciatica the pain is still present, but in lumbago it disappears.

Gaenslen's Sign.—One hip is flexed, and at the same time the other hip is extended. Then the procedure is reversed. The test supposedly

differentiates lumbosacral from sacro-iliac lesions and between right- and left-sided involvement. The patient lies on his back on the examining table. The knee and hip of one leg are flexed as far as possible and held in that position by the patient's hands clasped over the knee. The other leg at the edge of the table is allowed to hang and is further pressed down by the examiner, producing hyperextension of the hip. Pain is said to occur on the affected side.

Ober's Sign.—The patient lies on the unaffected side while the examiner places one hand on the pelvis to steady it and grasps the patient's ankle with the other, moving the leg until the knee is flexed at right angles. The thigh is abducted and extended in the coronal plane of the body. Ober contends that if contracture of the iliotibial band is present, the leg will remain abducted.

Ely's Sign.—With the patient prone, hyperflexion of the knee may cause elevation of the pelvis on the same side.

Linder's Sign.—If enforced passive flexion of the head occurs with the patient sitting or recumbent and the legs outstretched, pain may arise in the lumbar region or down the leg on the affected side. It is thought that a positive reaction indicates root sciatica.

Neri's Sign.—When the patient in a standing position forward-flexes the torso, there is noted flexion of the knee on the affected side.

Soto-Hall's Sign.—With the patient lying flat on his back, flexion of the spine beginning at the neck and proceeding downward, pain may be noted at the site of the lesion.

TREATMENT

Educational Campaign in Prophylaxis.—Industry as yet has not fully awakened to the tremendous cost of the disabling back, nor are any general preventive measures being adopted. True, while these measures are not so simple as merely installing ventilation or exhaust systems, the incidence of the disabling back can undoubtedly be reduced. Education of the industrial public upon the dangers residing in certain types of moving parts, punches, blades, and the like, by a safety-first campaign greatly reduced the severe traumata occurring in industry. Such a campaign should be similarly directed toward back disabilities. Moving-pictures of correct and incorrect methods of lifting, pushing, or pulling could be shown; and posters placed so as to catch the workman's eye would warn against faulty tendencies. Periodic lectures by one trained in physical education would be extremely valuable. Such a person could service a number of plants during a month's time, each company sharing the cost of his salary. Setting-up exercises twice daily would relieve the monotony of work and strengthen the muscles. If they are of value to nondescript soldiers

entering the army, they would also be of aid in industry. And, finally, careful selection of the right type of men for the various jobs should be attempted. How frequent, and at the same time how ridiculous, it is to see a frail, undersized laborer struggling with a heavy object, while close by stands a flagman or gateman of normal stature and physically fit!

Specific Treatment.—This depends upon the determination of the etiological factors. Actual sprain, strain, or contusion is best treated by conservative measures. In our clinic approximately eleven hundred such patients are treated each year by conservative procedures with very satisfactory results, particularly in regard to shortening the period of disability.

Physiotherapy.—A routine of physiotherapy treatment daily or on alternate days, depending upon the severity of the case, is usually instituted. This physical adjunct to treatment is useless unless carried out adequately. This physiotherapy does not consist of carelessly placing the patient under an infra-red lamp for from fifteen to thirty minutes. Instead, he is first given from fifteen to twenty minutes of infra-red therapy, and this is followed by fifteen or twenty minutes of diathermy. Finally, a gentle but adequate massage by a trained physiotherapist is given to the involved parts. For the first three to seven days we try to avoid strapping the patient's back, so that at home he may apply heat more effectively to the low back area. He is encouraged to take hot sitz baths for from thirty to forty minutes twice daily and to apply controlled electric pads, small heating lamps, hot-water bottles, or hot applications to the involved area, in addition to the baths. Most of these patients will be more comfortable and will improve more rapidly if boards are placed lengthwise on top of the springs underneath the mattress. During this period, acetylsalicylic acid, from 10 to 15 grains (0.65 to 1 gm.) four time daily, perhaps in combination with codeine phosphate, from ½ to 1 grain (0.0325 to 0.065 gm.), may be needed, and one of the barbiturates may be necessary to obtain adequate sleep. At the end of this time if the patient still has back pain and a feeling of weakness, the low back is strapped adequately with three-inch adhesive tape (Fig. 114). It may be necessary or desirable in some to apply the strapping when the patient is first seen, but if this is done it should be removed for physiotherapy treatments and then replaced.

Exercise.—We feel that it is particularly important to encourage these patients to be as active as their discomfort will permit. In our experience, hospitalization with absolute bed rest and the sometimes advised traction, definitely prolong the period of disability. The patient appears to develop a tenseness and tightness of the low back

from such procedures and for these requires an additional period of treatment. In this encouragement of the patient to remain active, exercises play an important rôle. These should be outlined carefully, and it is particularly important to attempt by some exercises to correct faulty posture. These may be started at the end of seven or ten days, although by this time many of our patients will have returned to active work or are ready to do so. This matter of activity is similar to some extent to the treatment of the sprained ankle, as outlined by Alexander,[23] in which definite shortening of the usual period of disability is obtained by the use of novocaine injection, strapping, and early use of the ankle.

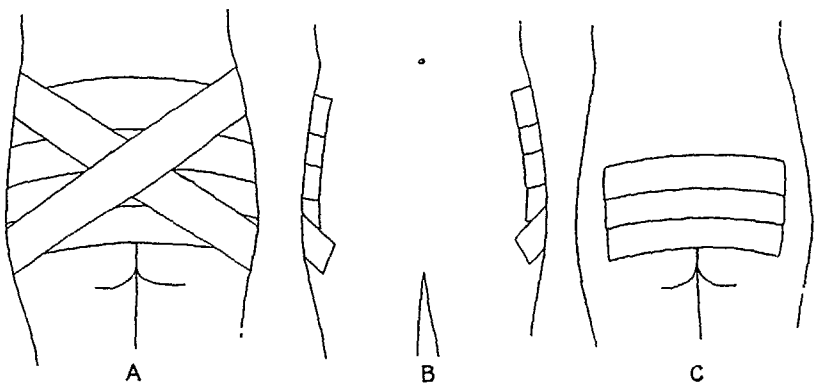


Fig. 114.—*A*, The back is first painted with compound tincture of benzoin to protect the skin. Three or four strips of 3-inch adhesive tape are then applied so that they extend over the flanks onto the abdomen. On top, these two adhesive straps are crossed so that they anchor each anterior superior iliac crest. Posterior view. *B*, Anterior view. *C*, Short strips of tape such as illustrated here are inadequate and of no value.

These back sprains and contusions do not need casts, braces, or belts, and their use may actually do harm in many instances and prolong disability.

Manipulation.—Manipulation has its place, although its sphere of application is relatively small. Occasionally dramatic results are obtained from its use, but also occasionally great aggravation of the pain is brought about by it. Not infrequently we have referred to us patients who have been manipulated and whose pain has been made much worse by the procedure. In the hands of physicians trained in this art, it may be of some value. It cannot be too strongly urged, however, that before resorting to manipulation, a thorough history, physical examination, and investigation as outlined earlier in this chapter be done, since much harm may follow manipulative treatment

of such conditions as a herniated nucleus pulposus, a cord tumor, an acute arthritis, or disease residing in adjacent organs. Details of these manipulative procedures are outlined in standard orthopedic texts.

Eucipin in Oil.—Much relief of pain in these patients and much shortening of the period of disability has been reported from the use of injections of eucipin in oil. Irwig[21] outlined the following technique:

With the patient lying on the abdomen, the sacro-iliac region is surgically sterilized. A 5 cc. syringe with a 1½ inch 21-gauge needle is used for the injection. To locate the point of injection, the course of the lumbar spine and the crest of the ilium posteriorly are palpated. A line is drawn parallel and 1 in. to 1½ in. lateral (according to thickness of spine) to the spinous processes of the vertebrae. A second line is drawn along the upper border of the ilium and continued posteriorly until the two lines intersect in the lumbo-sacral region of the back. This is the point of injection. The needle is inserted vertically to its hilt with a quick pressure, the plunger of the syringe is withdrawn slightly, and if no blood is aspirated, the injection of 2 cc. of eucipin solution in oil is begun. The greater amount of the oil is deposited in the deeper parts. The injection is continued as the needle is withdrawn and should be completed when less than one-third the length of the needle is above the surface of the skin. This procedure obviates the injection of the oil solution into the superficial tissues. The parts should be massaged vigorously after the injection. The needle does not endanger vital structures on full insertion.

He used:

Eucipin base	0.1
Ethylaminobenzoate	3.0
Benzyl alcohol	5.0
Oil sweet almond	q.s. ad. 100.0

He later found that eucipin solution in 2 per cent iodized oil possessed even greater therapeutic possibilities than the plain oil. He pointed out that such injections should not be considered a panacea for all cases of low back pain. In our experience this procedure has not greatly added to the treatment outlined. Perineural injections of 0.1 per cent β-eucaine in normal saline were also employed around the sciatic nerve in some instances by Irwig.

The treatment of those cases of back pain felt to arise from causes other than trauma, *e.g.*, foci of infection, will not be discussed here, since they, generally speaking, will not fall into the classification of compensation cases.

Correction of Congenital Defects.—Cases in which congenital defects are demonstrated, and on which an acute low back pain sprain

is superimposed, frequently respond well to conservative therapy as outlined, and this should first be tried. In these the use of braces or back supports may become necessary later and be of much value.

Operative Treatment.—As a last resort various operative procedures have been described. These in most instances should not be performed until an adequate period of conservative therapy has been followed, and, if possible, financial settlements made, although this latter is frequently not allowed by industrial accident commissions. Ober[25] described the use of fasciotomy in cases in which there was demonstrated a contracted iliotibial band as the cause of sciatica and back pain. Gratz[11] has used air insufflation for treatment of back pain thought to be due to fascial adhesions. Haggart,[26] after a definite trial of conservative treatment, treated cases of unstable lumbosacral joints by spine fusion. He stated that fully 95 per cent of patients with chronic low back pain were best treated by a conservative regimen, and only 2.5 per cent of 2000 patients with low back pain seen by him in a three-year period were subjected to arthrodesing operations.

Operation for herniation of a nucleus pulposus, or hypertrophy of the ligamentum flavum, may at times be necessary, although here again if such morbid anatomy is demonstrated, the rôle trauma played in its production needs to be very carefully weighed before responsibility is assigned to a specific, and frequently minor, incident. Here again conservatism and an adequate period of observation should be followed at first.

In compensation work, too, the end-result is frequently not so gratifying as that described following operations in private practice, since the desire for a large financial settlement frequently causes continuation of the previous complaints or the simulation of new ones, even though the cause of the backache has been removed.

A regimen for the correction of structural defects should be directed by an orthopedist; and diagnosis and surgical correction of herniated nucleus pulposus or hypertrophied ligamentum flavum should be made by a neurosurgeon.

REFERENCES

1. Goodwyn, T. P.: "Effect of Trauma on Mechanically Weak Back," *Am. J. Surg.*, *42*: 577–581 (Dec.), 1938.
2. Putti, V.: "New Concepts in Pathology of Sciatic Pain," *Lancet*, *2*: 53–60 (July 9), 1927.
3. Hodges, F. J., and Peck, W. S.: "Clinical and Roentgenological Study of Low Back Pain with Sciatic Radiation; Roentgenological Aspects," *J. Roentgenol.*, *37*: 461–466 (April), 1937.
4. Williams, P. C.: "Lesions of Lumbosacral Spine; Acute Traumatic Destruction of

Lumbosacral Intervertebral Disc," *J. Bone and Joint Surg., 19:* 343, 363 (April), 1937.

5. Barr, J. S., Hampton, A. O., and Mixter, W. J.: "Pain Low in Back and 'Sciatica' Due to Lesions of Intervertebral Discs," *J.A.M.A., 109:* 1265–1270 (Oct. 16), 1937.
6. Ober, F. R.: "Role of Iliotibial Band and Fascia Lata as Factor in Causation of Low Back Disabilities and Sciatica," *J. Bone and Joint Surg., 18:* 105–110 (Jan.), 1936.
7. Judovich, B. D., and Bates, W.: "Low Back Pain: Study of Over 1,000 Cases," *Indust. Med., 8:* 160–165 (April), 1939.
8. Steindler, A.: "Differential Diagnosis of Pain Low in Back; Allocation of Source of Pain by Procain Hydrochloride Method," *J.A.M.A., 110:* 100–113 (Jan. 8), 1938.
9. Makaroff, W. N.: "Etiology of Lumbago and Sciatica," *Indust. Med., 8:* 1–4 (Jan.), 1939.
10. Ridlon, J., and Berkheiser, E. J.: "Calcareous Degeneration of the Dorsal and Lumbar Aortae as a Cause of Backache," *J.A.M.A., 80:* 1831–33 (June 23), 1923.
11. Gratz, C. M.: "Fascial Adhesions in Pain Low in Back and Arthritis," *J.A.M.A., 111:* 1813–18 (Nov. 12), 1938.
12. Willis, T. A.: "Backward Displacement of the Fifth Lumbar Vertebra; Optical Illusion," *J. Bone and Joint Surg., 17:* 347 (April), 1935.
13. Pancoast, H. K.: "Importance of Careful Roentgen Ray Investigations of Apical Chest Tumors," *J.A.M.A., 83:* 1407 (Nov. 1), 1924.
14. Pancoast, H. K.: "Superior Pulmonary Sulcus Tumor, Tumor Characterized by Pain, Horner's Syndrome, Destruction of Bone and Atrophy of Hand Muscles," *J.A.M.A., 99:* 1391 (Oct. 22), 1932.
15. Browder, J., and DeVeer, J. A.: "The Varied Pathologic Basis for the Symptomatology Produced by Tumors in the Region of the Pulmonary Apex and Upper Mediastinum," *Am. J. Cancer, 24:* 507 (July), 1935.
16. Jacox, H. W.: "Superior Pulmonary Sulcus Tumor," *J.A.M.A., 103:* 84 (July 14), 1934.
17. Steinder, P. E., and Byron, F. F.: "Primary Lung Carcinoma," *Am. J. Cancer, 22:* 776 (Dec.), 1934.
18. Morris, J. H., and Harken, D. E.: "The Superior Pulmonary Sulcus Tumor of Pancoast in Relation to Hare's Syndrome," *Ann. Surg., 112:* 1 (July), 1940.
19. Clark, B. E.: "Superior Pulmonary Sulcus Tumor (Pancoast)," abstr., *Am. J. Path., 10:* 693 (Sept.), 1934.
20. Fried, B. M.: "Sternoclavicular Branchioma," *Am. J. Cancer, 25:* 738 (Dec.), 1935.
21. Graef, I., and Steinberg, I.: "Superior Pulmonary Sulcus Tumor," *Am. J. Roentgenol., 36:* 293 (Sept.), 1936.
22. Brahdy, L.: "Mechanics of Physical Signs in Lower Trunk Injuries," *Surg., Gynec. and Obst., 60:* 802–817 (April), 1935.
23. Alexander, H. H., Jr.: "Treatment of a Sprained Ankle," *Am. J. Surg., 50:* 581–584 (Dec.), 1940.
24. Irwig, F.: "Treatment of Low Back Pain," *Indust. Med., 8:* 105–112 (March), 1939.
25. Ober, F. R.: "Back Strain and Sciatica," *J.A.M.A., 104:* 1380–83 (May 4), 1935.
26. Haggart, G. E.: "Early Operation (Spine Fusion) in Unstable Lumbo-sacral Joints," *J.A.M.A., 115* (Dec. 21), 1940.

CHAPTER XXXI

HERNIA

HERNIA, like the "industrial back," cannot be considered an industrial disease in the true sense of the word, yet its presence in the workman is invariably attributed to occupational activity. The diagnostic features of a hernia are familiar to every practitioner and need not be discussed. Likewise, the cause of a hernia is well understood, and medical opinion in this respect has not changed with the passing years, but the legal attitude has. Originally, before a hernia was considered to be compensable, certain prerequisites had to be fulfilled in the establishment of the claim. These are now being ignored by the referees of our industrial accident commissions not only in California, where I am familiar with the opinions rendered, but in most other states. In this situation the physician has become a sort of nonentity. It is imperative that either the legal attitude be clarified or the status of a hernia be reclassified.

Compensability of Hernia.—Some states exclude hernia from the list of compensable conditions unless it is the result of direct trauma. Certain other states consider hernia as compensable if it occurred after unusual exertion or sudden strain, and in some commonwealths, as in California, the act does not specifically mention hernias. Their compensability is tested the same as any other incident occurring in industry for which benefits are claimed. The policy followed originally by the California commission was that if an inguinal hernia followed an industrial overexertion or strain it was considered compensable. They held:

> It is unimportant whether, from a medical or scientific standpoint, hernia be classified as a disease or a malformation; or be otherwise designated; or that the employee be predisposed thereto. When a hernia results from a strain during the course of and arising out of the employment, and is given impetus to appear at that time and place, the injured man is entitled to compensation.[1]

In view of the subsequent discussion, the reader is asked to note the phrase *given impetus to appear at that time and place* quoted in the previous sentence.

Prerequisites to Compensation.—With but slight variation the majority of states list the following prerequisites as necessary in considering a hernia compensable:

1. That the hernia is of recent origin
2. That its appearance was accompanied by pain, discoloration, and evidence of a tearing of the tissues
3. That it was immediately preceded by some strain arising out of and in the course of employment
4. That a protrusion or mass appeared in the area immediately following the alleged strain

In some states decision is not final until the allegation is affirmed or denied by the operative findings.

Allegations of Workman.—In analyzing a large number of claims, I find the workman alleges his hernia to be due to one of the three following causes: (1) direct trauma, severe or trivial; (2) excessive strain or overexertion; (3) minimal exertion, or to follow a normal act engaged in while at work. Let us consider the merits of these contentions:

Direct Trauma.—It is the consensus that direct trauma cannot be held responsible for a hernia unless there is demonstrable evidence of a laceration of the overlying structures. This was recently contended by Moorehead[2] after an analysis of 1376 herniotomies. His opinion is concurred in by Early[3] after an operative experience of over 5000 herniotomies during twenty-five years of industrial surgery. A direct traumatic hernia implies an open-wound hernia. Obviously such must be rare.

Excessive Strain or Overexertion.—If hernia results from an overt act or undue strain, it must be because of the indirect action of the increased intra-abdominal pressure. To dilate suddenly the intact rings and the canal, the force of the intra-abdominal pressure would have to be great. And if this action could produce a hernia where none previously existed, why is an inguinal hernia not a common complication of severe abdominal blows or crushing injuries? Why is it that in falls from great heights which result in visceral injury, or in crushing injuries in which fractures of the femur or pelvis occur, acute inguinal hernias are not also found? Dr. Wilmot F. Pierce, of our surgical staff, has treated a large number of patients having injury to the abdominal viscera following direct blows to the abdomen. In these cases he has observed laceration or rupture of the kidney, spleen, liver, and intestines, but no instance of acute hernia. Furthermore, if the inguinal rings and fascial coverings are to be forcibly dilated, pain should be intense and immediate. It has been pointed out that in countries where military service is compulsory, the presence of a hernia may be sufficient to cause rejection. Thus, it is reported, many seek the avoidance of military service by submitting to the produc-

tion of a hernia by a professional dilator, who frequently must attempt manual dilatation three or more times, during which procedure the patients faint from pain. It is also common knowledge that before the peritoneum can be handled at operation an anesthesia must be administered.

To believe that a hernia is suddenly produced, one must expect some or all of the following symptoms to be present: pain, nausea, local tenderness, swelling, and discoloration. To believe that a sac could be forced through narrow openings and through a canal so innervated and so well supplied by blood vessels without producing the aforementioned signs and symptoms is to ignore the anatomy and pathology of an inguinal hernia.

Trivial Exertion or a Normal Movement.—In view of the previous discussion, it is obvious that an acute hernia does not result from slight exertion or a normal movement, and no further consideration of such an alleged cause is necessary.

Medicolegal Status of Lump.—Since we believe, as all thoughtful physicians do, that certain signs, symptoms, and causative factors must be present in order to consider a hernia compensable, why is it that cases lacking these requisites are accepted by the commission? The answer can be given in these few words—the presence of a lump. The present philosophy of those who rule on these claims appears to be this: The man swears he never had a protrusion before, he has one now, so the alleged incident must have caused it. Such reasoning can be appreciated when it is realized that referees are not physicians, although they do have a rough idea of the modus operandi of a hernia. In fact, one wonders if this little knowledge is not a bad thing. In other types of claims less frequently heard by them, they evidence a willingness to learn and consider all the facts. But in the case of a hernia there is a tendency to consider only the fact that the claimant has or does not have an inguinal protrusion. Recently, before a local referee, a physician was asked by an attorney to describe the steps in the production of a hernia. The referee stopped the testimony, saying that such was not necessary since the court understood these things. Obviously, the only value the physician had in that court was to state whether or not the claimant had a hernia.

Mechanism of Hernia.—What can be said regarding the lump which appears and was not present prior to some alleged act? The answer is that a hernia is a slowly progressive (chronic) enlargement of a preformed sac. Over a period of time the sac, filled with intestine, or omentum, or both, keeps exerting gradual pressure upon the rings until they dilate sufficiently to permit passage of this sac to the point where it can be observed; or, that the sac bulges through at a time

the complications of hernia or from an operation for hernia has been claimed, as will be illustrated later.

Operation Refused by Workman.—Hernias are considered remediable by operation. If an operation is tendered by the employer, carrier, or by the award, and the patient refuses to have this operation, then within a reasonable length of time the carrier may be released from responsibility upon proper petition.

Illustrative Cases:

Case I.—Claimant, along with a helper, carried a case of books from one room to an adjoining room on the same floor. Two nights later while taking a bath he noticed a swelling in the right groin. He was ignorant of its nature or cause. At the time of medical examination he stated he had never been nauseated, nor had he been aware of any pain. Following the medical report of an incomplete inguinal hernia of congenital origin, the carrier denied his claim. He went before the Industrial Accident Commission and told the same story he told the physician except he stated that he later recalled that at the time he was carrying the books he noticed a tingling sensation in the groin where the mass later appeared.

None of the accepted criteria for the development or aggravation of a hernia was present in this case. No pain, no nausea or vomiting, and no tenderness were present, and the mass appeared two days after the alleged incident. Many causes could have been present during the non-working hours in the interval between the carrying of these books and the presence of the mass. The physician denied the occupational origin, but the referee awarded the patient compensation, medical care, and costs.

Case II.—A workman was engaged in reconstruction of a bridge following a flood. This bridge crossed a stream, which during most of the year was dry or nearly so. After the water had receded, he was working in deposited silt and sand. While holding a piece of timber he started to sink, became frightened, and attempted to free his right leg. He pulled and struggled until he got a new footing. At this time he developed a pain in his right groin. He reached shore, felt sick, and stretched out on the ground. The sharp pain diminished within from a half to three quarters of an hour, but the discomfort persisted. That evening he reported to a physician, who found a hernia.

Examination revealed the rings to be thickened and well relaxed. A similar condition existed on the opposite side but with no protrusion of the gut. Regardless of the fact that this patient probably had had a potential condition for years, he had a history of aggravation. Therefore, the case was held compensable.

Case III.—A thirty-two-year-old Mexican became acutely ill and was treated for three weeks by a Los Angeles County physician. The diagnosis was acute bronchitis. About the time he was ready to return to work he noticed a lump in his left groin, was examined by his physician, and was told he had a hernia. He entered claim for treatment and compensation. He admitted to the county physician and to a physician at our clinic that it appeared without his knowledge of any strain or overexertion. He had never had any discomfort at the area of the hernia. Subsequently, before the commission he stated that he had lifted a bundle of laundry and noticed a burning sensation at the time in the involved groin. He was awarded an operation and compensation, regardless of the testimony of the only two physicians who examined him.

This case is inserted only to illustrate the point previously made about the futility of medical opinion.

Case IV.—The claimant's husband had suffered from hernia since childhood. While the hernia had troubled him frequently, he had worked as a miner for at least thirty years. On March 12, 1937, in the course of his employment in a mine, he assisted in moving a piece of slate weighing about 700 pounds. About thirty minutes later he complained of being sick and went home. His condition continued to grow worse, and a physician was called, who caused the employee to be taken to a hospital where he was operated on for a strangulated hernia. A secondary operation was performed some days later; pneumonia developed, and the employee died on March 18. His widow, the claimant in the present case, filed a claim for compensation, which was denied by the compensation commissioner. This denial was affirmed by the compensation appeal board, and the claimant appealed to the Supreme Court of Appeals of West Virginia.

The Workmen's Compensation Act of West Virginia provides that an employee may be compensated for hernia: (1) if there was an injury resulting in hernia; (2) if the hernia appeared suddenly; (3) if it was accompanied by pain; (4) if the hernia immediately followed an injury; and (5) if the hernia did not exist prior to the injury for which compensation is claimed. The act, in the opinion of the court, intended to bar compensation for hernia in cases in which the disability existed prior to an injury for which compensation is sought. It refers to "all claims for compensation for hernia," and to obtain compensation therefor certain things must definitely be proved to the satisfaction of the commissioner, the first being "that there was an injury resulting in hernia." It cannot be said, the court concluded, that a hernia resulted from an injury in a case, such as the one at bar, in which the hernia had existed from childhood.

For the reasons stated, the denial of compensation was affirmed.[1]

Obviously this claim was denied because the law in West Virginia rules against any case in which the hernia was known to exist prior to alleged disability, regardless of the possibility of aggravation by some act incurred while employed. In most states, provided there was sufficient evidence of unusual stress, this claim would have been accepted upon the basis of aggravation.

