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POISONING

A GUIDE TO CLINICAL DIAGNOSIS
AND TREATMENT

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POISONING

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Dedicated to the many physicians
whose observations and reports have
enriched our knowledge of poisonings.



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PREFACE

While death is the most dramatic manifestation of poisoning, subacute and chronic poisonings are far more common and often offer serious problems for diagnosis, which in turn determines the treatment of the patient and the prognosis of his condition.

The incidence of poisoning, both acute and chronic, is constantly increasing as new chemicals for the most diversified purposes are placed on the market and are used in homes and industry. There are also many plant poisons which are of concern in rural districts, and finally there are many drugs on the market which may produce side reactions of varying severity. There are literally hundreds of substances in use or to which one may be exposed today which may give rise to poisoning. Some of these toxic syndromes closely simulate infectious or functional diseases, and it is therefore of paramount importance that they be recognized.

It is the purpose of this book to organize today's knowledge of the subject in a fashion which will most readily aid the general practitioner and the internist in diagnosis and treatment of poisoning. It is earnestly hoped that the material has been so arranged that it will save time for the busy physician in tracking down the toxic agent and instituting effective treatment when poisoning is suspected.

The book is divided into four parts. The first discusses the classification of poisons, the medico-legal aspects and responsibilities of the physician and the emergency measures and equipment necessary for the treatment of poisoning.

Part two takes up the clinical diagnosis of poisoning. Here it is shown how diagnosis is based on the patient's history and the structural and functional pathology observed in the course of a medical examination. The signs and symptoms are classified by body systems for ease of reference, and are discussed one by one as they present themselves in the course of a clinical examination. Under each is an alphabetical list of toxicants which may be responsible for such a change. The physician will first turn to this section when confronted with suspected poisoning, find-

ing under the symptoms he has observed a list of toxic substances which may give rise to them.

In this section of the book there are also chapters on blood and urine changes in poisoning and on laboratory tests which are within the compass of the private practitioner or small hospital facilities.

In part three each step in the management of poisoning is taken up separately. Emphasis is placed on the technics available for removal of toxic agents and on the symptomatic treatment which must often be employed at the same time or even preceding removal of the poison.

The final and largest section of the book is an alphabetical listing of 461 poisons. Under each poison is a short synopsis of the clinical picture and the treatment required. This arrangement is made with an eye to convenient reference by the practitioner. The section is based on the analysis of several thousand case reports.

The book aims to supply in a single source the information needed by the clinician for the accurate detection and successful treatment of poisoning. Therefore, it does not cover the mechanism of action of various poisons, nor does it cover the animal experimental studies which were undertaken to study those mechanisms. For more detailed study of toxic agents the reader is referred to the various review articles mentioned in the text and to the larger text books of pharmacology.

If this book helps the harried practitioner to detect poisonings more quickly and to take effective measures it will have achieved the author's aim.

The author is indebted to Mrs. Florence B. Hall and Mr. A. F. Collins and other members of the staff of the library of the National Institutes of Health for their untiring help in furnishing the many journals and texts required for the preparation of this book. Finally thanks should be given to the publisher for encouraging the writing of this book.

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PART ONE

INTRODUCTION

1. Classification and Diagnosis of Poisoning

The correct diagnosis of poisonings or of toxic conditions has become increasingly more important. It is not only essential for the prompt and effective treatment of the patient, but in addition it may have other far-reaching consequences. It may be of forensic importance and lead to the detection of a crime, or it may be instrumental in improving inadequate sanitary conditions and thus prevent later cases of a similar nature. The prompt recognition of a toxic condition which is due to the occupational environment may be of considerable economic importance for the patient in that in these cases the treatment of his illness and the resulting loss of gainful activity may be paid for through compensation by state and private insurance agencies.

The number of toxic substances to which modern man may be exposed is constantly increasing. The importance of some poisons as causative agents for suicide and for accidental poisonings has varied, especially as new chemicals have become readily available, while others like carbon monoxide and certain plant poisons have caused havoc for thousands of years. At one time poisonous antiseptics, such as phenol and lysol, were the prevalent cause of poisonings. With the introduction of essence of vinegar for domestic use this has caused numerous fatal and nonfatal accidents. With the advent of illuminating gas and more recently with the widespread use of the automobile, carbon monoxide poisoning has become much more prevalent than in the past. The unrestricted sale of sedatives and soporifics such as the barbiturates has caused an appalling number of fatalities from the intentional or accidental ingestion of these drugs. Similarly industrial poisons have varied considerably in their importance as causative agents. Sometimes their dangers are recognized and controlled in certain trades and operations, but they may be used in new processes under different conditions where their danger is not so apparent or even not suspected. The same holds true for toxic substances sold for domestic use such as solvents, spot removers, insecticides, de-

odorants, and antiseptics. It is therefore apparent that the physician may be confronted with a great variety of toxic agents.

Very often the medical examiner is unaware that a poisoning is the underlying cause for the condition of his patient and even more frequently he is not familiar with the toxic agent and its toxicologic properties. The clinical picture of parkinsonism may be the outcome of chronic manganese poisoning or of exposure of carbon disulfide. A Korsakow syndrome may be due to alcoholism, but may also be the consequence of carbon monoxide poisoning. Hepatitis may be of infective character or it may result from exposure to hepatotoxic agents such as carbon tetrachloride. On the other hand, the physician should not be misled in his diagnosis by impressive and widely publicized reports on the toxicity of certain chemicals. For it is just as dangerous to interpret erroneously a clinical picture as the result of exposure to certain compounds. For instance DDT, which has been publicized as being responsible for a great variety of pathologic conditions, has in most instances actually no relation to the condition of the patient. It is obvious that such mistakes may have far-reaching consequences in that the true cause may require a different therapeutic approach.

It has been customary to classify toxic substances according to their most characteristic action, such as nerve, liver, or blood poisons. Such arrangement may have certain advantages in that it affiliates specific toxic substances with specific pathologic conditions. But on the other hand, there is no poison which affects one organ only. Sooner or later, primarily or secondarily, other organs will be affected and in some instances these injuries may predominate, so that a liver poison like carbon tetrachloride may cause an obvious injury of the kidney or may be responsible for disfunctioning of the heart muscle. In other instances the sequelae of acute exposure may refer to one organ, and those of continued exposure to another. The inhalation of high concentrations of benzene vapors results predominantly in disturbances of the central nervous system causing headache, inebriation, somnolence, loss of consciousness, and occasionally convulsions. Chronic exposure to the same agent will cause a more or less severe injury of the blood-forming organs and will lead to leukopenia and anemia. It is therefore obvious that such classification is not strictly justified.

Others have arranged toxic substances according to their chemical characteristics such as aliphatic and aromatic hydrocarbons, chlorinated hydrocarbons, alcohols, phenols, alkaloids, glucosides, etc. Such classification may enhance the understanding of the mechanism of their action and may allow the appraisal of the toxicity of chemicals with known chemical structure. However, it does not lend itself for the clinical

diagnosis of poisonings unless one is familiar with the toxic phenomena of a great many different toxic substances. It appears impossible that a physician can keep in mind all signs and symptoms of toxic substances.

For this reason in the following pages an attempt is made to give a pathologic physiology of poisonings by describing the observable pathologic changes in different organs and organ functions as produced by various poisons. A similar approach has been successfully used by H. H. Meyer and R