Case V.—The claimant developed a right inguinal hernia as a result of lifting a heavy keg of nails in the course of his employment, and submitted to an operation. For a number of years prior to the operation he had a chronic inflammation of the spine and "hypertrophic spondylitis, that being a growth of bone and cartilage from the spine, causing pain." The long confinement in bed incident to the herniotomy aggravated these conditions and also caused spastic colitis. As a result of these sequelae, he was unable to do any manual work. The industrial accident board awarded him compensation in accordance with a special provision of the Texas Workmen's Compensation Act pertaining to hernia, which authorizes, in the event of a successful operation, compensation for a limited period in addition to expenses for necessary medical and surgical care. The claimant then sued his employer's insurer to set aside the board's award and to recover instead compensation for total permanent disability under the general provisions of the act. From a judgment in favor of the claimant, the insurer appealed to the United States Circuit Court of Appeals.

The Texas Workmen's Compensation Act, said the Circuit Court of Appeals, authorizes compensation for certain specific injuries, including hernia, and also for injuries in general resulting in partial or total incapacity for work. An employee who suffers a specific injury is not confined to the compensation allowed for that specific injury if that injury, or the proper or necessary treatment, causes other injuries which render him unable to work. The court was of the opinion that the aggravation of his preexisting ailments and his consequent total incapacity for work were traceable to his injury received in the course of his employment, and to the treatment called for by that injury. The court held, therefore, that the claimant was entitled not only to compensation for the hernia directly caused by the accident but also for the total and permanent disability resulting from the aggravation of his preexisting ailments, which prior to the operation had had no disabling effects. The judgment of the trial court was therefore affirmed.[5]

The contention that the operation and the period of convalescence aggravated a preexisting spondylitis and caused a spastic colitis appears to be so extremely remote as to question the equity of this decision.

Case VI.—The employee in this case was injured on September 11, when his left leg broke through the wood cover of a manhole. He quit work immediately and had difficulty in getting to his house. He complained of nausea and of pain in the lower left side of his abdomen. That evening a pronounced swelling and tenderness were present in the left inguinal region. Two days later his condition was diagnosed as traumatic hernia. Rest until the swelling and tenderness disappeared was advised. A herniotomy was performed on October 29. Twelve hours later the employee's abdomen became distended, and within a day or two his condition was described as toxic. He died on November 3, the immediate cause of death being "paralytic ileus; *i.e.*, paralysis of the small intestine." The employee's widow was awarded compensation under the Workmen's Compensation Act of Wisconsin, and the employer and insurer appealed to the Supreme Court of Wisconsin after the circuit court had affirmed that award.

The appellants contended that, since the operating physician found conditions at the time of the operation which clearly indicated that the hernia was not of recent origin, the commission was not justified in finding that the employee sustained a hernia as a result of the accident. But, said the Supreme Court, it was immaterial whether the hernia had existed prior to the accident. If it did exist, it was certainly greatly aggravated by the accident and resulted in a compensable injury. While there was no suggestion of carelessness or lack of skill on the part of the operating physician, it was clear, in the opinion of the court, that the commission was warranted in finding that an accident occurred; that it resulted in an injury which reasonably necessitated an operation; and that as a result of the operation infection entered the abdomen which ultimately caused death. Whether the infection entered in spite of the exercise of the highest skill, or as a result of lack of skill or carelessness, was immaterial. If an employer must compensate an employee for injuries caused by the malpractice of a physician who treats the employee for an industrial injury, and such is the law, an employer must likewise compensate an employee, or his dependents in case of his death, for injuries resulting from an operation skillfully performed. The award of compensation was affirmed.[6]

Case VII.—The worker, while engaged in the course of his employment in lifting railroad ties, sustained a strain which resulted in the strangulation of the contents of an existing hernial sac. A gangrenous condition ensued, and an operation performed three days later failed to avert death. An award of compensation in favor of the widow by the Workmen's Compensation Bureau of New Jersey was affirmed by the Court of Common Pleas but was reversed by the Supreme Court. The widow then appealed to the Court of Errors and Appeals of New Jersey.

The Workmen's Compensation Act specifies, in detail, the circumstances under which a hernia must occur in order to constitute it a compensable injury. The Supreme Court held that the death was not compensable because the hernia did not occur under the circumstances set forth in the act. But, said the Court of Errors and Appeals, the reasoning of the Supreme Court did not take into consideration the essential difference between the occurrence of a hernia and the traumatic aggravation of that bodily deformity. Manifestly, the workman in this case could not have met the requirements of the act, for the hernia did not follow but long preceded the accident made the basis of the claim for compensation. His widow did not seek compensation for the hernia but for the death directly traceable to the accidental aggravation of the preexisting diseased condition of the body. Concededly, the workman's death was caused by the strangulation and the supervening gangrenous condition of the intestinal content of the hernial sac. If extraordinary strain resulting from the lifting of the ties was the causative agent of the strangulation, the fatality was the consequence of an accidental injury within the meaning of the New Jersey Workmen's Compensation Act. The Workmen's Compensation Bureau found there was a definite and direct relation between the accidental strain suffered by the workman and his death three days thereafter. An accident which sets in motion the undeveloped and dangerous physical conditions with mortal consequences, is properly classifiable as the proximate cause of the fatality. The court, therefore, reversed the judgment of the Supreme Court and ordered an award for the widow.[7]

The ultimate decision favored the claimant upon the grounds that an aggravation of a preexisting condition occurred. The author questions whether the simple act of lifting a railroad tie constitutes an injury or undue strain when unattended by unusual factors of strain or stress.

Case VIII.—The claimant in this case contended that in the course of his employment he fell and immediately experienced a sharp pain in his left groin. He reported the accident to his foreman and was referred to the first-aid station. What was found there is not shown by the record, but the claimant applied for compensation, alleging that an inguinal hernia had resulted. From an order of the Workmen's Compensation Commission reversing an award that the deputy labor commissioner had entered in favor of the claimant, the claimant appealed to the Supreme Court of Michigan.

At a hearing before the deputy commissioner, a physician who had examined the claimant a few days after the alleged accident and again about two months later testified that he had found on both occasions a left indirect inguinal hernia. On behalf of the employer, however, a

physician who had examined the claimant in the interval between these two examinations testified that he had found an enlarged inguinal ring on each side, the ring on the left side being somewhat more enlarged than that on the right, but that he found no hernia. The condition that he found might be referred to, he said, as a potential hernia, but in his opinion nothing is a hernia unless there is a protrusion of a viscus or organ from its natural cavity. There was no protrusion of a viscus in the present case, although there was palpated "a sharp mass by inserting your fingers in the rings and getting up onto the abdominal cavity." "Some men," said this witness, "call any such sharp mass a hernia, but others refuse to call it a hernia until after it has descended." In this case, in the opinion of this witness, the viscus had not descended through the rings.

Quoting an earlier decision of its own,[8] the court said:

"All the experts seem to agree that the visible evidence of the hernia is the protrusion through the inguinal ring of the peritoneum and its contents; the hernia is the peritoneum going through, accompanied by the intestines or some other substance."

In the present case, said the Supreme Court, there was no protrusion through the internal inguinal ring, and the evidence sustained the finding of the Commission on Labor and Industry holding that the claimant had a potential hernia or a predisposition to hernia. The order of the commission denying compensation was affirmed.[9]

TREATMENT

Herniotomy.—Treatment in cases of hernia in most instances consists of herniotomy by whatever method the surgeon handling the case feels to be best adapted to the individual case and to give the best results in his experience. In our own clinic a modified Bassini technique is used, with very satisfactory results. Most of the herniotomies here are done under spinal anesthesia.

Contraindications.—As in all surgical cases, a careful preoperative check of the patient is made to exclude those cases in which contraindications to operation, *e.g.*, respiratory and genito-urinary tract infections, might be present. If acute infections are present, the herniotomy is delayed until these have cleared. In general, uncomplicated hypertensive vascular or cardiovascular disease is not felt to be a definite hindrance to successful management of these cases, and a large number of elderly persons have been operated on without postoperative complications occurring.

The usual routine calls for from twelve to fourteen days' hospitalization, with an additional four-week convalescent period. At the end of this six-week period the patient is returned to his ordinary work, whether it is heavy or light.

Trusses.—The use of trusses will not offer a solution for cases of hernia ruled as being either due to or aggravated by industrial occupation any more than it will in other non-industrial cases. In inguinal hernias, the use of a well-fitted truss may enable a patient to continue with his work without particular discomfort while his case is being settled or while he arranges his economic situation satisfactorily prior to the six-week period of inactivity. Such a truss does not cure the hernia, but it may during the period prior to operation lessen the possibility of strangulation. If the truss is ill-fitting, however, it may actually encourage strangulation or incarceration. For femoral hernias trusses are rarely satisfactory, and the danger of strangulation frequently seems to be increased by their use. No truss should be worn for treatment of any irreducible hernia.

Injection Method.—The question of the feasibility of injection of hernias[10] frequently arises. In our clinic this method is not used except in rare instances. If the point is simply that the patient refuses operation and instead wants the injection method of treatment, we refer him elsewhere to obtain it, since we do not feel justified in its use under these circumstances. Industrial workers will frequently be attracted by the chance to continue with their work while receiving the injection treatment, since even though compensation may be awarded for the period of disability necessary for the operation, quite a substantial loss is incurred by the worker. If he happens to be in a state where only the medical and hospitalization cost is covered, this is doubly true. Then, too, some people inherently fear surgery and will do anything to avoid being subjected to an operation.

The present status of the injection method of treatment for hernia was reviewed by the Council on Pharmacy and Chemistry of the American Medical Association recently. Their report[11] was based on replies to a questionnaire addressed to a selected list of hospitals throughout the country and was a sequel to a previous report on a similar questionnaire in 1936. The literature since publication of the last report had also been well reviewed. About 65 per cent of the hospitals consulted did not employ the injection method for hernia. The review of the recent literature, however, indicated that the weight of evidence was preponderantly in favor of the acceptance of the injection method of treatment of hernia in *carefully selected cases*. Most observers agree that the injection treatment should not supplant surgery and, except in certain cases, does not offer as favorable a permanent result as the latter. The criteria for safe and successful use of the injection method given by various observers were cited in this report and were as follows:

1. *Selection of Cases.*—Thin young adults with good muscula-

ture. (Obese or aged persons with atonic or atrophic supporting tissues are not suitable.)

2. *Type of Hernia.*—Recent, small, reducible, indirect inguinal hernia with external inguinal ring not over 3 cm. in diameter. (Scrotal, direct inguinal, femoral, umbilical, ventral, and incisional hernias are unsuitable. Sliding hernia associated with undescended testis and incarcerated hernias are contraindications.)

3. *Truss-Fitting.*—Skilled, adequate truss-maintained reduction before, between, and after injections. (Incompletely or inadequately maintained reduction may occur with improper fitting or uncooperative patients.)

4. *Injection Technique.*—Knowledge of inguinal anatomy and skill during injection are fundamental. (Surgical asepsis is, of course, imperative.)

5. *Choice of Solution.*—Sterile, non-toxic, fibrous-tissue-stimulating, non-irritating solution is ideal. The fatty acid (soap) solutions approach nearest to these criteria. (Powerful sclerosing solutions are to be avoided and are believed responsible for many complications.)

6. *Follow-Up.*—Repeated examination for periods of from six months to three years is necessary to determine length of treatment and period of truss-wearing required for cure. (Inadequate treatment and truss support blamed for failures.)

Not for General Use.—The council then voted to adopt the report that the injection method of treating hernia may not be recognized for general use and should be employed only by those with special experience and with full cognizance of the dangers involved in the use of such solutions. It then stated that it concurred in the opinion that the method involves less danger of serious complications than surgery when employed only in selected cases of hernia by those skilled in the injection of suitable standardized solutions of known composition and action.

None of the solutions are council-accepted. Those reported as being used include a mixture of oil of thuja, phenol, and alcohol; sodium linsoleate; sodium psylliate; tannic acid; and proprietary preparations, such as pina-mestre solution, galtanol, and proliferol.

Complications.—Complications reported in the literature include mild infections, the development of fibrous masses of cutaneous tissue, failure of the method to correct the hernia, painful scarring, occasional cases of swelling of the testis, a few instances of subsequent impotence, gangrene, death following perforation of the bowel from accidental intraperitoneal introduction of the needle, intestinal obstruction due to adhesions from inadvertent intraperitoneal injection, or strangulation from failure to maintain reduction, perirectal abscess,

fecal fistula, and thrombosis of deep epigastric, iliac, or femoral vessels. Most observers indicate that all serious complications can be avoided by skilful technique.

Evaluation of Results.—That a prolonged period of follow-up, rather than a six-month-to-year period, is needed for actual evaluation of the "injection cures" is evidenced by the report of Dobson.[12] In this he reported the results on 101 hernias in 70 males and 4 females operated on between September, 1935, and June, 1938, including 68 indirect inguinal, 21 direct inguinal, 10 postoperative inguinal, one postoperative femoral, and one umbilical hernias. Eighty of these were followed up for periods of from six months to two and a half years. Six months after beginning of the injection treatment, the results had been most promising, and it seemed that all types and sizes of hernia were being cured. But then there began disappearance of scar tissue and recurrence of the hernias. There was a recurrence percentage of 37.73 per cent in indirect inguinal hernias; 68.42 per cent in direct inguinal hernias; and 100 per cent in all postoperative hernias. Harris and White[13] reported on the follow-up of 573 cases, only 41 per cent (236) of which could be utilized for this check. In these 236, there were 57 per cent cures, 28 per cent possible cures, and 15 per cent complete failures after a six-month-to-three-year period. Many more long-term follow-up studies are needed to evaluate this method.

REFERENCES

1. Campbell, D. A.: *Workmen's Compensation*, Parker, Stone, and Baird Co., Los Angeles, 1935, Vol. 1, p. 306.
2. Moorehead, J. J.: "The Relation of Trauma to Inguinal Hernia," *Am. J. Surg.*, *27:* (Feb.), 1940.
3. Early, C. E.: Chief of Staff, Golden State Hospital, Los Angeles, personal communication.
4. Jordan *vs.* State Compensation Commissioner, W. Va. 197 S. E. 20.
5. General Accident and Liability Insurance Company *vs.* Daffern, Texas 81 F. (2d) 179.
6. Stiger *vs.* Industrial Commission, Wis. 265 N. W. 678.
7. Furferi *vs.* Pennsylvania Railroad, N. J. 189 A. 126.
8. Robbins *vs.* Original Gas Engine Company, 191 Mich. 122, 157 N. W. 437, 438.
9. Cessante *vs.* Ford Motor Company, Mich. 278 N. W. 671.
10. Riddle, P.: *Injection Treatment*, W. B. Saunders Co., Philadelphia, 1940.
11. "Present Status of Injection Treatment of Hernia. Report of the Council on Pharmacy and Chemistry," *J.A.M.A.*, *115:* 553 (August 17), 1940.
12. Dobson, L.: "The Late Results of the Injection Treatment of Hernia," *Surgery*, *7:* 836 (June), 1940.
13. Harris, F. I., and White, A. S.: "The Evaluation of the Injection Treatment of Hernia," *J.A.M.A.*, *111:* 2009–13 (Nov. 26), 1938.

PART VI

CHAPTER XXXII

THE DERMATOSES

ANY clinic devoted to industrial medicine is certain to have a fairly large admission of patients with skin lesions. Since there is no full-time dermatologist on our staff, it falls to the author either to make the diagnosis, or as is frequently the case to refer the patient to an outside dermatologist. It is with a full appreciation of the difficulties commonly encountered that the author sought to make the following discussion a concise but accurate description of only the more frequent occupational dermatoses, since obviously this subject in its entirety is too vast for discussion in other than a separate textbook. The material has been provided largely through the efforts of the collaborator.*

Incidence and Statistics.—Schwartz[1] estimates that the proportion of dermatoses to all other occupational diseases is 69 per cent; the average loss of time for compensated cases is ten weeks; the average compensation paid is $100; the average cost for medical care is about $90; and the annual loss for the occupational dermatoses in the United States is approximately $4,000,000. The U. S. Public Health Service made studies of 100,000 workers in various occupations and found that 1 per cent were affected by skin lesions resulting from contact. This figure excluded burns, splashes by alkalis or acids, and pyogenic invasion of occupational skin wounds. These figures should give a proper conception of the importance of this phase of industrial medicine.

Occupational Hazards.—It is impossible to list the occupational hazards, for it may be truthfully said that the majority of all trades present such a hazard to certain persons. It is known that any substance encountered in any trade or occupation may produce a dermatitis in one who is susceptible. The handling of oils, chemicals, dyes, paints, solvents, metals, fabrics, rubber, and heavy dusts, cement in particular, leads the list.

Epitheliomas and carcinomas resulting from occupational origin will be mentioned elsewhere in this book.

* Written in collaboration with Nelson Paul Anderson, M.D.

Definition:

An industrial dermatitis is any inflammatory disease of the skin for which industrial exposure can be shown to be a major causal, contributory, or eliciting factor. Industrial dermatitis is a subcategory of occupational dermatitis, for occupational dermatitis includes not only industrial dermatitis but also all other dermatitides attributable to any occupational pursuit.[2]

General Considerations of Etiology.—That allergy plays a rôle in the production of many types of occupational dermatoses seems to be an established fact. In spite of the tremendous amount of investigation on this subject, little is known regarding the true mechanism of the production of an allergic state. For practical purposes the physician handling occupational dermatoses should realize that allergy and hypersensitivity do exist. He should, however, fully realize that there are many other factors which may play an important part in the causation of dermatitis in industry, and that these factors may or may not deal directly or indirectly with allergy.

Many of the skin conditions met with are due to primary cutaneous irritants. These substances, which are generally strong acids, alkalis, or corrosive salts, are sufficiently irritating to produce a dermatitis in a large percentage of persons with normal skin.

The presence of focal infections in the teeth, tonsils, and sinuses undoubtedly contributes to the production of a dermatitis in certain cases. Superficial fungous infections of the feet and groin give rise to many perplexing problems in industrial dermatitis. The type of skin, perspiration, diet, and personal cleanliness are all factors which must be taken into consideration.

Finally, as pointed out by Sulzberger,[2] there are many eruptions which are due to an interaction of many elements and to a complex effect attributable to the cooperation of many factors. He terms this action "synergic" or "synergistic."

DIAGNOSIS

Certain lesions resulting from occupational exposure constantly present the same clinical manifestations. "Chrome holes" on the hand, or of the nasal septum, have constant characteristics which should entail no diagnostic difficulty when appearing in a man working with these fumes or with the chromates. The same may be said of the furunculosis occurring in oil or cement workers, due to mechanical blockage of the pores of the skin, or the acne from the action of tar. For these and many others, the industrial cause is usually apparent. The industrial physician, practicing in an area given to the manu-

facturing or processing of certain substances, rapidly learns the characteristics to be expected in lesions apt to result from such exposure.

On the other hand, there are a vast number of what appear to be nondescript eczematous lesions of occupational origin which are most baffling. With these it is not such a simple matter to find the causative agent. Furthermore, these lesions do not take on a characteristic appearance such as the chrome ulcer, but instead present an eczematous appearance similar to many other dermatoses. A dermatitis from a plant, a dye, a solution, a cosmetic, or a fungus may be clinically indistinguishable by the physician. It is this type of case which must frequently be referred to the dermatologist.

History.—A careful and detailed history should be taken before proceeding with the examination of the patient. This history must include an inquiry into all past illnesses of the patient and his family, with particular reference to any previous skin trouble, asthma, hay fever, urticaria, or other allergic disorders, and of any drugs recently taken. Detailed chronological statements should be obtained, concerning both the particular type of work done by the patient and the onset of the present eruption. Inquiry can be made about the working conditions, including light, heat, ventilation, and factory hygiene. One should try to obtain a complete and vivid picture of the patient's daily routine while at work. While doing this, a list can be made of substances, chemicals, or other materials which the patient contacts while at work. The history is completed with information regarding the patient's outside activities, conditions of home life, and hobbies.

Removal of Clothing.—There is one admonition which cannot be too strongly stressed. Any patient presenting a skin lesion should be examined with all clothing removed. A Colles fracture may be examined and diagnosed with only the forearm bared, but a skin lesion of the wrist requires removal of the entire clothing for adequate diagnosis.

Criteria for Diagnosis.—In order to establish proof of the industrial nature of a given dermatitis, Sulzberger and Finnerud[2] listed certain criteria which should be of aid. These are quoted as follows, except that under their "Criteria of the Second Order" I have considerably altered their seventh point.

Criteria of the First Order

1. *Inception.*—The dermatitis appears at any time during a period of industrial exposure or even after a lapse of a reasonable incubation period, following the cessation of the industrial exposure (usually a maximum of from two to three weeks).

2. *Amelioration.*—The dermatitis regularly disappears or is re-

peatedly improved within a reasonable period (days, weeks, or even months) after cessation of the causal industrial exposure. (While this is usually the case, retention of causal agents, complications, or ensuing polyvalent sensitization may prolong the course even for several years after the last industrial exposure.)

3. *Recurrences and Exacerbations.*—The dermatitis shows a tendency repeatedly to recur or to exacerbate when the worker returns to the identical industrial exposure after a certain period of absence (provided that there has been no change in working conditions or in the patient's manner of working, or in his susceptibility).

Criteria of the Second Order (Adjuvant Criteria)

1. The dermatitis appears first in, and is usually confined to, the areas of maximum exposure (in a small percentage of cases it spreads to, or may begin in, apparently unexposed areas or even becomes generalized).

2. The character and localization of the dermatitis correspond to the character and localization of dermatitis known in other cases to have been caused by exposure to the same or similar industrial hazards. (While many different substances and procedures can produce similar or identical eruptions, there are certain classes of substances and of procedures which regularly produce fairly characteristic lesions.)

3. The application of the presumptive causal agents to an unaffected site close to the site of the dermatitis produces a reaction, provided this application is made either during the active phase or after the proper interval following the cessation of the dermatitis.

4. The cutaneous test produces reactions of the same fundamental nature as the dermatosis under investigation.

5. Other workers similarly occupied are or have been similarly affected.

6. The dermatitis appears soon (days or weeks) after the patient begins work involving new potential hazards.

7. The dermatitis is proved to be of possible occupational nature; that is, it is shown to be of the type which may result from the industrial exposure sustained.

Skin Disorders Unrelated to Occupation.—The physician should recognize that such skin disorders as psoriasis, scabies, pityriasis rosea, seborrheic dermatitis, impetigo, erythema multiforme, and lichen planus are practically never related to occupation. Nevertheless, patients with these diseases frequently present themselves, believing the skin disorder to be caused by their work. It occasionally happens that a preexisting skin condition, particularly seborrheic dermatitis, may

be a predisposing factor in the production of an industrial dermatitis.

Patch-Test Technique.—The patch test is performed as follows: The substance to be tested is placed upon a small square of gauze, several layers in thickness. This gauze square is most convenient when about three-fourths the size of a postage stamp. In general, powders or dry substances must be moistened slightly with water. This small patch is then placed upon the skin in such a manner that the substance to be tested comes in direct contact with the skin. Any area of the skin may be selected as the test site. The areas preferred are the

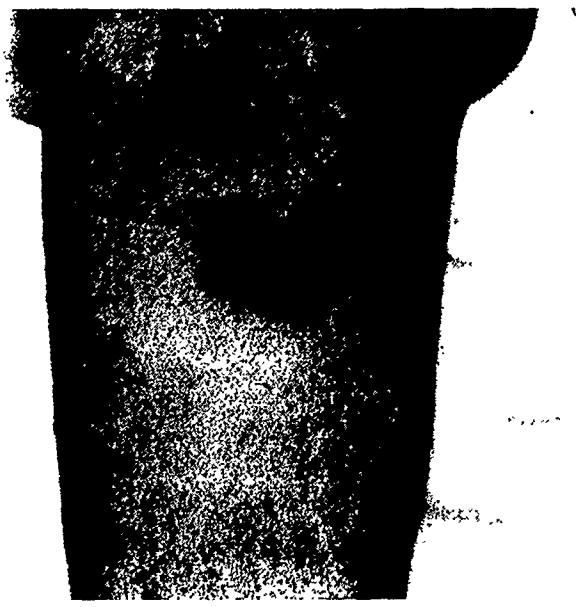


Fig. 115.—Positive reaction to patch test from 1 per cent formaldehyde.

back, inner aspects of the upper arms, and the anterior surfaces of the thighs. In industrial practice, particularly if one has any suspicions of the integrity of the patient, the back is the best location for testing. The patch is then covered by a square of impervious material, such as wax paper or cellophane. This covering should be about four times the size of the test patch. These are covered by wide strips of adhesive and left undisturbed for forty-eight hours. The patient should be instructed to remove the patch at once if burning, stinging, or marked discomfort occurs. In cases of known sensitivity to adhesive tape the patches may be held in place by flexible collodion, nail polish, or rubber cement.

Delayed Reaction.—After removing the patches, the test areas may be encircled with 2 per cent mercurochrome or 2 per cent gentian violet, in order to facilitate the correct reading of delayed reactions. The reading of the test is performed twelve to twenty minutes after removal of the patches and should be checked by subsequent readings at forty-eight- and ninety-six-hour intervals.

Characteristics of Positive Reaction.—A positive reaction is characterized by a sharply defined square of redness, corresponding in size and shape to the central test area (Fig. 115). Papules and vesicles are also usually present, corresponding to the appearance of the original eruption. Only rarely is a positive reaction manifested by redness and inflammatory edema. Such a positive reaction can best be determined by feeling or stroking the test area with the finger.

Evaluation of Negative Reactions.—Having performed patch tests, one would think it a simple problem to evaluate the results of such tests. In the ordinary case this is true. There are, however, numerous pitfalls in the interpretation of such tests. Negative reaction to patch tests with suspected irritants can present several sources of error:

1. The dermatitis in question may not be the result of any substances contacted at work.

2. Local skin sensitivity may be present. This phenomenon is rare, but it occasionally happens that patch tests on sound skin give negative results. In such cases the test may be repeated, using a site adjacent to that of the original eruption. It may be necessary to wait until the original condition has disappeared and then repeat the test on the healed area.[3]

3. The actual causative substance producing the eruption may not have been tested; or, if tested, it may have been used in too weak a concentration, or incorporated in an unsuitable vehicle.

4. Theoretically possible, but actually quite rare, is the occurrence of a refractory period of skin reactivity. During such a time the skin may fail to react to a definitely causative substance. If the patch test is performed during such a refractory period, one can easily see how erroneous conclusions can be drawn.

5. The actual conditions of industrial exposure are never duplicated by patch tests. It is well known that the noxious effects of many substances are magnified by numerous external factors. These include perspiration, heat, cold, sunlight, repeated cleansing with soap and water, repeated exposures and prolonged immersion in liquids leading to maceration of tissues. None of these contributory factors can be adequately reproduced by the patch test.

6. Occasionally, patch tests done with all suspected irritants are negative in spite of fairly definite clinical evidence that some industrial contact is causative. In such cases there seems to be definite evi-

dence to show that two or more substances act in combination or synergistically to produce the occupational dermatitis.[4] This is particularly true in the so-called "baker's dermatitis." In other instances, in addition to exposure to some irritant, an additional physical agent, such as sunlight or heat, is necessary to produce a positive reaction.

Evaluation of Positive Reactions.—Positive reactions to patch tests with suscepted irritants can present several sources of error:

1. Either substance tested may be a primary irritant which would produce irritation of any normal skin; or, if not a primary irritant, it may have been used in too strong a concentration in performing the test. Under no condition should the concentration employed in testing exceed that which occurs in exposure at work. The use of solutions in too strong concentration may give rise to localized gangrene and widespread irritation, and may even be responsible for the generalization of the heretofore localized process.[5] In fact, there are medicolegal possibilities in using the patch test. Certain chemicals must be incorporated into vehicles before testing. The proper vehicle and the concentration to employ have been made the subject of considerable study.[6]

2. A positive patch test with a substance contacted at work does not necessarily prove that the eruption was caused by this substance. Such a positive test may indicate only a latent sensitivity, and the real cause may be some substance not tested but contacted by the patient outside of his work.

3. The interpretation of the positive patch test is always open to the criticism that such a test can never actually duplicate the conditions of industrial exposure.

4. Marked difficulties of interpretation arise in cases where several different substances produce positive reactions to patch tests. This occurrence of polyvalent sensitivity, however, may furnish a clue to a non-industrial irritant and at the same time aid the physician in avoiding contact dermatitis as a result of his own therapy. Of course, a patient found positive to mercury among other things would not be treated by mercurial ointments or antiseptics.

5. For a substance to be incriminated as the cause of an eruption there must be a definite history of contact with the suspected material.[7]

6. Although stated elsewhere, it may be said that with one or two exceptions (cement), the elimination of contact with the suspected irritant should produce either a cure or marked amelioration of the skin condition.

In the following paragraphs consideration is given to a few representative and fairly common types of occupational dermatitis.

Epidermophytosis.—The relationship of superficial fungous infections or eczematoid ringworm of the extremities to industrial dermatitis is one that gives rise to more discussion and dissension than occurs perhaps in any other skin disorder. As Blaisdell[8] states, "Broadly speaking, it would apparently be easy enough to discriminate between the case entitled to compensation and the one that is not, but considering the cases individually it is oftentimes difficult to give a judgment that is fair to both the worker and the insurance company."

There are definite instances where epidermophytosis might be directly related to the occupation, *e.g.*, an attendant in Turkish baths. There are also cases where a true contact dermatitis of industrial nature may be followed by secondary infection with ringworm organisms. Again, a contact dermatitis may be superimposed on a superficial fungous infection. This not infrequently happens in cases of cement dermatitis.

In every case of suspected industrial dermatitis of the hands, the experienced clinician will examine the feet for evidence of any superficial fungous infection. Such examination should include microscopic study of scales for mycelia. There is considerable clinical evidence to indicate that some of the eruptions on the hands in such cases are allergic manifestations of the fungous infection on the feet,[9, 10] *e.g.*, epidermophytosis, and hence not of an industrial nature. However, in the presence of a positive patch test to some suspected and contacted irritant, such an infection of the feet may be looked upon as producing a non-specific irritability of the hands. In other words, such a fungous infection of the feet lowers the threshold of sensitivity of the hands and hence acts as a predisposing etiological agent.

The trichophyton intradermal test is believed to be of value if properly used. Lewis[11] is of the opinion that while a positive intradermal trichophyton reaction is of little or no significance, a negative trichophyton reaction at least excludes the possibility of a given skin disorder's being an epidermophytid.

Infectious Eczematoid Dermatitis.—Traumatic wounds of all types are extremely liable to the development of infectious eczematoid dermatitis (Fig. 116), often erroneously considered to be a ringworm[12] infection because of its tendency to annular configuration. The usual picture is one of a person who sustains an injury to an extremity in the form of a scratch, abrasion, laceration, or crushing injury. In the latter event an amputation of one or several digits follows. During the period of healing an acute eczematous dermatitis develops about the wound margins. This is characterized by an annular or circinate redness with the presence of small pin-head-sized papulovesicles, produc-

ing a weeping surface. The condition may spread only in the neighborhood of the wound, but often patches are found far removed from the original site. Severe itching occurs, and the scratching which this

Fig. 116.—Infectious eczematoid dermatitis of the hand simulating a superficial fungous infection.

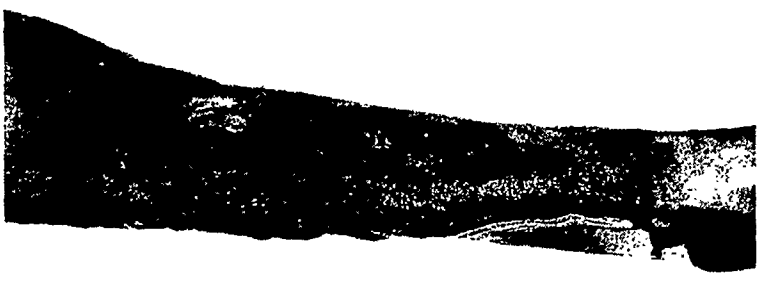


Fig. 117.—Diffuse infectious eczematoid dermatitis arising from multiple draining osteomyelitic foci.

causes may give rise to new lesions. It may also give rise to secondary infection in the form of lymphangitis, cellulitis, and regional adenitis.

A similar eruption may develop about draining sinuses subsequent to osteomyelitis and compound fractures (Fig. 117). Occasionally such an eruption is a true ringworm infection. In other instances the der-

matitis may be due to sensitivity to mercurial or other antiseptics which have been applied.

Turpentine Dermatitis.—Turpentine is a frequent cause of contact dermatitis in industry. According to Schwartz, it produces about 1 per cent of all cases of occupational dermatitis. Turpentine dermatitis may occur in those engaged in its manufacture; however, it is most frequently seen in painters (Fig. 118). Since it usually develops in people who have followed the painting trade for many years, it may present a serious problem both to the patient and to the insurance carrier. To the painter, turpentine dermatitis represents a serious obstacle to the pursuance of his trade. To the insurance carrier it represents a compensable occupational dermatitis, in which the initial temporary disability is often a matter of several weeks and recurrence is practically universal.

Fig. 118.—Contact dermatitis of the eyelids due to varnish-remover. Such involvement of the eyelids may occur through fumes, but is usually from actual contact through the medium of the hands.

There is no doubt that different kinds of turpentine vary a great deal in their irritating and sensitizing properties. Destructively distilled wood turpentine appears to be the least irritating.

Baker's Dermatitis.—Dermatitis in bakers is not uncommon and may result from contact with flour, sugar, cake dough,[13] bread dough,[13] cinnamon,[14] vanilla, various spices, and so-called "flour improvers," chiefly ammonium persulfate. However, only about two thirds of the patients present positive reaction to ordinary patch tests. In such cases one must perform additional patch tests, using different

combinations of the ingredients encountered at work. Even then there remains a considerable percentage of cases of unexplained origin.

The eruption involves the hands (Fig. 119) and at times the forearms and occasionally the face. It is usually of a dry, scaly, eczema-

Fig. 119.—Dry, scaly, hyperkeratotic dermatitis occurring in a baker who was proved sensitive to both cinnamon and nickel.

Fig. 120.—Occupational dermatitis of the hand due to cinnamon, in a pastry worker.

tous[15] nature. In general, the eruption tends to be diffuse, and secondary ridging of the finger nails occurs in long-standing cases. Involvement of the nails alone may occur.[16] There are not other special clinical characteristics (Fig. 120).

Cosmetician's and Barber's Dermatitis.—Occupational dermatitis is common in those engaged in beauty-shop work. Barbers are prone to develop a trade dermatitis from quinine, which occurs in many scalp tonics, the perfumes used in many hair lotions and from prolonged and repeated immersion of the hands in soap and water.

Cosmeticians handle many chemicals which may produce a contact dermatitis. These include *p*-phenylenediamine (Fig. 121), which is the active ingredient of most all hair dyes, nail polish, nail-polish removers, finger-wave solutions, perfumed creams, rinses of all types,

Fig. 121.—Occupational dermatitis in a hairdresser. Contact dermatitis of hand due to *p*-phenylenediamine; positive patch tests to same on thigh.

shampoos containing various oils, and above all formaldehyde, which is used frequently for the dry sterilization of combs and brushes. Regardless of other factors, it is important to find out the exact cause of the eruption in any given case. Once this has been determined, it is often possible for the beauty operator to continue in the same line of work, provided there is no exposure to or contact with the offending substance. The patch test with formaldehyde should be done with a 1 per cent solution.

Cement Dermatitis.—Dermatitis from cement is exceedingly common in masons, bricklayers, plasterers, and tile-setters (Fig. 122). It

differs from the ordinary contact dermatitis in that the eruption not only involves the exposed areas of the hands and forearms, but also occurs about the ankles and other covered parts of the body. Its persistence for weeks and even months after exposure has ceased and the tendency to secondary pyogenic infection are noteworthy. It is one of the most disabling of all the industrial dermatoses,[17] and from many standpoints deserves more consideration from the dermatologist,[18] cement-manufacturers, and insurance carriers than it has received. It should be noted that these patients may be sensitive to only one type of cement[19] and yet be able to handle other cements.

Fig. 122.—Contact dermatitis of the hand due to cement.

Dermatitis Artefacta.—One of the most difficult dermatological problems confronting the physician is that of self-produced eruptions. Usually occurring in emotionally unstable but nevertheless intelligent persons, such artificially produced lesions have the following characteristics[20]:

1. Conform to no other known type of skin condition
2. Occur in circumscribed areas, the surrounding skin being normal
3. Can usually be reached by the hands, and are often unilateral.

being on the left side of the body in right-handed persons, and vice versa

4. Other features of hysteria, *e.g.*, absent pharyngeal and conjunctival reflexes, may be found

To the above may be added the observations that practically every patient has suffered some sort of trauma before the appearance of the first feigned lesion; and also that the first manifestation is often in the immediate neighborhood of the preceding injury.

Such feigned lesions are most frequently produced by acids, alkalis, caustics, or vesicants. They are usually ulcerative in character

Fig. 123.—Dermatitis artefacta. Patient's first lesion was due to sulfuric acid from a storage battery. He subsequently intentionally produced the lesions seen in this picture in order to prolong compensation and avoid alimony payments.

but may be erythematous, papulovesicular, or bullous. Gangrenous[21] lesions on apparently intact skin should immediately arouse the suspicions of the physician.

Frequently in industrial practice, dermatitis artefacta is produced from self-evident motives, *e.g.*, compensation. At times other considerations come into play, including revenge or domestic difficulties (Fig. 123).

From a therapeutic standpoint, nothing is ordinarily gained by directly accusing the suspected person. Time and money will be saved

by hospitalization, the use of occlusive dressings sealed with collodion or adhesive tape. If the latter is used, it should be marked in such a way that tampering with the dressing may be readily detected. Continuous strict observation must be carried out day and night, but in such a manner that the malingerer does not realize that he is under suspicion.

Onychia.—Involvement of the fingernails and paronychial tissues occurs more frequently in industry than generally realized.[22] Deformity, ridging, and transverse striations of the nails occur especially in soda-fountain workers, cannery workers, fruit-juice-stand employees, and others who have their hands immersed in liquids over long periods of time (Fig. 124).

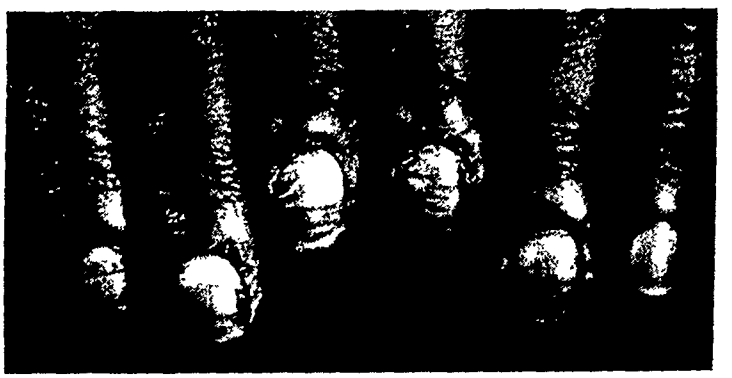


Fig. 124.—Paronychia with multiple involvement of the periungual tissues and secondary nutritional deformities of the nails. A common condition in soda-fountain workers.

Syphilis.—With the exception of doctors, nurses, and laboratory workers, syphilis does not ordinarily enter into the question of compensable dermatoses. Situations do occur in which trauma acts as a contributory factor. The most important of these conditions is interstitial keratitis, which may first appear in a congenital syphilitic following an eye injury. The *criteria for the acceptance of such a diagnosis* are well defined and have been aptly stated by Barkan[23]:

1. Trauma must be positively ascertained and more than such as the person is exposed to in the course of his employment
2. Trauma must be of the cornea itself and cause a definite irritation
3. An expert must determine that the interstitial keratitis is not present at the time of injury, and that the consequence of the injury is an interstitial keratitis

Unilateral tertiary squamous syphilides of the palm occasionally appear to be induced by frequent and repeated minor traumatisms. Such a syphilide must appear on the right hand in a right-handed person, and vice versa. Further, the location of the syphilide must correspond to the area traumatized.

Finally, annular and circinate nodular and nodulo-ulcerative syphilitic gummata may appear in any area after trauma, or after the removal of a foreign body. In general, compensability in all cases complicated by syphilis lasts only until the disappearance of the external manifestations of the disease.

MEDICOLEGAL ASPECTS

While the industrial dematoses are probably the most frequent of all industrial claims for compensation, the hearings of these cases before industrial accident commissions are infrequent. The insurer is usually willing to accept the diagnosis from a trained dermatologist. Less than 2 per cent of the cases handled at this clinic have ever been contested. Insurance companies also appear to be more willing to accept the skin lesion, since there is not the fear that permanent disability will face them if they accept the case as compensable.

Temporary Disability.—This ceases with the disappearance of the lesion, when the lesion does not interfere with ability to work, or when employment can be offered which is considered not apt to aggravate or produce further lesions. In some instances, loss of a wage difference between former occupation and the offered one has to be compensated.

Permanent Disability.—This is rare. Occasionally, limitation of motion follows contraction scars or keloids from skin lesions. If so, they should receive a rating based upon the percentage of loss of function of the part. *Disfigurement* of the body as a result of alteration of the former appearance of the skin is ordinarily considered as not compensable.

TREATMENT

Prophylaxis.—Preventive measures are highly important. *Preemployment examinations* should uncover the history of repeated skin lesions or the tendency to react to even mild irritants. In occupations which involve a special liability to dermatitis the employer would do well to insist on a routine examination of the skin of prospective employees.[24] Such an examination should take into consideration the importance of any existing skin condition. Special emphasis should be placed on seborrhea, ichthyosis, hyperhidrosis, the general type of complexion (blonds and red-haired persons are very susceptible to cutaneous irritants), and the presence of epidermophytosis of the feet.

Protective Procedures.—Each new workman should be given careful instruction as to maintaining good hygiene, avoidance of undue exposure, and the wearing of certain protective devices. Wherever

Fig. 125.—Contact dermatitis of forearms and arms in a janitor due to "sweeping" compound.

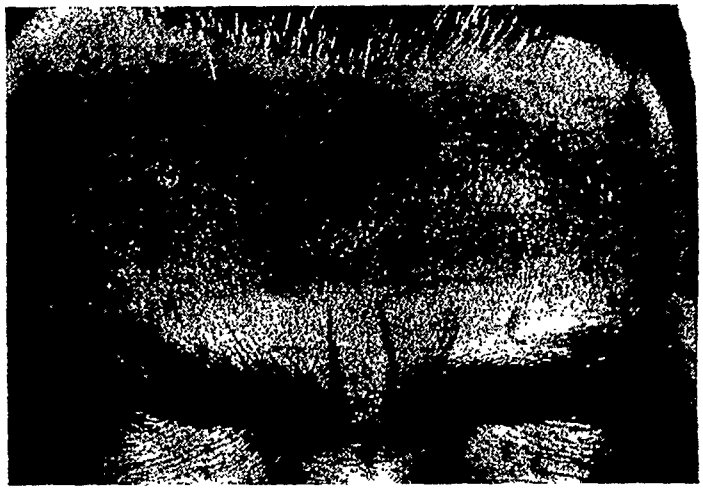


Fig. 126.—Occupational dermatitis due to leather. This hat-band dermatitis caused by safety helmet in an oil-field worker.

dust, grease, oil, grime, and the like, are prevalent in the work (Figs. 125, 126), the men should have two sets of lockers—one for the clean street clothes, and the other for the working apparel. At the end of the day's work, he should deposit his working clothes in one locker.

shower well, and then proceed to the second locker for his clean clothes. Men working where the substance is such that it may easily penetrate the clothing, such as many dusts, cement, or oils, should wear thick, closely woven clothes or rubberized material. Properly fitting gloves are often neglected where they would be advantageous. A film of oil or vaseline may be a protection to unprotected parts. Their value is questioned by most dermatologists, however. It is astonishing how many small plants fail to supply the above-mentioned protective devices. Immersion of the hands in irritating chemicals is often done without gloves, when these could be easily employed. It can be laid down as a general rule that in all industries involving exposure to chemicals, dyes, drugs, coloring materials, or fluids, it is always advisable to rinse the hands or exposed parts with a weak solution of some reducing agent. If the offending agent is alkaline, then a weak neutralizing solution of vinegar, acetic acid, lime water, or weak sulfuric acid may be employed. For general purposes, especially where acid is apt to affect the skin, a weak alkaline reducer may be used as, for instance, a weak solution of sulfate of soda. These reducing agents should be supplied and should form a routine part of the workman's hygiene. After the use of a reducing agent, the parts may be washed in distilled water.

Proper Use of Cleansing Agents.—It is no exaggeration to state that a very considerable percentage of cases of so-called "industrial dermatitis" is due to the improper use of cleansing agents rather than to the actual contact of some specific industrial irritant. This entire field of industrial dermatitis has been extensively studied by Klauder and his collaborators. The physician encountering negative reactions to patch tests in apparent and obvious cases of occupational dermatitis should take cognizance of this broad field. In probably 10 to 15 per cent of cases of proved contact etiology, soap, water, and other cleansing agents act as a definite contributory factor. The use of scouring or grit soaps, soap powders, washing powders, and laundry soaps is often followed by the onset of a dermatitis.

Substitute for Abrasive Soaps.—The proper cleansing agent for any particular plant or branch of industry can only be determined after proper study. As an efficient substitute for the popular mechanic abrasive soaps, Klauder recommends the following:

Formula 12:

	Cc.
Sulfonated neat's foot oil	45.0
Light liquid petrolatum	45.0
Gelatin, 25 per cent aqueous solution	10.0

This mixture is added to granulated corn meal in the proportion of $1\frac{1}{2}$ parts by weight of corn meal, and one part by weight of the above oil mixture. To prevent the growth of mold, a 0.5 per cent solution of chlorobutanol is added.

Protection against Chrome Compounds.—In industries where chromic acid and chrome compounds are encountered (Fig. 127), that is, in dyeing, tanning, electroplating, and blueprinting,[25] a 10 to 30 per cent solution of sodium bisulfite ($NaHSO_3$) may be used as a reducing agent. It can be applied before exposure and allowed to dry on the skin and is also used as a wash after exposure.

Fig. 127.—Contact dermatitis in a printer. Patient was sensitive to the bichromates used in this industry.

Protective creams, liquids, ointments, and *pastes* may also be employed, their formulae and composition depending upon the requirements in each case. Klauder gives a number of such formulae applicable to protection against prolonged contact with soapy water, or against dust-borne irritants. The industrial physician confronted with such a problem would do well to consult this article.[26]

Changing Working Conditions.—Confronted with a severe and undoubted case of industrial dermatitis (Fig. 128), there are two possible courses open. The first is to transfer the affected patient to another job, allowing most of the etiological factors to remain for the next susceptible person. The second and more important procedure, par-

ticularly if numerous cases occur from the same source, is to change either the process or the working conditions. This involves an exact knowledge of all the processes involved. Such steps at times are made

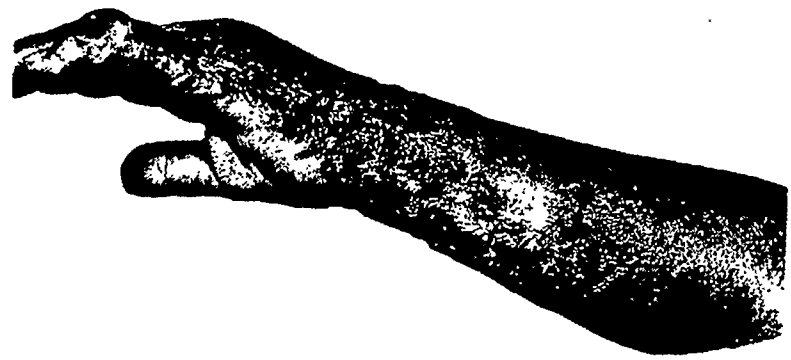


Fig. 128.—Severe bullous contact dermatitis of the forearms in a construction worker, due to poison oak.

Fig. 129.—Contact dermatitis due to rubber gloves. At times the very measures used to protect an employee may be the cause of a dermatitis.

only when either governmental authorities or the insurance carrier exerts pressure on the insured.

In most instances when a person severely afflicted with an occupational dermatitis changes his work, the skin condition clears and remains well. However, the skin condition may persist (Fig. 129). Even

if one continues at the same line of work, a certain percentage will eventually clear, although at least an equal number will continue to have skin trouble.[27]

Local Measures.—In the actual therapy of industrial dermatitis, certain local measures should be mentioned. If the dermatitis is acute and accompanied by vesicles and bullae, together with weeping and exudation, cold wet compresses are advised.

Liquor aluminum acetate (liquor burrowi, N.F.) ..... 480 Cc.
Sig: Dilute twelve to sixteen times with cold tap water and use as a continuous moist compress. Such compresses should be changed or remoistened every two to three hours.

If the process is acute and oozing but accompanied by any evidence of pus formation, the use of wet dressings of from 0.25 to 0.5 per cent of an aqueous solution of silver nitrate is indicated. Wet dressings of Alibour water (see "Infectious Eczematoid Dermatitis") are also of value.

As the weeping and exudation subside, which they generally will within from forty-eight to ninety-six hours after the proper use of continuous wet compresses, soothing ointments may be used, such as the following:

Liquor aluminum acetate 15.0
Lanolin, anhydrous 25.0
Lassar's paste, plain 20.0
Sig: Apply locally two or three times daily. If some evidence of infection still persists, either ammoniated mercury (2 to 3 per cent), or sulfur precipitate (2 to 3 per cent), may be added to the above ointment.

When a stage of chronicity has been reached and the skin lesions are dry, scaly, and thickened, the use of some tar-containing preparation is indicated. In milder cases this may consist of ichthyol, 2 per cent in Lassar's paste. Later, a 5 per cent oil of cade ointment may be applied. The most useful of all the tars, however, is white crude coal-tar paste (see "Infectious Eczematoid Dermatitis").

Constitutional Therapy.—This is rarely of aid in occupational dermatitis. Dainow[28] has reported desensitization from the administration of vitamins A and D. In very stubborn cases, removal of foci of infection may be necessary.

Infectious Eczematoid Dermatitis.—Notoriously resistant to therapy, this moist, vesicular, eczematous condition may often be ameliorated or cured by the application of a 2 per cent aqueous solution of gentian violet twice daily. As long as the oozing persists, continuous

wet compresses of from 0.25 to 0.5 per cent aqueous solution of silver nitrate may be used. Another highly satisfactory wet compress to use in this disorder is the following:

Alibour water:

	Gm. or Cc.
Copper sulfate	1.6
Zinc sulfate	5.6
Saturated camphor water q.s. ad.	240.0

Sig.: Use 2 tablespoonfuls of the above in a glass of tap water and use as a continuous wet dressing. Change dressing every two to three hours.

In addition, small doses of unfiltered roentgen rays are of value. In persons tolerant to tar, White's crude coal tar paste is often very efficacious, especially after the weeping has subsided:

White's crude coal tar paste:

	Gm. or Cc.
Crude coal tar	4.0
Zinc oxide	2.0
Corn starch	15.0
Petrolatum	15.0

REFERENCES

1. Schwartz, L., and Tulipan, L.: *Occupational Diseases of the Skin*, Lea and Febiger, Philadelphia, 1939.
2. Sulzberger, M. B., and Finnerud, C. W.: "Industrial Dermatitis; Definitions and Criteria for Diagnosis," *J.A.M.A.*, *111:* 1528–32 (Oct. 22), 1938.
3. Kesten, B., and Laszlo, E.: "Dermatitis Due to Sensitization to Contact Substances," *Arch. Dermat. and Syph.*, *23:* 221, 1931.
4. Bernstein, J. C.: "Dermatitis Due to a Sulfur-Meat Complex," *Arch. Dermat. and Syph.*, *40:* 414, 1939.
5. Bechet, P. E.: "Patch Test: Evaluation of Its Possible Dangers," *New York State J. Med.*, *39:* 829, 1939.
6. Rostenberg, A., Jr., and Sulzberger, M. B.: "A List of Substances for Patch Testing and the Concentrations to be Employed," *J. Invest. Dermat.*, *2:* 93 (June), 1939.
7. Sulzberger, M. B., and Wise, F.: "The Contact or Patch Test in Dermatology," *Arch. Dermat. and Syph.*, *23:* 519, 1931.
8. Blaisdell, J. H.: "Epidermophytosis as an Industrial Disease," *Urol. and Cutan. Rev.*, *31:* 414, 1927.
9. Peck, S. M.: "Epidermophytosis of the Feet and Epidermophytids of the Hands," *Arch. Dermat. and Syph.*, *22:* 40, 1939.
10. Osborne, E. D., and Putnam, E. D.: "Industrial Dermatoses," *J.A.M.A.*, *99:* 972 (Sept. 17), 1932.
11. Lewis, G. M., McKee, G. M., and Hopper, M. E.: "The Trichophyton Test—Its Value as a Diagnostic Aid," *Arch. Dermat. and Syph.*, *38:* 712, 1938.
12. McNealy, R. W., and Lichenstein, M. E.: "Tineal Dermatitis in Traumatic Surgery," *Illinois M. J.*, *55:* 298, 1929.

13. Parsons, A. C.: "Report on Baker's Dermatitis," *Brit. J. Dermat., 36:* 193, 1924.
14. Tulipan, L.: "Dermatitis from Cinnamon," *Arch. Dermat. and Syph., 25:* 921, 1932.
15. Van Vonna, N. C., Struycken, J., and Bonnevie, P.: "Contribution to Knowledge of Baker's Eczema," *Acta Dermat.-Venereol., 15:* 343, 1934.
16. Cummer, C. L.: "Onychia Due to Handling Sugar," *Soc. Trans. Arch. Dermat. and Syph., 41:* 142, 1930.
17. Meherin, J. M., and Schomaker, T. P.: "The Cement Burn: Its Etiology, Pathology and Treatment," *J.A.M.A., 112:* 1322 (April 8), 1939.
18. Burckhardt, W.: "Mason's Eczema," *Arch. f. Dermat. u. Syph., 178:* 1, 1938.
19. Schmidt, F. R.: "Occupational Skin Diseases," *Urol. and Cutan. Rev., 35:* 586, 1931.
20. Sequeira, J., cited by Brown, F. W.: "The Psychological Investigation of a Case of Dermatitis Artefacta," *Guy's Hosp. Rep., 88:* 356, 1938.
21. Towle, H. P.: "Gangrena Cutis Hysterica," *Med. Rec., 72:* 581, 1907.
22. Haldin, Davis H.: "Onychia as an Occupational Disease," *Brit. M. J., 2:* 845, 1931.
23. Barkan, H.: "Industrial Trauma in Relation to the Development of Ocular Tuberculosis, Syphilis and Neoplasm," *Arch. Ophth., 51:* 103, 1922.
24. Gardiner, F.: "Occupational Dermatitis," *Brit. J. Dermat., 34:* 297, 1922.
25. Parkhurst, H. J.: "Dermatosis Industrialis in a Blueprint Worker, Due to Chromium Compounds," *Arch. Dermat. and Syph., 12:* 253, 1925.
26. Klauder, J. V., Gross, E. R., and Brown, H.: "Prevention of Industrial Dermatitis, with reference to Protective Hand Creams, Soap, and the Harmful Role of Some Cleansing Agents," *Arch. Dermat. and Syph., 41:* 331, 1940.
27. Koch, F., and Peters, L.: "Follow-Up of Occupational Eczema," *Dermat. Wchnschr., 108:* 222, 1939; Abstr. *Brit. J. Dermat., 51:* 489, 1939.
28. Dainow, I.: "Treatment of Occupational Dermatoses by Vitamins A and D," *Acta Dermat.-venereol., 20:* 191, 1939.

PART VII

CHAPTER XXXIII

OCCUPATIONAL CANCER

No space will be given in this chapter to the relationship of single trauma to the production of cancer, although it is by far the more involved and constitutes a more delicate determination than cancer

Fig. 130.—Gas tar cancer on the wrist. The man is aged twenty-nine, and has worked during the last seven years as a gas-retort stoker. He says he frequently gets splashes of tar on this arm. At the age of fourteen he became a scavenger in the mule-spinning room of a cotton mill. Four years later he joined the army, in which he served four years. He thinks the growth has been present about eighteen months. The rest of his skin is apparently healthy. (White, *The Dermatergoses or The Occupational Affections of the Skin*, Paul B. Hoeber, New York.)

which arises in workmen in certain industries. This immediate discussion concerns itself primarily with cancer occurring in workmen in those industries dealing with substances or elements recognized as having a carcinogenic tendency.

Substances with Carcinogenic Properties.—These are chiefly tar, dyestuffs, paraffin, aniline, pitch, and actinic rays (Figs. 130, 131). It is rather startling to note that the majority of noxious agents dealt with in this book are derivatives of the hydrocarbons, and then to observe that in the field of occupational cancer the coal-tar and petroleum products are the chief causative agents. In addition to the ones mentioned—and to a less and very questionable degree, sulfur—arsenic, chromate, and fumes of acids and caustics have been alleged the cause of cancer.

Fig. 131.—Gas tar cancer on the scrotum. A man, aged sixty, worked all his life as a gas-works stoker. (White: *The Dermatergoses or The Occupational Affections of the Skin*, Paul B. Hoeber, New York.)

Site of Assault.—Occupational cancer is considered to be the end-result of chronic inflammation produced by exposure to irritating substances. It appears that there is a predilection for the skin, scrotum, and urinary bladder. Epidermoid carcinoma on the lip of fishermen has been observed. Cancer of the lung resulting from chronic exposure to dusts, gases, or fumes has never been established.

Relationship of Silicosis and Pulmonary Cancer.—Klotz[1] reviewed 50 cases of silicosis and compared the incidence of pulmonary cancer to that noted in 4500 necropsies performed in the Toronto General Hospital. He suggested that the incidence of cancer in those exposed to silicosis was higher than in the general population. To the author

this appears as an unfair comparison and is not in agreement with authorities on silicosis. Kuroda[2] in Japan noted cancer of the lung in several employees of gas works and began an investigation of this problem. He believed there was a relationship but would offer no conclusions until he had completed his study.

Coal-Tar and Petroleum Products.—It is not definitely known or understood just what the carcinogenic property is in the coal-tar or petroleum products. It is evident that the incidence of cancer among workers with these substances is greater in England than in America. Certain investigators have held that the carcinogenic property is less in the petroleum than in the coal-tar series. Others maintain no such differentiation can be made. Some feel that this agent is present to a greater extent at a certain fraction of distillation, supposedly from 200° to 450° C. It is contended by a few that the intensity of action and not the specific agent is responsible.

It should not be assumed that all petroleum oils have carcinogenic properties. This was brought out by Heller,[3] who, under the auspices of the Rockefeller Foundation confirmed the earlier work of Wood [4] in noting no scrotal cancer among the oil-refinery workers in Pennsylvania but did find it among the same class of workmen in the Mid-Continental oil fields of Ohio and Indiana. He stated that the latter oils contained carcinogenic compounds not present in the Pennsylvania oils. It is not uncommon to find in these refinery workers that the carcinomas are preceded by warts. Wax boils are common.

No statistics can be found regarding the incidence of cancer among the California or Gulf Coast oil workers. These oils resemble the Mid-Continental oils more than they do the Pennsylvania oils, in that they have an asphalt base. Dr. Alfred Soiland, head of the Cancer Clinic in Los Angeles, told me that he knew of no cases of cancer from this source. Mr. Phil Jones, of the Research Department of the Union Oil Company in Los Angeles, stated that if such cases did occur the incidence was so low as not to be brought to his attention. It is not known that the "cracking process" so commonly in use in this state creates or enhances the carcinogenic properties of oil, although Leitch[5] supposedly rendered an inert California oil active by heating it to 600° C.

Tar.—Tar cancer is not infrequent in fishermen. This substance is used on the nets to keep them from rotting. It becomes smeared on the hands, especially in hot weather, and is carried from the hands to the face. Fishermen have a habit of holding the needle in their mouths while the net is being mended. Shamlaugh[7] studied this type of affliction among the fishermen of Gloucester and reported eight cases of cancer of the lip. He believed that the tar was the cause and not the constant exposure to the sun.

Behan[5] pointed out the dangers of coal tar, and intimates that the constant inhalation of the dust and dirt of asphalt or tarred roads may subject the autoist to all the hazards of coal-tar cancer. This likelihood appears far-fetched indeed.

Dyes.—Gehrmann[6] reports the incidence of cancer in dye workers at the du Pont plant in Wilmington, Delaware. He states that while it has been observed that prolonged exposure to relatively low concentrations of certain aromatic amines (aniline, α- and β-naphthalenes, and benzidine) are known to cause the development of tumors in certain persons, the mechanism of development is not clearly understood. Absorption is by respiration, through the skin, and by the intestinal route, the latter being relatively unimportant. The lesions are predominantly papillomas or carcinomas of the urinary bladder. The classical symptoms are hematuria, frequency, urgency, burning, and pain. In his experience these occur only in well-advanced cases. Benign papillomas occur frequently and may be destroyed by fulguration. From his report it is evident that malignant growths occur only after prolonged exposure. The average length of time was twelve years.

Aniline Tumors.—Hueper[9] and his associates experimented with the production of "aniline tumors" in animals in order to establish some foundation for study of the occupational neoplasms and to observe any systemic effects following the prolonged exposure to one of the suspected carcinogenic aromatic amines. For this study they selected β-naphthylamine. By oral feeding and the subcutaneous injections of commercial β-naphthylamine they were able to produce preneoplastic and neoplastic papillomas or carcinomas in thirteen out of sixteen female dogs. Generalized papillomatosis was a frequent phenomenon. In addition, prolonged administration of this substance produced blood destruction, degenerative changes in the tubular epithelium of the kidneys and in the parenchyma of the liver.

Chromate Dust.—Teleky[10] contends that the inhalation of chromate dust by workers in this exposure produces a relatively high incidence of pulmonary cancer and possible cancer of the gastrointestinal tract. McNally[11] makes no mention of this possibility, and Hamilton[12] fails to cite any instance of it. She even mentions that the British medical inspectors were unable to find a single instance of carcinomatous degeneration of chrome ulcers.

Cancer of the Eye.—From eye clinics, institutions devoted to the study of cancer, from eminent ophthalmologists, and from the medical services of large industrial plants, Lane[13] collected 1000 cases of cancer of the eye. One hundred and fifty-four different occupations were represented. Her classification included exposure to oils and lubricants; sunlight and tar; sunlight and chemicals or irritants; coal, pitch, tar,

radioactive substances; and metals. Compared to the average general cancer rate per thousand, there was some increase in the number of workers exposed to oils in the automotive field. There was no increase among those who occasionally use oil in their trades, such as printers, plumbers, steamfitters, and the like. Workers exposed to the elements, tar, and chemicals showed a large increase, especially farmers and laborers. In the miscellaneous group, however, more than half had no occupational hazard; more than one third of the entire 1000 were housewives. The average age of the patients with ocular carcinoma was 57.15 years, and 48.5 for those with malignant melanomas (sarcoma). Her study does not convince this writer of the especial significance of occupational cancer of the eye. Certainly one should be very hesitant in attributing cancer of the eye in a fifty-seven-year-old farmer, for instance, to his occupation. Any such allocation would require a careful ruling out of all other factors.

Medicolegal Aspects.—The incidence of cancer of unquestionable occupational origin is low when consideration is given to the vast army of workers in contact with the alleged carcinogenic substances. Cancer of the skin from these substances develops slowly, usually taking from ten to fifteen years, at least. The growth is considered to be of low malignancy. Metastasis from these growths is rarer. Any patient presenting himself with a cancer allegedly due to occupation warrants a thorough investigation upon the following lines:

1. The nature of the substance alleged as the causative agent. Is it known to have carcinogenic properties?
2. The location of the lesion. Is it in an area commonly assaulted by such substances; or is it in a tissue or organ rarely if ever involved by these agents?
3. The time element. How long has the patient been exposed? Have others in the same industry been known to have developed similar lesions?

Upon the evaluation of such an inquiry should the decision rest.

Treatment.—The same hygienic measures recommended for all skin lesions apply to this disease. Clean working-clothes should be furnished daily, showers after work, protection of the skin by gloves, oilcloth aprons, head hoods, and ointments over the exposed parts. Operations employing suspected carcinogenic substances should be under a suction hood when possible or within an enclosed booth employing suction. In those occupations offering a known or suspected hazard, frequent physical examinations should be done in order to reveal early lesions.

REFERENCES

1. Klotz, M. O.: "Association of Cancer and Silicosis of the Lung," *Am. J. Cancer, 35:* 1–158 (Jan.), 1939.
2. Kuroda, S.: "Occupational Pulmonary Cancer of Generator Gas Workers," *Indust. Med., 6:* 304–306 (May), 1937.
3. Heller, L.: "Occupational Cancers," *J. Indust. Hyg., 12:* 169–197 (May), 1930.
4. Wood, H. B.: "Skin Lesions among Tar Workers," *J. Cancer Research, 13:* 54–59 (March), 1929.
5. Leitch, A.: "Mule-Spinner's Cancer and Mineral Oils," *Brit. M. J., 2:* 941–943 (Nov. 22), 1924.
6. Gehrmann, G. H.: "Papilloma and Carcinoma of Bladder in Dye Workers," *J.A.M.A., 107:* 1436–39 (Oct. 31), 1936.
7. Shambaugh, P.: "Tar Cancer of Lip in Fishermen," *J.A.M.A., 104:* 2326–29 (June 29), 1935.
8. Behan, R. J.: *Relation of Trauma to New Growths,* The Williams and Wilkins Co., Baltimore, 1939.
9. Hueper, W. C., Wiley, N. H., and Wolfe, H. D.: "Experimental Production of Bladder Tumor in Dogs by Administration of Beta-Naphthylamine," *J. Ind. Hyg. and Toxicol., 20:* 1–96 (Jan.), 1938.
10. Teleky, L.: "Occupational Cancer of the Lung," *J. Indust. Hyg. and Toxicol., 19:* 73 (Feb.), 1937.
11. McNally, Wm. D.: *Toxicology,* Industrial Medicine, Chicago, 1937.
12. Hamilton, A.: *Industrial Toxicology,* Harper and Brothers, New York, 1934.
13. Lane, L. A.: "Occupation in Relation to Cancer of the Eye and Adnexa," *Am. J. Ophth., 22:* 239–356 (March), 1939.

CHAPTER XXXIV

HEAT AND CLIMATIC AFFECTIONS

HEAT exhaustion, heatstroke, and heat cramps constitute an occupational hazard to firemen, stokers, mill and boiler-room workers, and to others forced to exert themselves in an atmosphere of high temperature. Under extreme heat conditions the discomfort becomes unbearable as the humidity rises. The peripheral vessels dilate, and there is an increase in the circulating blood volume. Hick[1] and his coworkers noted that if there was no rise in the rectal temperature the cardiac output remained the same, but if there was a rise in temperature there, an increase in oxygen consumption and cardiac output resulted. This state of hyperpyrexia which may be present in either heat exhaustion or heatstroke may terminate in death. In heat exhaustion there is an embarrassment to the heat-regulating center, while in heatstroke there is a paralysis of this center.

Circulatory Adjustment.—A person's ability to withstand excessive heat is a matter of circulatory adjustment. Keeton[2] and his associates studied this adjustment with healthy medical students and nurses under controlled conditions. All subjects were observed twelve hours after their last meal and after a satisfactory rest, being therefore under basal conditions. The environments used were hot dry (11.5° F. dry bulb and 70° F. wet bulb), and hot wet (99.5° F. dry bulb and 90° F. wet bulb). Their study involved observations chiefly with regard to the effect of posture. Circulation is well maintained in hot environments by healthy subjects while they are lying down. The adjustment of the circulation to the erect position becomes increasingly difficult for normal persons as environment becomes hotter and fever appears. Failure of this adjustment to the erect position leads to symptoms of faintness or fainting. These circulatory changes consist of a rise in the diastolic pressure, a lowered pulse pressure, and an increase and then leveling off of the pulse rate.

DIAGNOSIS

Signs and Symptoms. *Heat Exhaustion.*—In heat exhaustion, the symptoms simulate traumatic shock. The patient feels exhausted and faint. His face becomes pale, and the pulse quickens and becomes weak. Frequently if these patients are seen some time after they have been withdrawn from the heat environment they will show a normal

mouth temperature. However, if the rectal temperature is taken it will show fever. This lagging of the rectal temperature was noted not only by Keeton and his co-workers but also by McConnell and Houghton,[3] Adolph and Fulton,[4] and others.

Heatstroke.—In heatstroke (heat retention, sunstroke) the headache is pronounced, and dizziness is marked, as is visual disturbance. The skin becomes dry and hot, the face flushed, and the pulse full and rapid. Unconsciousness usually ensues, and the temperature may rise as high as 109° F.

Heat Cramps.—Heat cramps, with violent spasm of the abdomen and limbs, occur in workmen in hot atmospheres who drink too much water and perspire excessively, with a resultant loss of the body chlorides. Fever or elevated body temperature is not common, and the patient does not appear to be gravely ill. This condition is disappearing in industry, owing to the preventive measure of supplying chlorides to the workmen.

Pathology.—The pathological changes found in these cases of heat exhaustion or heatstroke at autopsy show certain alterations from the normal which occur quite regularly. In some instances they appear to be quite widespread, as indicated in the report of thirty-seven autopsies by Nuzum and LeCount.[5] They found edema of the brain and leptomeninges, marked general passive hyperemia, especially in the lungs and brain, edema in the lungs and spleen, cloudy swelling of the liver, kidneys, and myocardium, and hemorrhages into the mucous membrane and skin. Most of the reports in the literature, however, emphasize the hyperemia and edema of the brain, together with vascular changes of the nervous system.

Cold Weather and Cold Atmospheres.—Frostbite, pneumonia, bronchitis, coryza, and allied conditions associated with lowered temperatures frequently become claims for compensation. Policemen, firemen, cold-storage workers, and the like, as well as those of the general populace who are victims of weather abnormalities, constitute the group who present compensable or non-compensable disabilities. There is no need to discuss the signs, symptoms, or pathology of these conditions. Their status of compensability is the chief concern and is discussed under "Medicolegal Aspects."

MEDICOLEGAL ASPECTS

The medical phase of the various conditions pertaining to heat and cold is well known. The terseness of the description indicates the needlessness of informing the physician of their symptoms. But the misunderstood legal aspect of the claims which arise is worthy of a more lengthy discussion.

Arising Out of Employment.—To reiterate the principle explained in Chapter I, to arise "out of the employment" an injury or illness must be one that follows as a natural consequence from the hazard of the work being performed. Injuries resulting from inclemencies of the weather, heat, cold, lightning, tornadoes, etc., are accidental injuries. When these occur and are presented to the physician, he must determine if the disability is due to and arises out of the character of the employment. A man may suffer heat prostration while employed in a hot country, or in a district subjected to a heat wave. Such an illness is not compensable, since it is obvious that the heat is caused by the sun, over which the employer has no control, and the temperature is not the result of his employment. The general rule accepted by most states is clearly expressed in a decision in Wisconsin[6]:

As a general rule, an employer is not responsible for damages caused to a workman by lightning, storms, sunstroke, freezing, earthquakes, floods, etc. These are considered as forces of nature, which human vigilance can neither foresee nor prevent.

On the other hand, should one be required to fire a boiler during a period of excessive heat and suffer heat prostration, then the disability arises out of the work because the employee is exposed to a special risk that intensifies the general heat and directly contributes to the disability. This same may apply to policemen forced to walk a beat during extreme temperatures, or a fireman who fights a fire and becomes soaked with water during zero or freezing weather. The common-sense test in this, as in most industrial disease conditions, is: What did the employment, as contradistinguished from other causes, directly have to do with the disability?

Aggravation of Existing Disease.—An even more difficult problem than that of estimating the relationship between cause and sudden, acute affection, is the aggravation of an existing disease. For instance, exposure to cold may cause subjective symptoms of Buerger's disease to become manifest. Cold or frostbite cannot produce Buerger's disease, but it is to be conceded that an element of aggravation exists. Cold-storage men, or butchers who are frequently in and out of the refrigerator, claim colds, pneumonia, and arthritis as a result of their exposure. The justification of their claim is difficult to assay. Since there is such an element of commonalty to these conditions, the writer takes the viewpoint that the relationship is so questionable that responsibility should be the employee's.

Questionnaire on Heat Exhaustion.—Finally, the author wishes to discuss three phases of heat exhaustion which are not mentioned in

textbooks: (1) Can heat exhaustion have its onset some hours or days following an exposure to high temperatures? (2) If heat exhaustion does not terminate in death, are permanent sequelae apt to ensue? (3) What is the average length of temporary disability?

The experience of this clinic was too meager to satisfactorily answer the above questions. Most of these cases arise in large plants, or mills, where a plant physician, or medical director, is available, and we do not, therefore, see them. To obtain a complete answer, I sent seventy-five questionnaires to industrial physicians throughout America who have had adequate experience in such cases, as in the mills and plants of Carnegie-Illinois Steel, General Motors, Westinghouse Electric and Manufacturing Company, du Pont, and a number of the larger railroad shops. The questionnaire was as follows:

TABLE 24—QUESTIONNAIRE ON HEAT EXHAUSTION

1. Where does the onset of signs or symptoms occur?

   (*a*) Always while in the heat environment?———————————————

   (*b*) Have you, in your records, instances of men leaving an excessive heat environment apparently normal, only to develop evidence of heat exhaustion some hours, or days, later?

   ———————————————————————————————

   (*c*) How many (or approximate percentage)?———————————————

   (*d*) If you believe the onset of heat exhaustion could occur some time after departure from exposure, what limit would you establish as the maximum interval?

   ———————————————————————————————

2. What has been the average length of temporary disability?

   ———————————————————————————————

3. Have you had any permanent disabilities?

   (*a*) If so, what was the incidence or percentage?

   ———————————————————————————————

   (*b*) Of what has the permanent disability consisted?

   ———————————————————————————————

4. Do your first-aid men or plant physicians routinely record a rectal temperature in all suspected cases?

   ———————————————————————————————

5. (*a*) Are a majority of these cases definitely heat exhaustion without complications?

   ———————————————————————————————

   (*b*) Or do the majority present complicating factors, such as cardiac disease, alcoholism, etc.?

   ———————————————————————————————

Sixty-six questionnaires were returned completely answered: two were returned with the statement that they had insufficient experience to formulate a reply, and no replies were received from seven questionnaires. An analysis of the replies is as follows:

Question 1.—Sixty-four physicians stated that the onset always

occurred while the victim was in the heat environment. One set the upper limit of the interval between exposure and onset of symptoms as two or three hours, while the other set it at six hours.

Question 2.—The average length of temporary disability indicated from the replies was not longer than twenty-four to thirty-six hours.

Question 3.—In the experience of these sixty-six qualified physicians there had not been a single instance of permanent disability. Such positive evidence makes one wonder if the morbid anatomy reported in heat exhaustion is present only in extreme instances, in which the organs have been suddenly overwhelmed, causing immediate death; if they do not produce immediate death, the changes are transient.

I feel that the observations of these experienced physicians, many of whom have been leaders of thought in industrial medicine, will greatly aid in clarifying the status of those claims which, not frequently, but occasionally present themselves.

Illustrative Cases:

Case I.—The claimant, during the course of his employment as a carpenter, collapsed from sunstroke or heatstroke. The temperature was 106° F. The state industrial commission awarded compensation for temporary total disability, and the employer and his insurance carrier brought suit in the Supreme Court of Oklahoma to review the award.

The circumstances under which sunstroke may constitute a compensable injury, said the court, were set forth in Stanolind Pipe Line Co. *vs.* Davis, Okla. 47 P. (2d) 163. In that case it was held that an injury caused by sunstroke arises out of employment when the employee is placed, by the nature of his work, in a position or under circumstances subjecting him to a greater hazard of injury by sunstroke than other people in the same vicinity who are not engaged in such work. In other words, the employment must increase the danger of being injured by sunstroke. In the present case, the conditions under which the claimant worked were, in the opinion of the court, such as to accentuate the heat and subjected him to a risk greater than that to which other persons not similarly employed were exposed. The injury was, therefore, compensable.

The claimant, however, was awarded compensation for temporary disability for a longer period than he had actually proved. The cause was remanded to the commission, therefore, for further hearing as to the actual duration of the claimant's disability.[7]

Case II.—The claimant, a policeman, sustained frostbite of his hands while on night patrol duty during the winter. Gangrene set in, and "some amputation was necessary." From an award of the workmen's compensation bureau granting him compensation, the township

of Woodbridge, his employer, appealed to the Supreme Court of New Jersey.

The weather was unusually cold, said the Supreme Court, and the claimant's employment obliged him to use his hands and prevented him from seeking shelter. He was exposed to a risk different from that of the public generally, because he was on duty from 6 P.M. to 4 A.M., with the exception of a lunch hour. The claimant, the court concluded, sustained a compensable accidental injury arising out of and in the course of his employment.[8]

Case III.—Death from exposure to the elements, including the heat of the summer and the cold of the winter, said the Supreme Court of Iowa, is not compensable if the hazard is the same as that to which the general public is exposed. For compensation to be recoverable for death from sunstroke, the deceased must have been subjected to a greater hazard from heat than that to which the public generally in that locality was subjected. This distinction is recognized by all the authorities. Compensation was denied in this case.[9]

Case IV.—The claimant's husband was employed by the defendant company to deliver ice on a commercial route. On July 11, 12, and 13, the heat was extreme, the temperature reaching a maximum of 106° F. during the day. On the evening of July 11 he was more tired than usual after his day's work. On July 12, after delivering ice until noon, he quit for the rest of the day. He complained of the heat and staggered on walking. On July 13 he returned to work, but a helper made most of the deliveries for him. Late that afternoon, on his way home from work, he was overcome by the heat and suffered what was described in the medical testimony as a "collapse." He died the following day, and an autopsy confirmed the diagnosis of heatstroke as the cause of death. The claimant thereupon brought proceedings under the Workmen's Compensation Act of Minnesota against the employer for compensation for the death of her husband. From an order of the industrial commission awarding compensation, the employer appealed to the Supreme Court of Minnesota.

The employer contended that the claimant's husband had not sustained an accident within the meaning of the Workmen's Compensation Act because this particular heatstroke was "of slow, gradual onset." The Supreme Court, however, could not agree with that contention, citing Minnesota cases to show that in that state heatstroke is accidental *per se*. Following those decisions, the court held that the heatstroke in the present case was accidental. Furthermore, in the opinion of the court, the accident occurred in the course of the deceased's employment even though his collapse had not occurred during his working hours. It is enough, said the court, that the causative agency was applied during the employment and was followed directly and immediately by the collapse without the intervention of a cause

independent and unrelated to the employment. Accordingly, the Supreme Court affirmed the order of the industrial commission awarding compensation.[10]

One notes in this case the peculiar line of defense set up; that is, the contention that the condition was not an accident, owing to the slow, gradual onset. And also is noted the wording of the court that "the causative agent was applied during employment." This reasoning is directly opposite to than handed down in the preceding case. It is contrary to the accepted viewpoint.

Case V.—The claimant, in the course of his employment as a truck-driver for a transportation company, was exposed to the rain and to chilling winds because of the lack of glass in the windows of the cab. He contracted a severe cold. Congestion of his throat, chest, and lungs, associated with an elevated temperature and the coughing up of blood, developed. Finally he was forced to go to bed, where he remained for about three weeks. He then attempted to return to work but was so weak and suffered such pains and aches that he went back to bed. Thereafter he was unable to perform manual labor. He claimed that as a result of his illness he tired easily; was unable to work more than a few minutes at a time; that his heart had been affected, causing him pain and suffering at intervals; that he had lost weight and was debilitated; and that his lungs had been affected and weakened. The industrial accident board denied him compensation under the Workmen's Compensation Act of Texas, whereupon he brought suit to set aside the board's decision. From a judgment of the trial court denying relief, he appealed to the Court of Civil Appeals of Texas, Fort Worth.

The purpose of the Workmen's Compensation Act, said the court, is to compensate an employee for injury sustained during the course of employment. The injury contemplated is defined by the act as "damage or harm to the physical structure of the body and such diseases or infection as naturally result therefrom." It was not contemplated that the act should cover health insurance. It is a matter of common knowledge that colds, influenza, and pneumonia are the result of germs attacking the body and that many such germs appear to be in the very atmosphere surrounding us at all times. Any and every person is exposed to them without being conscious of the fact. Medical science teaches that human beings fall victims of these germs because at the time they are not physically able to withstand the assaults of the germs. If, said the court, the employee in this case is permitted to recover compensation, then every employee who is engaged in labor which tires the body and causes what the layman calls a "run-down condition" can recover compensation by showing that his work weakened him and lowered his resistance and caused him to succumb

to the attack of a disease which he would otherwise have been able to resist. Attempts so to enlarge the purpose of the Workmen's Compensation Act will destroy its usefulness, it was ruled.

Accordingly, the Court of Civil Appeals affirmed the judgment of the trial court upholding the order of the industrial accident board denying compensation.[11]

Case VI.—A butcher employed in a chain store in the suburbs of Los Angeles awoke one night with a nosebleed which defied home treatment. The patient was taken to the hospital where periodic bleeding occurred over a period of ten days. The workman was unemployed for two additional weeks. He filed for compensation, alleging that the frequent trips in and out of the refrigerator during the warm weather had produced this condition. He was substantiated in his belief by his physician.

The past medical history was unimportant, his only illness being typhoid fever twenty years previously. The physical examination was essentially negative. The blood pressure six days after onset was 138/80. According to his physician it was 142/80 at the time of onset. Urine showed a heavy trace of albumin; the blood count was normal at the time of hospitalization. Blood Wassermann negative.

The nosebleed occurred nine hours after he left his employment and while at rest in bed. No known cause could be found to account for it. The defense contended that nosebleeds are common and appear in many persons without known provocation. It had never been recorded that nosebleed occurs in refrigerator workers or cold-storage men more frequently than in men of other occupations, and medical literature fails to reveal any association. It was held that the claimant's viewpoint was untenable. The decision in this case denied the patient's claim.

Case VII.—The plaintiff was employed by the defendant to operate a machine removing snow from the highway. His work required him to keep his eyes constantly on the snow. On March 23 he worked at his task for twelve hours, the day being "quite warm for that time of year" and the sun shining brightly. At the end of the day he was burned about the face, as if sunburned, and his eyes were inflamed. The skin of his face was desquamated. His eyes grew worse, and during the week an ulcer developed in the right eye, and on or about May 18 another developed in the left eye. There was a severe infection and ulceration on the conjunctivae and the corneas of both eyes, with the appearance of pus. The plaintiff instituted proceedings against his employer, under the Nebraska Workmen's Compensation Act. An award in his favor by the compensation commissioner was sustained by the district court, and the employer appealed to the Supreme Court of Nebraska.

The condition of the plaintiff, said the Supreme Court, was produced by "snow blindness" or "snow ophthalmia." The condition is rare in the climate in which the plaintiff was working, and therefore the court concluded that it was unexpected and unforeseen. There was no dispute as to the existence of a "burning" in snow blindness, and the injury to the eyes of the plaintiff was therefore violence to the physical structures of the body. The injury, said the Supreme Court, was due to the reflection of ultraviolet rays of sunlight from the bright snow, which had to continue for several hours before it manifested itself or became known to the person exposed. In this instance, the condition manifested itself on the day of exposure and therefore may be said to have occurred suddenly. In the light of these facts, the judgment of the court below sustaining the award of the compensation commissioner was affirmed.[12]

Case VIII.—For about ten years the workman in this case had been employed by the defendant petroleum company to fire furnaces and boilers. On May 28, in adjusting a valve on a particular boiler, he was forced to look at the flame for five minutes to adjust the flame to a target in the boiler, a task he had performed many times before. Thereafter he became blind and instituted suit under the Workmen's Compensation Act of Louisiana. The trial court gave judgment against the workman, and he appealed to the Court of Appeal of Louisiana, first circuit.

The evidence before the trial court showed that the workman had had syphilis for several years before the incident to which he attributed the loss of his sight and that his vision had been impaired. A physician who had examined the workman about fourteen months after the alleged industrial accident testified that he found him suffering from an advanced stage of neurosyphilis and that the optic nerve was atrophied. There was medical evidence to the effect that syphilis is the principal cause of atrophy of the optic nerve, although it is not the sole cause. Medical experts testified that heat and glare, without some severe burns around the eyes, would not cause atrophy of the optic nerve and that exposure to heat and glare in firing a boiler would not affect or aggravate a syphilitic condition so far as it relates to the optic nerve. If, said the Court of Appeal, the glare and heat from the boiler or furnace served to aggravate or hasten the atrophy of the optic nerve because of the weakened condition of the nerve brought on by syphilis, and if this heat and glare on that particular day was unusual and out of the ordinary routine of the day's work, it could be said that the heat and glare from the boilers had a causal connection with the blindness, and the workman would be entitled to compensation. There was, however, no evidence to this effect. On the contrary, the evidence showed that the atrophy of the optic nerve and the resulting blindness were caused by syphilis, not

by the exposure of the workman to the heat and glare of the boilers. The workman, therefore, failed to show that his disability was caused or superinduced by an accident or injury in the course of his employment. The judgment in favor of the employer was accordingly affirmed.[13]

TREATMENT

Prophylaxis in Reactions to Heat.—As aids in the prevention of heat exhaustion, heatstroke, and heat cramps, better working and improved living conditions, rest periods, and suitable light clothing are of value. Stokers, firemen, and the like, often find it of value to wear heavy shirts which are kept soaked with water, instead of light clothing. Certain employments, where excessive heat is necessary, have introduced air-conditioning systems, but these are not always feasible.

Sodium Chloride.—The most valuable preventive measure, however, is the use of sodium chloride tablets for periodic daily ingestion; or, still better, the use of 0.1 to 0.5 per cent solution of sodium chloride chilled to a temperature of from 45° to 50° F. for drinking-water. The sodium chloride tablets may cause gastric irritation, while the physiologic saline solution does not.

Glucose.—For the prevention of heat exhaustion, the use of glucose in the form of jelly drops containing approximately equal parts of glucose and granulated sugar has been reported to be very effective. Beverages, such as lemonade sweetened with sugar, may be equally beneficial, and the lemonade will help with the glucose to prevent acidosis. These procedures are also of value in the prevention of heatstroke, and in addition it has been stated that heatstroke is absolutely preventable if the patient is treated immediately as a case of hyperpyrexia as soon as he presents himself with a dry skin indicative of suppression of perspiration.

The Medical Director of E. I. du Pont de Nemours and Company writes[14]:

> . . . since we have subjected all of our men who are exposed to high temperatures to a very careful examination, with special efforts to determine any disease of the circulation and eliminating any who have any circulatory disturbance as shown by careful examination, we have had no difficulty. Furthermore, most of our men who are exposed to high temperatures are now and have been taking at least 100 Mgm. of vitamin C each day, and this seems to be a very decided factor in preventing any circulatory changes as a result of heat exhaustion.

Heat Cramps.—The treatment in heat cramps depends on the replacement of sodium and chloride ions in the blood serum. This is accomplished by the administration of 0.9 per cent sodium chloride

by intravenous infusion and orally, or by hypodermoclysis. Intravenous infusions of this physiological saline with from 5 to 10 per cent dextrose may also be used. The patient should be kept quiet for from twenty-four to forty-eight hours and encouraged to drink milk and other salt-containing fluids and foods.

Heat Exhaustion.—The treatment in heat exhaustion is similar to that in shock. The patient should be kept warm if the temperature is subnormal, and care should be taken that the temperature does not rise quickly to dangerously high levels. Intravenous infusions of 0.9 per cent sodium chloride with from 5 to 10 per cent dextrose should be given. The use of one of the following may be necessary for treatment in circulatory collapse: caffeine sodium benzoate, 7½ grains (0.5 gm.); epinephrine, 1:1000 solution, from ½ to 1 cc.; coramine, 1.5 cc.; or metrazol, from 1½ to 4½ grains (0.1 gm. to 0.3 gm.).

Heatstroke or Sunstroke. *Lowering Body Temperature.*—In heatstroke or sunstroke, the opposite treatment is given. The patient is removed to a cool atmosphere at once, and the body is sprinkled or sponged with cool water, with fans placed to evaporate the water. This is continued until the body temperature is lowered to normal, although some authorities advise not lowering it below 101° F. During this time the head is elevated, and the cutaneous circulation is maintained by vigorous friction. The use of ice baths, packs, or enemas is usually not advised, since they may so diminish the circulation of the skin as to drive the blood to deeper portions of the body and thus contribute to further circulatory collapse.

Fluids.—These should be given orally, intravenously, or by hypodermoclysis. For the intravenous infusions 0.9 per cent sodium chloride with from 5 to 10 per cent dextrose is used, while the physiological saline without dextrose is usually used for the subcutaneous administration. Venesection of from 400 to 500 cc. is performed if cyanosis, venous distention, or pulmonary edema is marked.

Artificial Respiration and Circulatory Stimulants.—Artificial respiration may be necessary, as may also the circulatory stimulants mentioned above in the treatment of heat exhaustion. These stimulants are to be avoided as much as possible, however, since they sometimes make the patient worse and are reported frequently to produce numerous petechial hemorrhages in these cases. Following the acute stage there should be advised prolonged rest in bed, and the patient should avoid high temperatures for the remainder of his life.

Prophylaxis in Reactions to Cold.—Prevention of reactions to cold atmospheres is best secured by interrupting work in such atmospheres by short periods of rest in a warm atmosphere. At the first sign of numbing and blanching of the skin, the worker should leave the cold

environment, since the longer he is exposed to such cold the more severe will be the degree of frostbite. Footwear and handwear should be carefully fitted and should in no instance be constricting. Those suffering from cardiovascular diseases or from diabetes mellitus should not be employed where they will be subjected to cold atmosphere.

Sensitivity to Cold.—For sensitivity to cold the immediate treatment consists of injecting epinephrine hydrochloride, or of immediate plunging of the arms into water at 107.6° F. for relief of the local lesions. Desensitization of these patients can sometimes be accomplished by the taking of cold baths with gradual decrease of the temperature of the bath and increase of the length of the period of immersion. Gradually increasing doses of subcutaneous histamine have also been used.

Frostbite.—For frostbite no well-proved method of treatment is known. The lesions should be thawed slowly, since severe pain, burning, and tingling are apt to occur if they are otherwise treated. In addition, rapid thawing at times seems to give rise to the development of an inflammation of an exudative type which is accompanied by more damage than that produced by the freezing alone. Some authors, however, hold that the rapidity of thawing makes little difference. Vertical suspension during the thawing and for several days thereafter has been advised, as has the use of heated air. A cold cloth or ice may be used in a rubbing process to cause friction and thus induce reestablishment of circulation. If open lesions are present, strict surgical cleanliness is needed, and surgical dressings should be applied to blistered areas. Minor traumatism or infection should be warned against. For the general treatment of those who have suffered from extreme cold, hot blankets, a heat cradle, and hot drinks internally are indicated.

REFERENCES

1. Hick, F. K., Keeton, R. W., Glickman, N., and Wall, H. C.: "Cardiac Output, Peripheral Blood Flow and Blood-Volume Changes in Normal Individuals Subjected to Varying Environmental Temperatures," *Heating, Piping, and Air Conditioning, 2:* 50 (Jan.), 1939.
2. Keeton, R. W., Hick, F. K., Glickman, N., and Montgomery, M. M.: "The Peripheral Type of Circulatory Failure in Experimental Heat Exhaustion; the Role of Posture," *Heating, Piping, and Air Conditioning, 12:* 122 (Feb.), 1940.
3. McConnell, W. F., and Houghton, F. C.: "Some Physiological Reactions to High Temperatures and Humidities," *Heating, Piping, and Air Conditioning, 29:* 129, 1923.
4. Adolph, E. T., and Fulton, W. B.: "Effects of Exposure to High Temperature upon Circulation in Man," *Am. J. Physiol., 67:* 573, 1924.
5. Brahdy, L., and Kahn, S.: "Heat Exhaustion and Heat Stroke," *Trauma and Disease,* Lea and Febiger, Philadelphia, 1937, p. 345.
6. Hoenig *vs.* Industrial Commission, 159 Wis. 646, 150 N. W. 996, L. R. A. 1916–A, 339 (1915).

7. Smith *vs.* Zweifel, Okla. 54 P. (2d) 649.
8. Matthews *vs.* Woodbridge Tp., N.J. 183 A. 150.
9. Wax *vs.* Des Moines Asphalt Paving Corporation, Iowa 263 N. W. 333.
10. Ueltschi *vs.* Certified Ice and Fuel Company, Minn. 276 N. W. 220.
11. Amann *vs.* Republic Underwriters, Texas 100 S. W. (2d) 778.
12. Hayes *vs.* McMullen, Neb. 259 N. W. 165.
13. Laughlin *vs.* Magnolia Petroleum Co., La. 182 So. 178.
14. Personal communication from Dr. G. H. Gehrmann, Medical Director, E. I. du Pont de Nemours and Company.

CHAPTER XXXV

ELECTRICAL INJURIES

Injuries to the body resulting from lightning or electrical forces are so comparable that no differentiation is to be made here, nor is it of practical importance to consider the factors which are likely to play a part in determining the extent of injury, such as voltage, amperage, and type of current, for it is not probable that a knowledge of these factors will enter into the diagnosis, treatment, or medicolegal aspect. A discussion of electrical injuries finds a place in this book since the effects are primarily medical, in contrast to the surgical nature of most traumas. Another interesting fact regarding electrical injuries is that whereas in most occupational diseases the etiology or occupational origin is to be questioned, it is readily apparent in this instance, since the occasion and effect of such an injury are dramatic, instantaneous, and often observed by others at the time of occurrence. It is the questionable sequelae with which the medicolegal phase is most concerned.

Occupational Hazards.—These need not be delineated since electricity is a necessary adjunct to almost any phase of man's endeavor. However, some workers are under a greater hazard than others; for example, electricians, linemen, power-plant operators, tree surgeons, operators of electrical transportation equipment, and the like.

Physical Findings and Symptoms. *Burns.*—Next to bone, the skin is most resistant to electrical current and, therefore, burns are almost an invariable finding. The lesions vary in appearance. Some may appear punctate or may resemble a bullet wound; others are oblong, round, or linear. "The area is slightly elevated and pale gray, grayish-white, yellowish-gray, or yellow. The heat makes the horny substance of the epidermis plastic."[1] Deep burns may extend into the underlying structures, muscles, and even bone. Flash burns of the eye are not infrequent. Cataracts, with minute tears to the lens capsule, have been reported and have been thought to be due to a concussion effect. Cataracts resulting from high-tension current and appearing two years later have also been reported. The reason is not clear to the writer, and any instance of such a claim should be referred to the ophthalmologist.

Unconsciousness.—If seen soon after the electrical contact, the patient may be unconscious. This phase may be from a few seconds

to several hours in duration. Restlessness, excitement, and muscular twitchings or convulsions are sometimes present. Cyanosis has been noted. If the contact has been severe, the patient is invariably in shock. After regaining consciousness the patient may complain of ringing in the ears, light flashes, and other hallucinatory phenomena. Paralysis of one or more of the extremities, with sensory disturbance, may occur. Upon recovery from immediate effects of the injury, symptoms resembling a disorder of the nervous system may have to be evaluated. These strongly resemble a hysteria and will subsequently be discussed.

Pathology.—Since animals differ greatly in their resistance to electrical currents, experimentation has been of little value. Most of the pathological studies are to be found in the foreign literature. Such reports indicate that a fairly frequent incidence of neuropathological conditions is to be found following electrical injury. Most of the articles reviewed in the American literature quoted the observations of Critchley.[2] The following abstract of Critchley's description was made by Pollock[3]:

(*a*) Focal petechial hemorrhages scattered throughout the brain, especially the medulla. They may be found in the spinal cord, chiefly in the anterior horns. When the cranium has been struck by lightning, large vascular tears may be found in the cerebrum.

(*b*) Chromatolysis, especially of the pyramidal cells, of the medullary nuclei, and of the anterior horns and the Purkinje cells of the cerebellum. The changes are patchy in distribution, so that healthy and diseased cells may lie in close proximity.

(*c*) Curious wide dilatation of the perivascular spaces, bridged across by delicate strands, and surrounded by a zone of condensation, supposed to be due to bubbles of gas, occurring particularly in cases of electrocuted criminals.

(*d*) Changes in the peripheral nerves, such as fragmentation and tortuosity of the axons, breaking down of the sheath of Schwann, and infiltration of the epineurium with endothelial cells.

(*e*) After very severe injury, as from a direct stroke of lightning, or in experimental electrocution with high tension currents, the entire brain and parts of the cord may be swollen, softened, and even diffluent. The neuronic changes are variable, and Critchley was unable to demonstrate them in three cases, in one of which death occurred after a shock of 66,000 volts, in another seventeen days after a shock of 132,000 volts, and in the third, eight days after a shock of 19,000 volts, death being due to the burns and toxemia. The

changes described in the literature are not specific for electrical injury, as it is difficult to differentiate these changes from those produced by heat, toxemia, and cerebral concussion.

Thus it is apparent that both the clinical findings and the pathology indicate that the nervous system alone is damaged by strong electrical current. When death is immediate, however, it may be due to paralysis of the respiratory muscles or from fibrillation of the heart, or both. Postmortem examinations fail to reveal any lesions in the heart due to electrical injury. The heart is often empty, although the right side may be full of fluid blood. It should be reiterated that although ventricular fibrillation is the common cause of immediate death, there exists no evidence that permanent damage exists in the heart of one who has recovered from electrical injury.

Medicolegal Aspects. *Temporary Disability.*—Complications of the burn may prolong the temporary disability; otherwise temporary disability is short. It is sometimes difficult to get patients who have been injured by electricity or lightning to believe that they have not been seriously injured. I do not mean the patients who evidence hysteria or malingering, but rather a small group who believe that electric shock is a devastating experience and who require time to adjust themselves. Diplomatic handling of these patients by the physician at the time of injury will usually prevent such an attitude, or at least greatly allay their fears.

Permanent Disability.—Permanent disability is not common. Damage to the brain or spinal cord may leave a permanent disability of varying degree. It is held that disability may appear months after the injury, owing to necrosis, secondary hemorrhage, or disturbance of the central nervous system due to cell degeneration. Such instances have not been experienced at this clinic. When a disability is claimed because of a heart defect, hysteria or malingering must be suspected.

In the chapter on neurosis and malingering it is mentioned that the element of fright or severe mental shock is considered by some as a causative factor of the so-called "traumatic neurosis."[4] If fright and shock are to be accorded recognition as causing neurosis or hysteria, then it would be expected that the incidence of neurosis or hysteria following electrical injury would be great. However, it does not seem to be a prevalent aftermath.

True epilepsy, paralysis agitans, and multiple sclerosis are to be differentiated from the sequelae of electrical shock. Naville has been unable to find in the literature an indisputable example of epilepsy, general paralysis of the insane, or multiple sclerosis following electrical injury.

Treatment. *Prophylaxis.*—The prevention of these injuries depends upon educational measures designed to indicate the inherent dangers in electrical force and upon the use of measures for safety in industry and in the general public.

Artificial Respiration.—The patient who has suffered a severe electrical shock should immediately be given artificial respiration by the Schaefer method. This is continued until he breathes naturally or until all evidence that life has ceased is present. The criteria for death in such cases differ to some extent from those in other cases, since no reflex reactions may be present whatsoever. The patient should not be pronounced dead until rigor mortis sets in or until cooling of the body occurs. Artificial respiration may need to be continued for hours. If natural breathing ceases after it has been once restored, artificial respiration should be commenced again.

Prevention of Ventricular Fibrillation.—The use of epinephrine in these cases is to be particularly avoided. Ventricular fibrillation appears to be a common cause of death from electric shock, and it has been shown that epinephrine may also induce fatal ventricular fibrillation in conditions in which the heart is hyperirritable from the effects of poisoning such as benzene and chloroform. Hoff and Nahum[3] used acetyl-β-methylcholine chloride for the prevention of ventricular fibrillation by electric shock and found that epinephrine counteracted the effect of the acetyl-β-methylcholine chloride, or in larger doses increased the possibility of fatal ventricular fibrillation. This work to some extent explains the reasons for avoiding epinephrine in treatment of other cases of poisoning, *e.g.*, carbon tetrachloride, and raises the question of whether epinephrine may not be inadvisable in most of the toxic states which have been discussed in this text. The clinical use of acetyl-β-methylcholine in these cases is suggested by this work of Hoff and Nahum.

Rest.—Patients in severe cases of electric shock should be kept in bed for two or three weeks because of emotional as well as physical shock. There may also be latent effects which may to some extent be avoided by such bed rest.

Measures in Burns.—Electrical burns may frequently be treated like other burns. Hemorrhage rarely immediately follows such burns, but secondary hemorrhage may occur after shock has been controlled and the arterial blood pressure rises. All of these burns are treated as serious, no matter how trivial they may appear at first examination. Most authorities recommend avoidance of early surgical intervention, but some recommend early débridement and excision of the burned region beyond the hyperemic margin, followed by suturing. The conservative method permits nature to build up new tissue beneath or

around the destroyed tissue, and is felt by most to lead to the most satisfactory ultimate results. It is felt particularly that tendon sheaths, etc., may be opened to infection by early surgical intervention. Edwards and Bowie[6] gave details of a conservative regimen which had given very satisfactory results in their experience. According to their routine, the surrounding skin was thoroughly cleaned with soap and water, and then dried with ether followed by alcohol. Compresses were used on the surrounding skin for from twenty-four to forty-eight hours. Then sterile dry dressings were applied until the coagulum began to separate. The surrounding normal skin was then covered with vaseline or lanolin, and azochloramide in triacetin or olive oil (preferably olive oil) was applied to the burned area. Dressings were changed daily, and the coagulum was permitted to curl. The curled, loosened edges were removed as rapidly as possible. When the base of the ulcer presented uniform, healthy granulations, skin-grafting was done.

REFERENCES

1. Jaffe, R. H.: "Electropathology," *Arch. Path., 5:* 837, 1928.
2. Critchley, M.: "Neurological Effect of Lightning and of Electricity," *Lancet, 1:* 68, 1934.
3. Pollock, L. J.: "Trauma and Diseases of the Nervous System," *Trauma and Disease,* Lea and Febiger, Philadelphia, 1937, p. 334.
4. Davidson, H. A.: "Neurosis and Malingering," *Am. J. M. Juris., 2* (Feb.), 1939.
5. Hoff, H. E., and Nahum, L. H.: "Role of Adrenalin in the Production of Ventricular Rhythms and Their Suppression by Acetyl-β-Methylcholine Chloride," *J. Pharm. and Exp. Therap., 52:* 235 (Nov.), 1934; and *Am. J. Physiol., 110:* 675 (Jan.), 1935.
6. Edwards, C. R., and Bowie, H. C.: "High Tension Electric Burns," *Am. J. Surg., 47:* 299 (Feb.), 1940.

CHAPTER XXXVI

CAISSON DISEASE

Caisson disease, frequently referred to as "the bends," or less frequently as "compressed-air illness" or "diver's palsy," is caused by a sudden reduction of the atmospheric pressure at a ratio which exceeds 2.0 or 2.5 to 1. Few physicians will be called upon to diagnose this disease or institute treatment for it, since it is peculiar to a type of employment in which the danger is so well known that trained men are constantly on hand to recognize the symptoms and treat the patient promptly.

Occupational Hazards.—Those affected are: divers, builders of tunnels and bridges, or wherever compressed air facilitates construction underneath water.

Pathogenesis.—The solution of gases in fluid depends upon their partial pressures (Dalton's law). Variations in pressure in a man's normal environment are negligible; therefore, no symptoms are produced, but under high barometric pressure, blood reaching the lungs is exposed to increased partial pressures of nitrogen and oxygen which are taken up in amounts dependent upon the existing pressure. These are carried to the tissues where the oxygen is readily absorbed, but the nitrogen, being inert, is not absorbed and so accumulates and diffuses into the tissues. This process of taking on nitrogen in the lungs and giving it off into the tissues is repeated as long as exposure exists. All tissues do not absorb the nitrogen in like amounts. While fat is saturated more slowly than other tissue, it has a greater capacity and desaturates more slowly. Hence nitrogen storage occurs to a high degree in the white matter of the central nervous system. The gray matter is less affected. Upon the reduction of the barometric pressure, the nitrogen in the blood escapes into the alveolar air, and the nitrogen in the tissues comes out of solution. If this occurs rapidly (without proper decompression) bubbles of nitrogen will form in the tissues, veins, venules, and capillaries. These bubbles, or more accurately air emboli, compress the tissues, obstruct the blood flow and cause hemorrhage. Because of multiple hemorrhages, the pathology is varied.

Signs and Symptoms.—Abdominal pain, often girdlelike in type and excruciating in severity, is common, the victim usually bending over—hence the phrase "the bends." Dizziness, nystagmus, and vomiting are frequently associated symptoms. Pulmonary edema may be

present with evidence of dyspnea. In the more severely exposed, aphasia, diplopia, changes in the visual fields, monoplegias or paraplegias, and such manifestations of cord involvement as urinary incontinence, are observed. Requarth and Benson[1] state that at low pressure a blockage of the eustachian tube, with attendent inability to equalize the pressure in the middle ear, is apt to occur, especially in those who are troubled with pharyngitis and sinusitis.

Medicolegal Aspects.—The etiology of the disability is always obvious. Temporary disability is usually short, but will depend upon the resolution of hemorrhagic areas. Permanent disability may result.

Treatment. *Prophylaxis.*—Preventive treatment is dependent upon a careful physical examination and proper selection of applicants. Men above thirty should not be employed unless they have had previous experience and are known to tolerate the exposure. Men above forty should not be employed. In addition to age, other factors barring employment should be obesity, vascular impairment or sclerotic changes, alcoholism, and upper respiratory disease or a disposition to such, and limitation of the number of working hours.

Decompression.—Operations in compressed air are now generally carried out in such a manner that the workman who is returning to the normal atmosphere passes through chambers which are graduated in their pressure so that by the time he reaches normal atmosphere he has been gradually decompressed. Actual decompression is still carried out upon the lines suggested by Haldane in 1906. The first step is to reduce the absolute pressure by one-half. A time interval is then permitted to elapse during which the body comes into equilibrium. Pressure is again reduced by one-half, then another time interval is allowed, and so on until normal pressure is reached. The duration of the intervals depends upon the extent of maximum pressure and the duration of exposure.

Recompression.—In the actual treatment of caisson disease, recompression is the best therapeutic measure. Specially designed chambers have been constructed in which the various levels of atmospheric pressure may be controlled and the stages of decompression repeated. Helium, because it diffuses more rapidly, has been proved experimentally to be of extreme value. As yet it has not had extensive clinical trial.

REFERENCE

Requarth, Wm. H., and Benson, R. E.: "Compressed Air Illness," *Indust. Med.*, 9 (March), 1940.

PART VIII

CHAPTER XXXVII

THE MEDICOLEGAL RELATIONSHIP OF TRAUMA TO DISEASE

THE original design of this book did not provide for any discussion of the relationship of trauma to disease. But in collecting material for the preceding pages, in hearing and reading the testimony given before the Industrial Accident Commission, and in the perusal of case reports submitted to me for an opinion, it became apparent that in medicolegal controversies, medical opinion is frequently not based upon postulates which are recognized as acceptable. Nor does the average medical witness appear to know where these postulates might be found. It was believed therefore that a book of this nature would serve its purpose better by including the precepts concerning the relationship of trauma to disease which have been evolved from the experience of the passing years. Obviously space will not permit the application of these precepts to all of the diseases of the various systems. What follows is merely a concise consideration of the general principles which should be applied to any given case in rendering an opinion regarding the occupational relationship.

Desirability of Scientific Approach.—Can trauma cause diabetes, acute yellow atrophy of the liver, tic douloureux, or appendicitis? Will a strain cause a gastric ulcer to rupture, or produce hemorrhoids, or epididymo-orchitis, or kinking of the ureters? What is the relationship between a single trauma and cancer, syphilis, or tuberculosis? Confronted by claims of this nature, a physician may make dogmatic statements not founded upon past experience or in accord with the prevailing opinion. He may succumb to the wishful thinking of the claimant or defendant. The following cases serve to illustrate this point.

Case I.—On December 5, 1932, a workman fell, contusing his back and hip. For this he was paid compensation. On March 21, 1933, he was operated upon for appendicitis. On July 31, 1935, more than two years later, he was operated upon for a strangulated bowel resulting from adhesions. The following day he died, and his widow then brought suit for death benefits on the grounds that the fall in 1932

had caused the appendicitis in 1933, which resulted in adhesions, causing strangulation of the bowel and death in 1935. A physician who treated the patient just prior to his death testified that he believed that, "The inflammation resulted from the industrial accident and, by a process of contiguity, progressed through the muscles, organs and tissues of the body from the locus of injury through the psoas and other muscles, organs and tissues in the lumbar region and through the peritoneum and finally reached the appendix, necessitating the appendectomy three months later." Upon the decision of the jury a verdict was rendered in favor of the widow. The decision was eventually reversed by the Texas Court of Appeals.

Or, consider the merits of the following case, which the author was asked to review prior to a hearing before the industrial accident commission.

Case II.—A welder had assisted three other men in lifting a piece of sheet metal. He noted nothing that day, but two days later he developed abdominal cramps and diarrhea. In trying to account for this condition he recalled the lifting episode of two days previously. He related this history to his physician, who made a diagnosis of mucous colitis following strain. The patient entered claim for medical care for a disease resulting from industrial trauma.

A third example of "loose thinking" can be found in this instance of recent experience.

Case III.—A twenty-two-year-old male presented himself, stating that he was suffering from pains in the joints, especially in the hips, knees, and wrists. Thirteen months previously he had slipped while mopping the floor and fell into a sitting position. This accident was so trivial that he continued working and did not seek medical care. One year later he noted the multiple joint pains, some loss of weight, shortness of breath, and general fatigue. At this time he consulted a physician who told him he had arthritis. The boy then told the physician of the previous accident and wondered if the condition could have been caused by this fall. The physician said it was likely that the fall had caused or aggravated the arthritis. At the time he was examined at our clinic he had a history of repeated attacks of tonsillitis, and the examination presented a typical picture of rheumatic fever.

These three examples illustrate a credulity manifested by some physicians that is not warranted by the facts or findings. A healthy skepticism, bred of long experience and of considered thought, appears desirable. It has been constantly reiterated within these pages that

the meaning of the Workmen's Compensation Act has been extended within recent years. Previously the awards of compensation were to victims of an obviously direct injury in which the elements of time and place as related to injury were not remote. Today laymen present claims wherein the relationship of trauma to disease is very remote or entirely absent. An entire book could be filled with such instances. For this state of affairs, the medical profession can be held largely accountable.

Reason for Discredited Testimony.—As a profession we are steeped in the belief that ours is an art, not a science. Yet our advancement is due to scientific discoveries. It is fallacious to think that our entire success has been due to the laying on of hands. Our specific therapeutic measures are based upon proved procedures, as in the treatment of lues or diphtheria. Much of our present knowledge concerning the diagnosis and treatment of certain diseases resulted from the establishment of positive corollaries by investigators after prolonged observation of the behavior of diseases, such as tuberculosis, smallpox, malaria, typhoid, and the like. But despite the fact that we daily rely upon proved truths, we are inclined to feel that we deal only with the speculative. In contrast, the legal mind relies upon precedent, even though mistakes of past decisions are perpetuated and project themselves into future decisions. In legal practice, what is infallible today may be erroneous tomorrow. Because of this attitude of sureness on the part of the legal profession and the tendency on the part of the physician to vacillate, the courts are inclined to discredit medical testimony in concrete questions. Law takes advantage of the old adage—when doctors disagree. It is high time the medical profession realized that certain fundamental facts concerning the aspects of disease are constant or rarely vary. We need to create a greater spirit of solidarity in the expression of our opinions.

Formulating Relationships between Trauma and Disease.—In accepting or denying a single act of trauma as the cause or aggravating factor of a disease, some of the cautions cited in the first portion of the book bear repeating. Common sense, balanced judgment, and a mind free from preexisting prejudice are the first essentials of a sound opinion. Next in importance is a knowledge of the disease under consideration and of the accepted postulates regarding the production of disease by trauma. Also, the physician on the witness stand must guard against being induced by the legal inquisitor to make an admission of probability when under studied circumstances the physician would hesitate to admit a remote possibility. One of the pitfalls of medicolegal controversy is the confusion and delusion arising out of the meaning of the words "likely" and "perhaps."

Classification.—How should the physician proceed in formulating his opinion regarding the relationship of trauma to disease? He reasons best who fashions his thoughts in an orderly manner. Heraclitus once said of his predecessors that they had much knowledge but little sense. The sensible application of knowledge resides in logical deductions—especially is this true in the type of case under discussion. When any given case presents itself, it should first be classified in one of the three following groups:

1. A group representing cases which obviously are the result of trauma, as infection in the site of a burn, laceration, or fracture
2. A group representing cases which never result from trauma, as diphtheria, trichinosis, or hemophilia
3. A group which consists of diseases usually not produced by trauma but which might on occasion result from or be aggravated by trauma, as toxic goiter; urinary calculi forming after a fracture of one or more vertebrae; or a rupture of a gastric ulcer following a forceful blow to the stomach

The first two groups do not concern us at this time. If the case under consideration falls into the third group, then the validity of the claim must be tested by applying further reasoning. This is done by comparing the factors of the case in question with the recognized postulates as they are applied to a given disease. These postulates vary in their application to different diseases. For instance, those which are adhered to in a case of alleged traumatic appendicitis differ from those to be considered in a case of carcinoma. As previously stated, space forbids a discussion of all the possible diseased conditions which might arise from trauma. A few illustrations must suffice.

Trauma and Appendicitis.—Let us consider the debatable question of traumatic appendicitis. There are those who hold that a direct, severe contusion to the right lower abdominal quadrant may initiate appendicitis in an otherwise healthy, normal appendix. Moorehead[2] sees no more relationship between external trauma and appendicitis than between a blow on the neck and tonsillitis. Others deny the possibility of initiating appendicitis by trauma but admit the possibility of aggravating a previously diseased appendix. This state of uncertainty regarding traumatic etiology of appendicitis is true of many other conditions. But the experience of the majority should aid in forming an opinion in most cases.

Postulates to Be Satisfied.—Rhodes and Birnbaum[3] (quoting Shutkin and Wetzler) set forth postulates which to the writer appear most sound. They are:

1. There must have been absolute freedom from abdominal complaints associated with pain, nausea, vomiting, and tenderness before the trauma.
2. Direct trauma must be severe and forcible, involving the abdominal wall in the right half especially.
3. Indirect trauma must be violent, acute, and unexpected.
4. Symptoms must appear immediately after the trauma.
5. Symptoms must be persistent and progressive, assuming the signs and symptoms of acute appendicitis.
6. The pathological findings must indicate a suppurative, destructive, or necrotic process.

With these postulates before us, let us apply them to two cases.

Case I.—A board from a ripsaw flew back with great force striking the operator on the right side of his abdomen. He suffered immediate pain, which subsided greatly within a half hour. He felt too knocked out however to continue working, so he went home. During the night he was awakened with cramps in his belly, and at noon of the next day he began to vomit. Late on the second day after injury, his condition being worse, he was operated upon. At that time the abdominal wall was rigid, especially on the right, where he also had marked tenderness. The skin in the lower right quadrant showed deep, purplish ecchymosis. He had a temperature of 101.2° F.; pulse of 108; leukocytes, 16,500; with a polymorphonuclear count of 84 per cent. Upon opening the abdomen there was evidence of hemorrhage into the muscles, the appendix was lying over the pelvic brim, and was gangrenous at its distal end, with a small perforation present. There was no evidence of adhesions or previous inflammation.

This case satisfies the postulates previously outlined. The blow was direct and forceful, symptoms appeared immediately after injury; they were persistent, progressive, and assumed the recognized signs and symptoms of acute appendicitis. The findings at operation indicated an acute process with no evidence of previous inflammation.

Case II.—While cartons of furniture polish were being stacked, one of them fell from the stack a distance of two feet, striking the workman a glancing blow across the right upper abdomen. He subsequently alleged that it hurt him at the time but that he continued working. Three weeks later while in a movie he became nauseated. The next day he vomited several times and noted mild pain in his right side. The vomiting and pain subsided but four days later recurred more severely, at which time he was operated upon. Following recovery he entered claim for compensation and medical costs. The operative notes of his surgeon stated that the appendix was rectocecal

and bound down by adhesions. The pathologist reported the lumen obliterated and healed scars in the submucosa and in the interstices of the musculature.

The facts in this case do not satisfy the postulates for traumatic appendicitis. The blow was neither severe nor direct. No symptoms were produced immediately or reasonably soon afterwards. The operative and pathological findings gave evidence of previous attacks. A check-up among his fellow employees revealed that the patient had complained on prior occasions of "stomach-ache" or "indigestion."

Appendicitis Following Strain.—Many cases could be cited in which the allegation is made that appendicitis followed a strain of varying degrees of severity. None of these will be illustrated, since I have never seen nor read of a case of appendicitis following strain which came near satisfying the requirements of the above postulates. In my experience the relationship of strain to appendicitis has been either coincidental or too remote to be seriously considered. The postulates outlined for traumatic appendicitis can be applied in most instances to other acute abdominal conditions, such as rupture of a peptic ulcer, or of the intestines, spleen, liver, and so forth.

Trauma and Tuberculosis.—Let us consider the relationship of trauma to tuberculosis. Lesions other than pulmonary tuberculosis will be dispensed with by simply stating that tuberculous infection of traumatized tissue, the finger for instance, by way of the bloodstream from a distant focus rarely, if ever, occurs. The truth of such a statement resides in the knowledge that subjects with active tuberculosis of the lungs frequently injure themselves without developing this type of infection in the traumatized part. Also, we know that trauma to the chest or lungs does not initiate tuberculosis. The study of wounds to lung tissue incurred during the first World War is sufficient proof of that. But how shall we judge the case wherein the claim is made that pulmonary tuberculosis set in after an injury to the chest or lungs?

I suggest that when a claim of this type awaits a physician's opinion, he first consider the general known facts about tuberculosis. It is a widespread, infectious disease, occurring in all walks of life without being induced or aggravated by trauma. Its incidence among the unskilled laboring class is greater than in the non-industrial class. Its incidence is greater in the productive years, between twenty and fifty years, than at any other time. In weighing the facts, other statistics must not be overlooked. Men in industry receive frequent injuries to the chest, yet the claims for tuberculosis arising from injury are less than the incidence of tuberculosis in the general population. In

Kessler's series of 1107 cases of chest injuries, less than 2 per cent claimed tuberculosis as a result of injury, which is the normal incidence of this disease among the American population. If tuberculosis is higher among the laboring class and injury common, we would expect the incidence to be far higher if a relationship existed. In addition to these general considerations, the elements of time, severity, location of injury, past medical history, the worker's health at the time of injury and whether tuberculosis already existed, are to be evaluated.

Postulates to Be Satisfied.—The following postulates are suggested when considering the acute, immediately active case of tuberculosis:

1. Severe injury, as indicated by:
   (*a*) Ecchymosis, fracture of ribs, tear of muscles, etc.
   (*b*) History from eye witnesses
2. Immediate (or within a few hours) evidence of:
   (*a*) Hemoptysis
   (*b*) Pneumonia or bronchopneumonia
   (*c*) Pneumothorax
   (*d*) Pleurisy with effusion
   (*e*) Fever
3. Good health just prior to alleged injury
4. Positive findings of tuberculosis:
   (*a*) Tubercle bacilli in the sputa
   (*b*) Manifestation of a tuberculous process in the *x*-ray films

In applying the above postulates I believe one should demand a trauma of sufficient degree before admitting it as the aggravating factor (Fig. 132). It is true that cases have been reported where an acute active tuberculosis followed a trivial strain. In such an instance I should want to be most certain that the subject was not about to have a "breakdown" and that the strain was not purely coincidental. And also, a sudden, active tuberculosis should not be attributed to a trauma which allegedly occurred a year or two previously, when in the interim the patient has been in good health and has not complained of any disturbance following the trauma. It is held by some that severe trauma to the chest may induce pathological changes which permit the slow progression of the disease with the appearance of signs or symptoms six weeks or several months later.

Let us apply these postulates to several examples.

Case I.—A fireman in the course of his duty one cold winter night became drenched with water. He was in these wet clothes a number of hours. The following day he developed chills, fever, and cough; and examination of the lungs revealed congestion. Pneumonia set in, which failed to resolve, and subsequently he developed a dry cough, after-

noon temperature, night sweats, loss of weight, and other indications of tuberculosis. Examination revealed tubercle bacilli in the sputa, and the *x*-ray films showed right apical involvement.

The severity of injury was sufficient. While not a direct trauma to the chest or lungs, it was an injury to the body which lowered his resistance, permitting pneumonia to set in, which further lowered the resistance and gave rise to the activity of tuberculosis. The onset was immediate. The immediate signs and symptoms indicated lung

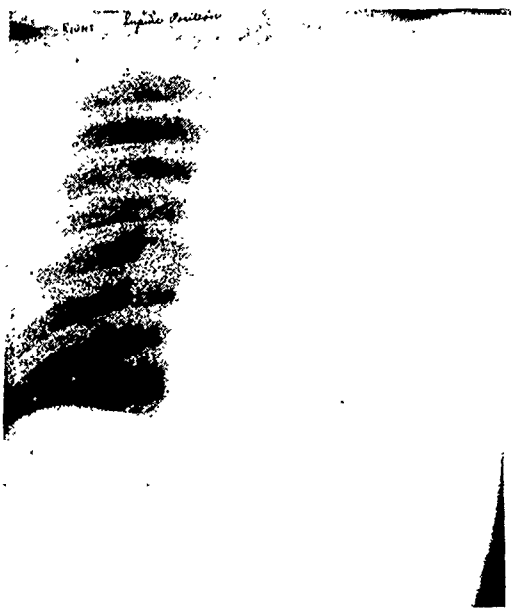


Fig. 132.—Tuberculous pleural effusion. This case was not discussed in the text. A thirty-four-year-old carpenter fell twelve feet, striking his chest against a projecting cornice. No ribs were fractured. Four days later he began to spit up blood, developed a cough, and noted fatigue. Twelve days later his malaise was so marked that he was forced to bed. This film was taken one month after injury, at which time tubercle bacilli were profuse in the sputum and were recovered in the aspirated fluid. History of previous good health was proved.

infection. His previous health had been established, for he had three weeks before passed his medical examination. Finally, the tubercle bacilli were recovered from the sputa, and the *x*-ray films revealed apical involvement in keeping with the picture of tuberculosis.

Case II.—Following the accidental release of the brakes of a heavy truck, it crushed the driver between the endgate and a loading platform. He suffered great pain and within half an hour had a severe hemoptysis. These occurred at intervals for the next several days.

During the first two weeks he complained of severe pain resulting from three fractured ribs. The stereo films revealed several small cavities as well as one large cavity in the right apex with bilateral fibrosis. During the first few weeks he was markedly ill, ran a fever, and coughed a great deal, and tubercle bacilli were present in the sputa.

The trauma was severe, and evidence of injury was present. The evidence of lung involvement was immediate. At the time of trauma his general state of nutrition was good, his weight in keeping with his age and height. He had worked on the same job for ten years without loss of time due to illness, and his employer stated that the patient had been unusually energetic. Tuberculosis was proved to be present soon after the injury. Although the *x*-ray examination left no doubt of a previous pulmonary tuberculosis, the patient denied knowing he had ever had it. Regardless of any skepticism one might have in an instance of this type, it must be admitted that all evidence pointed to his being apparently well, healthy, and able to work prior to the trauma. One must, therefore, conclude that the case was compensable.

Case III.—A girl, occupied as a file clerk, alleged that as she pulled out a cabinet drawer she noted a pain in her side. That night she coughed up some blood. A continuous cough with occasional blood-streaked sputum over the next several days induced her to see her family physician, who found active pulmonary tuberculosis present. Some months later she filed for compensation, alleging the act of pulling open the drawers of the cabinet "weakened her and brought about the onset of tuberculosis." Investigation showed that she was twenty-two years old, 5 feet 8 inches tall, and weighed 102 pounds. The employment record indicated frequent absences from work over a period of two years due to colds, and one month prior to her discovery of the disease she had been sick with a "very heavy chest cold." Her fellow-employees testified that they had noted her coughing a great deal for a number of months and that she had complained to them of a loss in weight and constant fatigue. The family history showed the mother had died four years previously of pulmonary tuberculosis.

When the postulates are applied to this case, it fails to meet them. The drawers of the file were on ball bearings, light and easy to manage. The alleged cause, therefore, could not be considered in any way a trauma, from either single or accumulative effort. The history indicated an insidious progression of the disease over a long period of time. A known contact existed in that the mother had had tuberculosis. Finally, the presence of hemoptysis on the evening of the day

she noted a pain in her side was coincidental and not the result of a single effort.

Claim for Latent Tuberculosis.—All claims do not contend that the signs and symptoms of tuberculosis present themselves immediately following a trauma. There are numerous cases recorded, or have been personally examined, where the claimant alleged his pulmonary tuberculosis was the result of an injury sustained a year or more previous to the onset of the disease. In contrast to the immediately active tuberculosis, this type of case might be called the claim for latent tuberculosis. For such the following *postulates* might be considered:

1. Sufficiently severe trauma
2. Reasonable length of time between the trauma and onset of the disease
3. Presence of one or more signs or symptoms during the interim—the bridging evidence
4. Good health prior to the alleged injury
5. Positive findings of tuberculosis

Case IV.—A cook, upon entering the kitchen early one morning to begin the day's work, noted the presence of strong fumes and discovered a leak in the refrigerator. While attempting to locate the leak, he was almost overcome by these fumes. His eyes burned; he coughed violently and suffered a throbbing headache along with a feeling of constriction in the chest. The cough lasted for several days, as did the sense of constriction in the chest. Within one week the victim was entirely normal, being discharged from further medical care. One year and four months later he consulted a physician because of a dry cough and afternoon fatigue. A diagnosis of early pulmonary tuberculosis was made and confirmed. Sometime later he filed for compensation, alleging the tuberculosis to be the result of the previous gassing.

Investigation brought out these facts. The gas was sulfur dioxide, a single exposure to which produces no permanent effect upon the lung. Following this exposure he continued working for that employer for six months, during which time he made no complaint of any residual disturbance. Following this six-month period, he changed positions twice, each time being required to undergo a physical examination. Six weeks prior to the discovery of pulmonary tuberculosis he went fishing in the high Sierras, caught a cold, and was absent from work for ten days.

Workmen exposed to the fumes of sulfur dioxide are frequently seen in our clinic. The writer has personally seen in the last five years about one hundred such patients. In none of them has there been a temporary disability beyond a few days or a week or two, at the most.

In none has there been any permanent morbid anatomy of the lung. In many, the initial exposure has been much greater than that experienced in the case under discussion. So, we can conclude that the trauma or injury was not severe. The interval of time is too long between date of injury and the onset of symptoms. This gassing certainly did not introduce tubercle bacilli into the lungs, and if it had aggravated a preexisting condition, the evidence of aggravation should have been apparent sooner than one year and four months. There were present no bridging symptoms—such as cough, fatigue, loss of weight, afternoon temperature, and the like. The condition of health prior to injury was good, but an illness which could have lowered his resistance was known to have been present six weeks previous to the onset of the signs and symptoms of tuberculosis. This case does not satisfy the postulates.

Case V.—While lumber was being stacked, the pile gave way, causing the patient to be buried underneath it. Upon being removed, the patient complained of chest pain and was brought to the clinic. *x*-Ray study at that time was confined to a picture of the lower rib cage, which showed fractures of the ninth and tenth right ribs. He was accorded the customary treatment, but he was the father of eight motherless children and because of financial circumstances refused to stop working. Nine months after the trauma to the chest, the patient was incapacitated from further work because of active pulmonary tuberculosis. He was sent to a sanatorium. He did not suspect or claim any relationship of his disease to the trauma of the chest, but an inquiring friend did.

Investigation revealed that a public health nurse visited his children, one of whom was a cripple. She testified that shortly after the accident to the claimant, the children told her their father was not well, that he coughed a great deal, was observed to spit up some blood occasionally, complained of fatigue, and had been losing weight. On one occasion a physician, called in to see the crippled child, examined the father and told him he had bronchitis.

This case was opposed by the insurance carrier. It was my testimony that the relationship of the trauma to the disease could not be denied. I based my opinion chiefly upon two of the postulates. First, he had suffered a sufficiently severe trauma. Secondly, there was evidence of definite bridging symptoms. The children of this claimant were good witnesses, although they did not realize the significance of their testimony. Their observations were made to a nurse without prompting or prejudice. These bridging symptoms began soon after the injury and were progressive. In the writer's experience, this is the

only case of latent tuberculosis that satisfies the postulates, and my opinion was that the trauma aggravated an inactive tuberculosis.

Case VI.—A lifeguard at the beach slipped on the steps of his observation platform, skinning his right knee and right chest. Two years later he developed pulmonary tuberculosis. A claim of tuberculosis resulting from the injury mentioned was filed. During the interim, as a student at a local college, he was a member of the water-polo team, and of the track team. Periodic health examinations by the school physician revealed no evidence of disease.

This case does not meet the stipulations of the postulates. His trauma was not severe, no immediate lung disturbance appeared. During a period of two years there were no bridging symptoms, and the claimant exhibited extraordinary strength and vigor.

Trauma and Cancer.—That cancer can follow a single act of trauma is a highly controversial question and one which must await further scientific investigation before dogmatic statements can be made. At the present time any such relationship remains speculative and doubtful. Regarding this relationship, Leila Knox states[4]:

In studying the possibility of the production of a malignant tumor by a single injury, it is important to keep in mind the established facts concerning the etiology of cancer and to understand clearly all the factors which are thought to be involved in the production of a neoplasm. These biological and chemical facts may or may not find their way into the evidence which determines legal liability, in cases where a decision has to be made as to the influence of a single trauma in the causation of a malignant growth. Since verdicts and claims are presumably based upon such evidence as is presented before the court, legal decisions are not the final word concerning the possible relationship between a trauma and a malignant tumor, for the evidence adduced may not be of such a nature as to afford a true explanation of the occurrence. Possibly such evidence is, at the moment, not available. The fact that it is not available may be determined by the lack of medical knowledge concerning the plaintiff's state of health previous to the alleged accident, and by the absence of other important data necessary for reaching a conclusion as to the true nature of the condition under consideration. The patient may refuse a biopsy on the tumor on legally justifiable grounds, and thus prevent a microscopic study of the tissues, which is regarded by the medical profession as essential for diagnosis. Thus, in any human case, many scientific facts may be missing, and the court must render its decision upon what is produced before it in evidence. Hence, it is possible that, under certain conditions, the decision may of necessity be contrary to the real

facts, which can only be revealed at autopsy after the plaintiff's death. The use of modern methods of investigation, such as the roentgen ray study of tumors of bone, has helped to establish the fact that many neoplasms, which in the past were considered to be due to injury, are definitely not of traumatic origin. The court also is largely dependent upon the competence of the medical evidence, and it is sometimes impossible for any accurate opinion to be rendered, when the evidence is more or less contradictory. A remedy for this phase of medical testimony has long been discussed but no practical solution of the problem has been reached.

History.—In considering the relationship of a single trauma to cancer, a complete, exacting, and relevant history is most important. Such a history must evaluate the patient's viewpoint, for in most every instance the patient is swayed in his story by his own wishful thinking or by ideas which have been disseminated to the laity regarding cancer. Such a history must be supplemented by facts obtained from every available source, which includes friends and relatives, fellow-workmen in the shop, neighbors, family physician, and previous employment examinations.

Postulates to Be Satisfied.—A set of postulates to test a claim of cancer resulting from single trauma has been suggested by various writers and in the main is as follows:

1. Authentic trauma

To establish this, not only the patient's statement, but others from eyewitnesses must be secured. Sometimes these cannot be obtained and, therefore, the validity of the claim will have to depend upon the remaining postulates.

2. Sufficiently severe trauma

This may be described by the patient or by eyewitnesses. Also, medical reports of one or more physicians who have seen the patient immediately after the injury are to be sought. The presence or absence of evidence of trauma such as swelling, ecchymosis, laceration, hemorrhage, and the like, aids in the estimation of the adequacy of the trauma.

3. Normal tissue in wounded part before injury

While the patient may claim that his health was normal and the part of the body involved was in a healthy condition just prior to injury, such fact cannot be assumed as true or accurate from a medical and scientific standpoint. This is a difficult postulate to adhere to in many instances. However, occasionally biopsy, *x*-ray examination, or

other procedures may establish the fact that the tissue involved contained a growth or neoplastic change which by its character must have been present prior to the date of injury.

4. Tumor or malignancy in the area that was damaged by trauma

Occasionally this cannot be held to in a rigid sense, as in lung injuries in which the principle of contrecoup might be applied, the lesion being found in contralateral lung. Brain injuries often follow this principle. However, the site of the tumor must be *related* to the site of the injury.

5. Reasonable time interval between trauma and appearance of tumor

No set rule can be applied for this postulate, for the interval is variable. This is easily understood when one realizes that a tumor may be present for a considerable length of time but yet be unnoticed by the victim. In addition, there is a relationship between the time interval and the type of malignancy; for instance, it is believed that sarcoma may develop much more rapidly than carcinoma. In general, it can be held that the longer the interval, the less likely the tumor should be considered the result of trauma.

6. The presence of bridging symptoms

This postulate considers the evidence of a continuity of symptoms between the time of injury and the period of malignancy. For instance, when normal tissue has been traumatized and the wound fails to heal or continues to discharge for a long period of time, or a swelling fails to recede, then the relationship between the development of malignancy to the preceding injury must be strongly considered—a relationship which if proved in any medicolegal case would be most difficult to deny.

7. Diagnosis of neoplasm

Often a biopsy may prove the presence of a slow-growing tumor which antedated the injury. Often, however, the biopsy can throw no light on the length of time the growth has been present. *x*-Ray examination of parts of the body distant to the malignancy may reveal a primary lesion and thus establish that the part claimed to be malignant as the result of injury is actually a metastatic lesion.

Limitation of space forbids any consideration of the effects of trauma to the heart, nervous system, or to other systems not mentioned here. The purpose of this short discussion of an extensive sub-

ject is to offer a pattern which can be followed or enlarged upon by the reader. It is hoped it suggests to the reader that, in a majority of cases, definite opinions can be expressed by the physician based upon sound thought and reasonable postulates and that we physicians do not need to vacillate in medicolegal testimony.

REFERENCES

1. Texas Employer's Insurance *vs.* Burnett, Texas 105 S. W. (2d) 200.
2. Moorehead, J. J.: "The Relationship of Trauma to Inguinal Hernia," *Am. J. Surg., 27* (Feb.), 1940.
3. Rhodes, G. K., and Birnbaum, W. D.: "Traumatic Appendicitis," *California and West. Med., 53* (Oct.), 1940.
4. Knox, L. C.: "Trauma and Neoplasms," *Trauma and Disease,* Lea and Febiger, Philadelphia, 1937, p. 441.
5. Kessler, H. J.: *Accidental Injuries,* Lea and Febiger, Philadelphia, 1931, p. 352.
6. Brahdy, L., and Kahn, S.: *Trauma and Disease,* Lea and Febiger, Philadelphia, 1937, p. 442.

CHAPTER XXXVIII

NEUROSIS ASSOCIATED WITH TRAUMA—MALINGERING

ORDINARILY the name applied to any disease entity has a uniform meaning to all physicians. When lues, bronchopneumonia, malaria, or measles is spoken of, the clinical and pathological picture of each disease registers no gross dissimilarity upon the individual minds of a varied medical audience. But what does "traumatic neurosis" mean? From the profuse material to be found in medical literature devoted to this subject, it is impossible to extract a clear-cut definition that finds universal acceptance. The condition has been referred to as "terror neurosis," "litigation neurosis," and "post-concussional neurosis." Traumatic neurosis usually follow injuries to the head, but it frequently follows injury to other parts of the body as well. In my experience, back disorders not easily recognizable as of psychic origin form a large group. By comparing the opinions of a few of the outstanding thinkers in the field of neuropsychiatry concerning this subject, the reader may be aided in forming a concept of practical value.

In order that the starting-point for the evaluation of traumatic neurosis may be of common origin, patients with objective signs of brain injury and those with definite evidence of brain damage or defect, in the nature of convulsions or mental deterioration, should be excluded from consideration.

Elements of Syndrome.—Wechsler[1] breaks down the remaining post-traumatic syndrome group into the four following divisions: (1) malingering, (2) traumatic hysteria, (3) concussion or traumatic encephalopathy, and (4) traumatic neurosis. Since malingering is to be subsequently and separately discussed, it will be omitted here. Of the remaining three groups, Wechsler writes:

Traumatic Hysteria.—The diagnosis of traumatic hysteria must be based on one set of negative and one set of positive criteria. Patients falling in this group give either no history of unconsciousness or only a history of very brief loss of consciousness, show no clinical signs of brain injury, no evidence of fracture on the *x*-ray plate, no history of subarachnoid bleeding in cases in which a spinal tap was done, and, in cases in which grave doubt as to diagnosis justify an encephalogram, no evidence of a cerebral pathologic condition. Merely negative evidence, however, is not sufficient, and the diagnosis cannot be made by exclusion alone. There must also be positive evidence that the trauma

called forth psychogenic symptoms; that there is a hysterical reaction in an individual who has an underlying neurotic personality make-up. The patient makes unconscious use of the trauma to solve personality difficulties. The accident brings to a head a series of inner and outer conflicts which the patient was unable to face squarely or to solve adequately. The trauma precipitates the neurosis, sets in motion the same mental mechanisms as in any other hysteria and serves as a convenient solution of industrial, social, marital, familial or psychosexual conflicts. It should be emphasized that the individual is not consciously aware of the mental mechanisms; when he is, one is not dealing with hysteria. Nor must the litigation motif be paramount. Not that litigation and compensation do not play some role; but when they play the chief role or become the sole cause for the perpetuation of symptoms one comes dangerously close to malingering. The trauma may therefore be regarded as a definite precipitating and, in this sense only, as a causative factor. It is interesting to note that compulsion and obsessional neuroses are never, or rarely ever, precipitated by head trauma.

Concussion or Traumatic Encephalopathy.—This occurs in a fairly large percentage of patients suffering trauma to the head. In eleven cases in the series the diagnosis of encephalopathy was made outright and in seventeen a diagnosis of concussion plus hysteria was made; that is, a total of 28 per cent. In every case there was a history of more or less prolonged unconsciousness. The symptoms were entirely subjective in all of them and in but few more were there even minimal clinical signs of organic disease of the nervous system.

There is an increasing tendency to speak of concussion of the brain in terms of traumatic encephalopathy; in fact, the two terms are beginning to be used interchangeably. There is no doubt that the old definition of a molecular disturbance does not hold any longer. The presence of minute hemorrhages, glial changes, cell and fiber degeneration, disturbed dynamics of the cerebrospinal fluid within the skull, ventricular distortion, and brain atrophy justifies the designation of traumatic encephalopathy. Martland's work on brain changes in prize fighters threw great light on the subject. The investigations of Foerster and Penfield and of Bielschowsky also added to the knowledge. More recently Rand and Courville have studied the effect of head trauma on the fiber system of the brain. The encephalographic studies of head injuries by Friedman and others have shown the extent of ventricular and brain changes in patients whose main complaints were of a subjective nature. Kennedy has laid down a few excellent criteria for the appraisal of the effects of head injuries. Without citing further references to the literature it may be emphasized that concussion is not a simple process and that it consists of fairly definite pathologic changes consequent on trauma to the head, which is ac-

companied by unconsciousness at the time of the accident. The diagnosis of concussion or encephalopathy is beginning to depend, therefore, more and more on other criteria than on those furnished by the symptomatology. In itself this differs in no wise from that of traumatic hysteria. One must rely chiefly on the history of prolonged unconsciousness, on the presence of "small signs" of neurologic involvement, on the absence of psychogenic causal factors, on the persistence and severity of symptoms, and on the evidence furnished by the encephalogram when other facts are not sufficient to establish a positive diagnosis.

Traumatic Neurosis.—This is, in my opinion, a comparatively uncommon condition. I made the diagnosis in but five of the hundred cases of head injury. The correctness of the statement as to its infrequency obviously depends on the acceptance of certain criteria for diagnosis. If traumatic neurosis and traumatic hysteria are used synonymously and interchangeably, the terminology is not only confusing but rests on sheer caprice, in which case one of the terms should be discarded. If, on the other hand, one is dealing with two distinct neurologic entities, as I believe to be the case, then it becomes incumbent to delimit them and, if possible, to adhere to a more strict definition of each term.

The clinical syndrome of traumatic neurosis differs little from that of hysteria but is not dependent, as far as one can tell, on the unconscious use of the accident to solve difficulties that have nothing to do with the trauma. That is, one is not dealing with a patient who had a neurotic personality to begin with. As far as possible the existence of concussion or traumatic encephalopathy must also be excluded. This leaves a small group of previously well adjusted individuals, who, following a serious threat to life, in which there was comparatively mild or even trifling injury to the head, beget a stubborn set of symptoms which are not amenable to suggestive therapy or other forms of psychotherapy. Their condition bears resemblance to some of the war neuroses of soldiers who had been through particularly harrowing experiences, which they subsequently relived in their dreams. To use a psychoanalytic explanation, there is narcissistic regression, but the ego instincts rather than the psychosexual instincts are affected. Traumatic neurosis might possibly be grouped with the true or actual neuroses, in which neurasthenia, anxiety neurosis and hypochondriasis are included, but the intrapsychic conflict with regard to the ego or self-preservation instincts, which is assumed to exist in traumatic neurosis, would argue against its inclusion. Whatever the explanation, the fact remains that there is a small group of patients, without demonstrable cerebral pathologic changes, in whom hysterical mechanisms cannot be demonstrated, in whom compensation or litigation does not play a predominant role, who are resistive to treatment and who find it diffi-

cult or impossible to return to their occupation at which they sustained the accident. It is to this group that the diagnosis of traumatic neurosis may be correctly applied.

Traumatic Neurosis as Effort to Resolve Conflict. — Davidson[2] states:

A neurosis is a set of symptoms which, to the best of our knowledge, do not spring from organic or structural change in the body. It is an effort to solve a conflict. Traumatic neurosis is no exception. Here, too, an unconscious conflict tills the soil from which the symptoms bloom. Conflict between what factors? First, at the time of the trauma, is the fright, the shock, the pain—in a word the body-threatening and life-threatening experience of the accident itself. Second appears the influence of suggestion. The patient hears the physician remark to the nurse: "Look at the way those nerves are damaged. He's lucky the whole arm isn't paralyzed." And the suggestion thus offered burrows into the patient's unconscious mind until it has become part of it. Third comes resentment at real or fancied lack of sympathy or understanding. An over-anxious plant doctor urges the still neurotic patient to return to work, implying that his symptoms are unreal. The brusque company examiner barks commands of "Stand-up!" "Put out your tongue!" et cetera, until the patient feels that he is being treated like a piece of machinery. The skeptical adjuster drips disbelief as he lists the symptoms with undisguised contempt. In all, the injured plaintiff faces a battery of unsympathetic, disbelieving officials, whose authority breeds resentment and evokes unconscious memories of punishing school principals, driving foremen and military martinets. Finally, the mind of the patient, already traumatized by pain and shock, influenced by suggestion, humiliated by disbelief, is challenged by one of life's fundamental conflicts: the conflict of adventure vs. security. The security of damages or compensation forms one horn of the dilemma; the adventure of being an independent wage-earner forms the other. Here, then, is the basic source of the conflict which underlies traumatic neurosis. Independent employment is uncertain, but adventurous. Offering theoretically unlimited opportunities for earning money, it carries the seal of community approval and self-respect. It stands for uncertainty. Against this is the security which comes with a check for several thousand dollars, or a guaranteed weekly compensation payment. Between the *unconscious* desire for adventure and community approval and the *unconscious* desire for security, is a genuine conflict. To say that the patient coolly weighs the advantages of being healthy and employed, against the advantages of being idle but compensated, and then makes a deliberate decision to stay sick, is to be blind to the complex psychologic factors involved.

Classification Based on Etiology.—Fetterman,[3] who, as Examiner for the Industrial Accident Commission of Ohio, has had a wide experience with a large number of cases, makes the following classification. It is not based on symptomatology but upon the principle of dominating causative factor:

Type I: "Injury" neuroses due to severe trauma to central nervous system
(*a*) Traumatic encephalopathy with symptoms of a neurosis
Type II: "Industry" neuroses induced by relationship of injured person to work
(*a*) The trauma offers an "escape," a flight into illness from duties which are disagreeable or dangerous
Type III: "Indemnity" neuroses motivated by a combat for compensation
(*a*) The possibility of indemnity mobilizes the neurotic tendencies towards illness
Type IV: "Inherent" neuroses due to essential nervous difficulties
(*a*) The injured individual (always nervous) "projects" the blame upon the trauma for his inherent weakness
There are many mixed forms.

Insurance as Motivation.—Reinhardt[4] believes that after the patients with severe trauma to the brain are excluded, the remainder, many of them with concussion, are influenced in their emotional symptoms by the factor of insurance. He cites the experience that among the uninsured and in the pre-insurance era, patients who suffered a concussion of the brain cleared up in a shorter time than those who are insured.

As Psychological and Sociological Problem.—Lewy[5] takes the stand that the problem is primarily a psychological and sociological one and contends that compensation should be denied every patient with post-accident neurosis if no permanent organic damage exists, believing that when the general public became aware of such a law, there would be a rapid decline of claims. He says:

A definite change of opinion came when the existence of a volitional factor as a contributing element was discovered by more careful psychological examination. It became evident that neither the event of the accident nor the subjective experience of being in an accident created the accident neurosis. The accident neurosis was created in part by the subjective conviction of having been in a compensative accident. In addition to the definite volitional factor, different favoring circumstances may exist for the patient—financial worries, family troubles, a wish to escape from responsibility—but they only provide

the *motive* for the volitional factor. Those responsible for the damage cannot be held responsible for the wishful thinking on the part of the patient, even though we grant that this volitional factor is a stranger to the conscious mind of the patient. The volitional factor becomes a purposeful factor in a post-accident neurosis and the development from the genesis of the dominating idea of being incapacitated to the hysterical accustoming and fortification of the whole psychological set-up is a simple one. But the results of any severe shock will wear off after a short time if there is no volitional factor seeking to gain something from the experience.

Interpretation in Terms of Subconscious and Organic Factors.—Strauss and Savitsky[6] evidence an attitude diametrically opposite to that of Lewy. They say:

The clinical features—headache, dizziness, irascibility, abnormal reaction to effort, vasomotor instability, fatigability, intolerance of intoxicants and of changes in weather, are evidences in almost all cases of alteration of the activity of the intracranial tissues. Precisely the same reactions occur in non-litigation patients as in those who can claim compensation.

In a later paragraph these two observers qualify their opinion somewhat:

While we admit the frequent existence of a volitional trend, its role and significance can be evaluated only in the setting of the subconscious and organic factors. Its importance must not be overestimated. When such a volitional element is discerned, there exists an all too frequent tendency to consider the whole reaction as malingering and to evaluate the whole case from this angle.

Strauss and Savitsky feel hurt by the attitude of a large part of the medical profession. They feel that:

The frequent expression of unjustifiable skepticism on the part of examiners engenders resentment, discouragement, and hopelessness, and too often forces people to resort to more primitive modes of response (hysteria).

As Evidence of Organic Changes in Brain.—The paper of Strauss and Savitsky from which the above quotation was taken was read at the Fifty-Ninth Annual Meeting of the American Neurological Association, Washington, D.C., May 9, 1933. At this same meeting and immediately following this paper was one read by Winkelman and Eckel.[7] This second paper dealt with the histopathology of the brain during the early stages; that is, a histological study of the brain in

persons who died soon after injury. It made the following admissions prior to the presentations of case histories—that there is an increase of patients in head-injury cases seeking compensation and redress; that the duration of unconsciousness is usually in direct proportion to the severity of the brain injury; that symptoms following a head trauma are usually uniform even in the absence of legal complications; that the brain of a patient who dies after a severe head injury reveals a remarkably uniform finding of subarachnoid hemorrhage with maceration of the brain on the under surfaces of the frontal and temporal lobes; and that it was their purpose to attempt to determine all the minute changes in the brain after trauma and to correlate the clinical and histological features. After presenting seven cases, one of their conclusions was:

Sufficient evidence has been found in the brains of patients with severe head trauma, especially those in whom unconsciousness had been prolonged, to convince us that most of these persons have organic changes in the brain, the result of trauma. These changes explain the post-traumatic symptom complex usually characterized as "traumatic neurosis."

Organic Changes Not Always Present.—Believing that this conclusion expressed by Winkelman and Eckel regarding the relationship of trauma to neurosis is not justified, I wish to quote the remarks of Bernard Sachs,[8] who criticized the paper at the time it was read as follows:

I should like to say in connection with the whole subject, so as to avoid one danger that neurologists seem to be steering for, that the cases that have been presented are instances of rather serious head injury and I do not think there has been any doubt that organic lesions have followed such injuries.

However, before one absolutely dismisses the idea of anything like the picture of traumatic neurosis, an important question and one that has great practical value, particularly in medicolegal experience, is what happens in the far greater number of cases of rather slight injuries to the head and to the spine, in which there is a remarkable similarity in the claims made and in the symptoms that are supposed to follow. Whether or not there is distinct organic change in those cases is still open to discussion. At all events, I think it should be allowed that there may be something in the form of a general neurosis or of neurotic symptoms not necessarily based on actual organic changes which frequently follows mild injury to the brain or to the spinal cord. The mistake that should be avoided is the insistence that in all cases of head injury there are the changes that have been

shown here. It has been valuable to see the evidence of the changes that occur, particularly in such cases as Dr. Winkelman presented, but one should not jump at the illogical conclusion that there are organic changes in all cases of skull trauma. I am inclined to feel that evidence is lacking that there are organic changes following even the milder forms of skull trauma.

Review of Literature.—The following "brevities" are concise extractions of opinion found in the literature:

Hassin[9]: That the majority of neuroses once considered functional are actually pathological and microscopic changes in the nervous tissue can be demonstrated.

Winkelman[10]: In studying a series of cases histologically was "surprised to find changes in the brain that were indisputable. In many cases of these, hysteria had been considered as a diagnosis."

Riggenbach[11]: Cranial trauma may produce not only a neurosis but even a psychosis.

Charcot[11]: Could see no fundamental difference between the neuroses that followed trauma and those of ordinary life.

Page[11]: Suspense of litigation, want of occupation, and anxious reflection influence recovery.

Rigler[11]: Increase in the incidence of traumatic neurosis since the introduction of insurance.

Lottig[11]: Attempted a differentiation between an illness caused by the immediate factors of injury and that arising because an existing neurotic tendency preceded the injury.

Fetterman[11]: Suggested a distinction between those nervous before, and those nervous after, an accident (pre- and post-traumatic groups).

Volitional Factor versus Organic Basis.—The review of the literature just presented reveals that among authorities there is general disagreement concerning two phases of the so-called "traumatic neurosis syndrome," namely the presence or absence of psychogenic and organic factors, and the estimation of the extent of injury. From the preceding discussion it will be noted that there are those who feel that traumatic neurosis arises entirely from a functional or volitional factor and that this volitional factor, possibly minor in character at the beginning, progresses into a strong purposeful factor with or without manifestations of hysteria. Opposing this viewpoint are those who contend that there is always an organic basis for the neurosis following trauma, even though the extent of the trauma may be minimal. Here again, "the extent of injury" comprises a phase of the syndrome about which there is dispute.

Effect of Concussion on Consciousness.—Some hold that trauma of a degree sufficient to produce significant intracranial changes must result in unconsciousness, that the possibility of concussion without unconsciousness is not to be considered. Others hold that concussion can be present without unconsciousness (a minority viewpoint). They feel that a mental dysfunction without unconsciousness may follow a head blow, and cite instances of prizefighters who, although they continued to fight, were so dazed by a blow that the events of the fight and those following it were not remembered.[12] Finally, there are those who maintain that neither a history of unconsciousness nor of severe trauma is necessary for varying degrees of disability to be present following a head injury. Unfortunately, there seems to be no common ground or unified understanding of the definition of concussion. The majority of commentators have discarded a belief in the molecular disintegration or anatomical change theory, but lend support to the theory that concussion occurs following the disturbance of the cerebral vasomotor mechanism. Regarding this, Munro[13] says:

> The primary and simplest form of craniocerebral injury is true concussion. The term has been widely used to cover all forms of brain injury regardless of the pathology present. It should be restricted solely to those cases in whom a blow on the head has produced some degree of unconsciousness but who on recovery present no residual symptoms or signs whatsoever. Upon it as a foundation are built the complicated and more serious succeeding brain injuries. It is probable that the primary unconsciousness practically always associated with craniocerebral injuries is due to this concussion.

Wechsler's concept of concussion has been stated in a previous paragraph. Bennett,[14] in his classification of concussion includes a mild form "in which no loss of consciousness occurs at all." Strauss[6] prefers to think of concussion clinically as "a series of events resulting from a blow to the head severe enough to cause a disruption of intracranial equilibrium."

Malingering. *Definition.*—Before summarizing and attempting to clarify the situation so far as the viewpoint of the writer is concerned, malingering should be defined. A person becomes a malingerer only when he consciously and purposely, in order to deceive, to evade responsibility, or to derive gain, feigns illness and voluntarily tries to reproduce signs and symptoms which he really does not have, or extravagantly exaggerates minor ones which he has.[1] Invariably the claims made by a malingerer follow a minor injury and one in which the initial symptoms have been slight. The malingerer is to be dis-

tinguished from the neurotic, for in the latter the symptoms are unconsciously manifested. The experienced clinician usually has no difficulty in spotting true malingering, although on occasion a cunning person will perplex one. In the specialized fields, such as ophthalmology or otology, tests will establish the allegation to be true or false. These are too detailed for description here.

Difference between Malingerer and Neurotic.—The following is a broad classification of the malingerer as compared to the neurotic:

TABLE 25—DIFFERENTIATION OF THE MALINGERER FROM THE NEUROTIC

MALINGERER	NEUROTIC
Attitude towards Work and Play	
Claims inability to work, but enjoys ball games, movies, cards, or participates in light games, such as billiards	Cannot get interested in games. Evidences no desire to be a participant or spectator
Attitude towards Treatment	
Unfaithful and irregular in reporting for treatment or in taking medication. Proffer of an operation is usually refused. Annoyed by reexaminations or examinations by "specialists"	Faithful to all details of treatment and medication. Willing to submit to an operation. Willingly submits to frequent and repeated examinations by many doctors
Attitude as Revealed by Past History and Background	
Irresponsible, occupational migrant; has not held responsible positions; not a community worker or asset	Responsible, absentee record good; long duration of employment for one or more employers; active in community or civic enterprises
Attitude as Revealed by Investigation	
When away from doctors or observers uses parts allegedly disabled (as revealed by movies, detectives, etc.)	Condition of disability constant
Attitude towards Rehabilitation	
Refuses work offered which could be done without involving part of body allegedly disabled. Unwilling to accept state rehabilitation schooling to fit him for a new trade	Accepts work of a limited nature which he may be able to do. Distinction here not readily discernible, since many neurotics feel they cannot function in any capacity

Nomenclature.—That there is need of a better descriptive title for the traumatic neurosis syndrome is evident. The one used as a title for this chapter "Neurosis Associated with Trauma" was borrowed from Fetterman,[3] who remarked that such a name suggests at once the distinct types or forms of the disorder and does not prejudice the relationship of cause and effect which is implied by the terms "compensation neurosis," "purpose neurosis," "traumatic neurosis," and the like.

Procedure in Classification.—With this definition as a starting-point, the following procedure should be employed to classify any given patient:

History.—The history in these cases is of great importance. Obtaining a complete story calls for tact, ability to bring out the whole

story, to create an atmosphere which the patient feels is congenial and an attitude which the patient senses is not prejudiced. Repeated history-taking is invariably necessary in order that a picture of the entire background of this patient's life be obtained—conflicts of youth, his past and present social and economic status, congenial or conflicting factors in his present occupation, evidence of being a responsible or irresponsible type, his previous compensation or litigation experience—all these and many more need to be weighed. Only after repeated observations and a complete compilation of facts, together with the physical and laboratory findings, should a final classification be made.

Physical Examination.—This includes not only the general and neurological examination, but frequently a special examination by an ophthalmologist, otologist, or other specialist. It is not infrequent that symptoms considered to be of nervous-system origin, and arising out of an injury, may be found to have their cause in an unrelated systemic disease. For example, a man claimed headache, vertigo, lack of concentration, and loss of weight two months after a trivial head injury. One physician contended the man was a malingerer; another that his symptoms were due to his injury. Neither of them evaluated, or at least mentioned, the findings of a blood pressure of 210 systolic, 120 diastolic, generalized arterial sclerosis, sugar in the urine, and a blood sugar of 280 mg. per 100 cc.

Laboratory Investigation.—This should include routine measures, such as urine and blood studies, *x*-ray examination, spinal tap, and (when strongly indicated) encephalograms.

Psychiatric Examination.—Undoubtedly this should be more frequently employed. When the physician feels that organic causes do not account for the symptoms claimed and if he feels he is not successful in getting an adequate picture of the patient's mental background, the psychiatrist should be called into the case.

Basis for Diagnosis.—In considering the neurosis associated with trauma we are not concerned with the patient in whom the history of severe injury is accompanied by compatible signs and symptoms, but rather with the patient alleging a disability following a minor or negligible injury. The importance of an adequate history, a complete physical examination, and exhaustive laboratory study has been emphasized. But after all that is done, many of these cases will still remain a puzzle. It appears to me that in a case of neurosis associated with trauma, ultimate opinion is influenced by three situations:

1. *Character and Background of Claimant.*—The following characteristics were mentioned under "History," but bear repeating:

(*a*) *Stability, Solidity.*—Has worked for the same employer for

many years; rarely been absent from work because of illness; never been involved in litigation or compensation claims; is not an agitator in labor activities; obviously is not of a nervous disposition.

(*b*) *Instability.*—Is constantly changing jobs and is dissatisfied with his present employment; has had previous experience in litigation or compensation; is an agitator and feels that labor is unjustly dealt with; is a type that obviously has always been nervous; his economic status is such that compensation, although a reduced income, is adequate for his needs.

To illustrate the difference in character between two persons having the same type of injury and in whom the age, build, and constitution were comparable, a fracture of the right transverse process of the first lumbar vertebra occurred in both a longshoreman and a welder. The longshoreman took off from work only that time required for an examination. The welder, employed at much lighter work, had a temporary disability of eight weeks. The welder, in this instance, is not cited as being a malingerer, but a study of his character revealed his attitude towards work.

It has been previously stated in this book that the average workman is not a malingerer. This is most certainly true, but the *incidence of malingering* is not so rare as some writers state. Case after case can be extracted from our files wherein the claimant has faked the same injury in several states to gain compensation. A case in point is here recited. After receiving a minor head injury, an oil-field worker alleged that he had headache, got very dizzy when he had to bend over or walk at high levels. Following an award for total permanent disability, he and his wife retired to a little home in the Mariposa Woods, where they had a garden and he could hunt. More than a year later, without the man's knowledge, movies revealed him to be doing all the acts he alleged he couldn't do. He admitted deceit, but stated he liked that kind of life. In this instance compensation had created a situation desirable to the injured one. Space does not permit the recounting of numerous other cases. Truly, the incidence is not high, but certainly not rare.

2. *Character of Circumstance in Which Injury Occurred.*—In view of subsequent statements, consider the following conditions: (*a*) A man sustains a head injury in a home which he owns. He has no one to blame but himself, and he has no insurance; (*b*) a man, driving his own car, fails to traverse a bend in the road because of speeding, strikes a tree, and receives an injury but has no insurance of any type; (*c*) the same conditions as case "*b*" except that the man has insurance to cover his accident; and (*d*) a man falls from a platform, receiving an injury covered by the Workmen's Compensation Act.

It is possible that all four men in the above-mentioned circumstances would evidence no subsequent malingering or neurosis. But if either of these two conditions arose, would it be likely to occur in the first or the second two instances? The answer is obvious. It is impossible to ignore the factor of possible financial reward for an injury suffered. To believe or insist that the element of compensation or award from a civil court does not enter into the allegation of disability is to ignore the increased incidence of such claims. The validity of the claim should not be ignored because the element of possible award exists, but the character of the circumstances in which injury occurred must be evaluated.

3. *Character of Experience of Physician Offering Opinion.*—Medicine has always placed major emphasis upon the importance of experience. In a case of questionable yaws, an opinion from one such as myself should be held of no value when compared to an opinion from a medical missionary who has seen large numbers of these cases. A neuropsychiatrist may well define the psychogenic components which comprise a neurosis, but in any given case is his experience such that his definition considers the changing complex of living? Does his definition consider the social philosophy newly adopted by a class of people previously denied justifiable rights? The definition of a neurosis offered years ago remains largely unaltered, but the people to whom it is on occasion applied have changed markedly.

Intimate association with any group of people teaches one to understand their characteristics, tendencies, attributes, and philosophy. This experience constitutes an inestimable factor in the country doctor's "acumen." He may not have the specialized training of his urban confrère, but he knows his people. This is likewise true of the physician who is associated with industry. If he believes that the claims presented today show an increased tendency towards a wilful or volitional purpose of obtaining more than is justly due, it is not evidence that he is prejudiced. It is evidence that he is acquainted with the attitude of the age.

In substantiation of the writer's viewpoint, one fact must not be overlooked. Most of the expressions of opinions reviewed in this chapter were made prior to the date when the complete legislation of acts designed to aid the victims of this depression became effective or reached the height of their effectiveness. One wonders if there would not now be a revision of opinion from those who disallowed the influence of financial award in the neurotic. Would not the trained sociologist agree that social legislation—such as old-age pension, unemployment insurance, the eight-hour day, and the five-day week—has influenced the attitude towards work? The movies, the radio, and the

automobile have encouraged newer concepts of living. Who of us that have sons or daughters around the age of fifteen years does not deprecate their attitude towards work? Would anyone say that their attitude towards work is not different from that of their parents or grandparents?

Finally, the character of one's experience varies not only as to the degree of contact but also as to the state of society one deals with. How often would malingering or neurosis be noted among the stolid, rugged men inhabiting the outposts of civilization. Would Dr. Grenfell, of Labrador, have noted the frequency of neurosis that a doctor practicing in Detroit or Pittsburgh, or a similar industrial center, experiences? Manifestly, no; because in the first instance the very life of the individual is dependent upon his own efforts. He cannot fall back upon any benevolence created for his misfortune.

The argument offered in the summary of this chapter is that the physician who considers the element of compensation as a factor in his estimation of the cause of neurosis or malingering should not be condemned as prejudiced. He is only considering all the factors which comprise the entity and is just as sincere as the naval doctor who considers lues in an old sea dog.

REFERENCES

1. Wechsler, I. S.: "Trauma and the Nervous System," *J.A.M.A., 104:* (Feb. 16), 1935.
2. Davidson, H. A.: "Neurosis and Malingering," *Am. J. M. Juris., 2:* (Feb.), 1939.
3. Fetterman, J. L.: "The Neurosis Associated with Trauma," *Indust. Med., 4:* (Jan.), 1935.
4. Reichardt, quoted by Fetterman, *ibid.*
5. Lewy, E.: "Contribution to the Problem of Compensation Neurosis," *Mellinger Clinic Bulletin, 4:* (May), 1940.
6. Strauss, I., and Savitsky, N.: "Head Injury—Neurologic and Psychiatric Aspects," *Arch. Neurol. and Psychiat., 31:* (May), 1934.
7. Winkelman, N. W., and Eckel, J. L.: "Brain Trauma, Histology during Early Stages," *Arch. Neurol. and Psychiat., 31:* (May), 1934.
8. Sachs, B.: Remark made in discussion of Winkelman and Eckel's paper, *op. cit.*
9. Hassim, G. B.: Remarks made in the discussion of Wechsler's paper, *op. cit.*
10. Winkelman, N. W.: "Brain Trauma, Histology during Early Stages," *Arch. Neurol. and Psychiat., 31:* (May), 1934.
11. Fetterman, J. L.: *Op. cit.*
12. Tunney, Gene: "A Man Must Fight," *Collier's*, March 26, 1932.
13. Munro, D.: "The Modern Treatment of Craniocerebral Injuries with special reference to the Maximum Permissible Mortality and Morbidity," *New England J. Med., 213:* (Nov. 7), 1935.
14. Bennett, W.: "Some Milder Forms of Concussion of the Brain," in Allbutt, T. S., and Rolleston, H. D.: *A System of Medicine*, by many writers. The MacMillan Co., London, 1910, Vol. 8, p. 231.

CHAPTER XXXIX

THE PREEMPLOYMENT EXAMINATION

Workers' Objections to Examination.—On first thought it would seem that there could be no possible objection on the part of the workman to the preemployment or routine periodic physical examination in industry. Actually, however, there exists a great deal of opposition. Workmen feel that the physician is an agent of the employer and as such will convey to him information ordinarily considered confidential. They fear that some minor physical defect may be detected and because of it, employment be denied. Labor unions have objected especially to the periodic examination of men under hire, claiming that this system is used by employers to get rid of men who the employer feels are undesirable or agitators.

Objections to a physical examination are not without some valid reasons. The preemployment examination may be unjust to a workman in certain instances, especially in those states which are extremely liberal in the granting of compensation for aggravation of a preexisting condition. The potential hernia serves to illustrate this point. When it is detected and reported, the applicant is usually turned down for employment, even though the position sought is one not apt to call for excessive manual labor. But the employer knows that if the potential condition becomes an actual hernia, he will be held responsible by the accident commission. The applicant often is willing to sign a statement relieving the employer of any responsibility, but this is not acceptable to commissions in the majority of states. The unfortunate victim of such a system is obviously the man who presents a minor physical defect and yet is able to work. Concerning the periodic physical examination, the objection raised is that if a physical defect is discovered, the workman will be discharged. Some industries honestly attempt to care for such an employee by changing his position and placing him in a department where effort is reduced. However, this commendable act is often blocked by the labor unions. In centers where the labor unions are strong, they insist in upholding the right of seniority and insist that if the man is placed in a different department, he goes in as a new man, losing his seniority. The answer to this dilemma is certainly up to the "National Labor Relations Board," or a similar governmental agency.

Capacity to Work of Men of Forty.—Allied closely with the above two phases of the physical examination is the problem of the aging. Gerontology is now a unit of the National Health Institute. It is not a new problem, for Cicero wrote *De Senectute* in 45 or 44 B.C., but the period of "old age" has been extended in both directions. Thanks to medical science, the present mean life expectancy at birth has been increased to sixty-one years. Yet the prevailing opinion in industry is that the age when a man becomes old has been lowered. Thus a man in his forties today has difficulty obtaining a job because he is looked upon as being old. Men in the late forties and fifties are being replaced by younger men, thus being forced to sit idly by and increase the aging process. To any physician this is a great mistake. Sir Humphrey Rolleston once stated, "There is more danger to the individual rusting out than wearing out." Except for certain occupations, not man's age but rather his capacity to work should determine his status. It is a pitiful thing to see the anguish on a man's face who wants to work, but who has been replaced by a younger man. In the vast socialization program being conducted by our government, there should be some system evolved which will find employment for these older men—not just a pension.

Extent of Examination.—A preemployment examination should be comprehensive, and records should be of permanent value. When a defect is noted, the applicant should be informed, and he should be referred to his family physician. The latter should be permitted to have access to the findings noted by *x*-ray or laboratory examination. The plant physician has a great opportunity to advance preventive medicine by cooperating with the community physician in this respect. In some plants, a copy of the examination is given to the applicant so that he will understand why he has been rejected and so that he will attempt to remedy his defect. Routine *x*-ray examination of the chest and tests for lues are advisable.

Employee's Permission to Report Findings.—The physician who makes preemployment examinations and periodic reexaminations should be warned that this practice presents a legal problem. Roemer[1] points out that by reporting to an employer the findings of a physical examination without first securing the permission of the employee, a doctor violates the patient's trust and in many instances the civil law of the state relating to confidential communications. According to Roemer, it is not safe to assume that when a workman submits to an examination at the request of his employer, he gives tacit permission to the physician to communicate the findings to the employer. While the risk of a suit for slander is certainly not great, it is advisable for a physician to protect himself by obtaining the written

consent of the patient to make the examination and to disclose the findings to the employer or organization requesting the examination. This consent should

(*a*) Grant permission to the physician to disclose the findings to the employer
(*b*) Waive the patient's rights or other person's in the provisions of the law relating to confidential communications
(*c*) Be signed by the patient
(*d*) Dated as of the day of the examination

Routine and Classification.—The best advice the author can give is that a comprehensive routine and a satisfactory method of classification be established. Of all those that have been suggested, the method employed by Selby,[2] at General Motors, appears best. The following is the substance of his discussion:

Provision for Handicapped Workman.—In discussing this code, tuberculosis will be used as an example. Arrested tuberculosis falls in Class C. There are many occupations at which workmen with arrested tuberculosis may be employed, provided they have medical supervision, and periodic examinations sufficiently often to guarantee them against aggravation by employment. Obviously, they cannot be employed on dusty work, regardless of the type of dust.

Active tuberculosis (Class D) renders workmen unemployable. They are possible sources of the disease to others, and they need treatment, probably in a sanatorium.

There are many men with defects requiring medical attention who are employable. Management is content to leave the responsibility of that problem with the plant physician. He always gives the benefit of the doubt to the workman, and this is exactly as it should be. With him, the only question is whether or not employment will be harmful to the handicapped workmen. This should be the consideration with reference to the placement of this Class C group.

Of course, on the reexaminations, Class C men should not be discharged. The reason for the reexamination is to assist them to maintain their health while employed, to give them a longer period of gainful occupation. The details of the placement cannot be specified. Every doctor has to work them out for himself. He cannot do it unless he knows his plant.

The doctor with imagination and inquisitiveness, who goes out in the plant and familiarizes himself with every possible occupation that may have influence on health, avails himself of the service of the industrial hygienist and works with the engineer for the control of the occupational-disease exposures, examines employees thoroughly be-

fore placement, examines them after employment often enough to guarantee them against diseases that might result out of occupation, and institutes a complete health-maintenance program, in cooperation with the local general practitioners of medicine and health authorities, is determining the place of physical examination in industry and effectively placing employees. He is the symbol of modern industrial medicine.

Examination Record Code.—Findings are recorded by minus (—) or plus (+) signs to show absence or presence of defects, except where figures are called for, as with age, height, weight, pulse and blood pressure, or where special comments are needed, as with *x*-ray, etc. If description is required, an identifying numeral (1), (2), (3), etc., is placed after the plus sign in this manner (1), and details are recorded under "Remarks," with the identifying numeral preceding the description. An applicant who has no defects (minus signs throughout) is given a placement rating of Class A. Extent and degree of defects are recorded by one (+), two (++), or three (+++) plus signs, and the placement rating is Class B, C, or D.

PLACEMENT CODE

Class A (—).....Physically fit for any work
Class B (+).....Defect negligible or correctable; otherwise fit for any work
Class C (++).....Defect limits fitness for work and may or may not need medical attention, but does require medical supervision
Class D (+++)....Defect requires medical attention and disqualifies for employment

Classes A and B may be employed. Classes C and D may not be employed while qualifying conditions exist. If already employed, these classes require handling subject to instruction of the physician in conformity with restrictions set forth by him.

TABLE 26.—PHYSICAL EXAMINATION RECORD

Name: *Address:* *Nationality:*

| | | |

32. Urinalysis _____

33. Blood Analysis _____

34. Fluoroscope _____

35. x-Ray _____

Remarks: _____

_____

_____

_____

Illustrative Classification.—No attempt is made to set up standards for exclusion, because medical judgment must be the basis of all decisions. The following classification is merely illustrative:

GROUP I

Epidemic, Endemic and Infectious Diseases:

Acute infectious diseases, including measles, scarlet fever, erysipelas, active tuberculosis (any organ)	Class D
Arrested tuberculosis	Class C
Venereal diseases, acute	Class D
Chronic venereal diseases under treatment	Class B or C

GROUP II

General Diseases:

Cancer—all forms	Class D
Cancer—postoperative cure	Class C
Benign tumors, disabling	Class D
Others	Class B or C
Acute rheumatic fever, acute arthritis	Class D
Chronic arthritis, marked deformity	Class D
Other forms	Class B or C
Diabetes mellitus	Class D
Under diet and control	Class C
Pernicious anemia	Class D
Under diet and control	Class C
Secondary anemia, severe	Class D
Mild	Class B or C
Leukemias	Class D
Hodgkin's disease	Class D
Chronic poisoning, lead, etc.	Class D
Drug addiction	Class D

GROUP III

Diseases of the Nervous System:

General diseases such as encephalitis, meningitis, sclerosis, cerebral hemorrhage or thrombosis, epilepsy, chorea, convulsions, psychosis, imbecility, neurasthenia (severe), brain tumor	Class D
Neurasthenia (mild), neuritis, etc.	Class B or C

Diseases of the Eye:

Vision 20/100 or better, both eyes, without correction	Class B
One eye blind or defective 20/200 or less with or without glasses	Class C
Both eyes 20/200 or less with or without glasses	Class D
Trachoma, glaucoma, etc.	Class D

Diseases of the Ear:

Chronic suppurative otitis media, one ear	Class C
Both ears	Class D
Hearing normal tones of voice	Class A
Otherwise	Class B
Disease of the mastoid, not resolved	Class D

GROUP IV

Diseases of the Circulatory System:

Active pericarditis, endocarditis, myocarditis, angina pectoris	Class D
Other diseases of the heart, if well compensated	Class C
Decompensation or history of severe decompensation	Class D

Diseases of the Arteries:

Aneurysm, severe embolism or thrombosis, varicose veins with ulcer, hemorrhoids, severe	Class D
Varicose veins, moderate	Class C
Varicocele	Class B
Hemorrhoids, moderate	Class B or C

GROUP V

Diseases of the Respiratory System:

Sinusitis, acute	Class D
Chronic	Class C
Bronchitis, acute	Class D
Chronic	Class C or D
Asthma, mild	Class B or C
Severe	Class D
Silicosis, first stage	Class B
Second stage	Class C
Third stage	Class D
Pneumonia, pleurisy, empyema, gangrene	Class D

GROUP VI

Diseases of the Digestive System:

Acute tonsillitis, quinsy, acute peptic ulcer, acute enteritis, pancreatitis, acute appendicitis, acute yellow atrophy, acute cholecystitis	Class D
Chronic peptic ulcer, chronic cholecystitis, chronic appendicitis	Class B or C
Hernia, unsupported	Class D
Supported	Class C

GROUP VII

Diseases of the Genito-urinary System:

Acute nephritis, acute cystitis, acute pyelitis, acute prostatitis, acute salpingitis	Class D
Chronic infections as above if under treatment	Class C
Pregnancy	Class D
Pregnancy if self-support necessary, first four months only	Class C

GROUP VIII

Diseases of the Skin:

Acute abscesses, carbuncles, poison ivy, extensive dermatitis, severe eczema, scabies	Class D

GROUP IX

Diseases of the Bone and Organs of Locomotion:

Osteomyelitis, acute and chronic	Class D
Amputations, fingers, limited	Class C
Many fingers or other extremities	Class C or D
Scoliosis, kyphosis, lordosis, ankylosis, myalgia	Class B, C, or D

REFERENCES

1. Roemer, M. L.: "Sued for Slander," *Med. Economics, 18:* 67, 1941.
2. Selby, C. D.: "The Place of Physical Examinations in Industry," read before the Occupational Disease Symposium, Northwestern University Medical School, Sept. 26, 1938.

APPENDIX

Table of Toxic Thresholds of Common Industrial Substances

THE following list of suggested maximum permissible concentrations of potentially harmful industrial dusts, gases, vapors, and fumes is recommended by the Industrial Accident Commission of California. With but few exceptions it agrees with the one compiled by Bowditch, Drinker, et al.[1]

Substance	*Limit*
	Million Particles per Cubic Foot
Dusts:	
Asbestos	5 (0.5 to 10.0 μ)
Cement	15
Organic	50 (0.5 to 5.0 μ)
Pottery	4
*Silica (25 to 35% SiO_2)	10 (0.5 to 5.0 μ)
Silica (over 75% SiO_2)	5 (0.5 to 5.0 μ)
Slate	15
Talc	15
Gases and Vapors:	Parts per Million
Acetone	200
Ammonia	100
Amyl acetate	400
Aniline	5
Arsine	1
Benzol	100
Butanol	100
Butyl acetate	400
Carbon disulfide	15
Carbon dioxide	5,550
Carbon monoxide	100
Carbon tetrachloride	100
Chlorine	1
Chloroform	100
Dichlorbenzene	75
Dichlorethyl ether	15
Ether (ethyl)	400
Ethyl alcohol	250
Ethyl bromide	1,700
Ethyl chloride	20,000
Ethylene dichloride	100
Formaldehyde	20
Gasoline	1,000
Hydrogen cyanide	20
Hydrochloric acid	10

* Dust count (million particles per cubic foot of air), multiplied by percentage of free silica (silicon dioxide), should be less than 5.

Gases and Vapors:	Parts per Million
Hydrogen fluoride	3
Hydrogen sulfide	20
Methanol	100
Methyl bromide	50
Methyl chloride	500
Monochlorbenzene	75
Naphtha	5,000
Nitrobenzene	1
Nitrogen oxides	10
Ozone	1
Phosgene	1
Phosphine	2
Phosphorus trichloride	0.7
Sulfur dioxide	10
Tetrachlorethane	10
Tetrachlorethylene	200
Toluol	100
Trichlorethylene	200
Turpentine	700
Xylol and coal-tar naphtha	100

Metallic Dusts and Fumes:	Milligrams per Cubic Meter
Cadmium	0.1
Chromic acid	0.1
Lead	0.15
Manganese	30
Mercury	0.1 to 0.2
Zinc oxide	15
Chlorodiphenyl	1.0
Pentachlornaphthalene	0.5
Trichlornaphthalene	5.0

REFERENCE

1. Bowditch, M., Drinker, C. K., Drinker, P., Haggard, H. H., and Hamilton, Alice: "Code for Safe Concentrations of Certain Common Toxic Substances Used in Industry," *J. Indust. Hyg. and Toxicol.*, *22* (June), 1940.

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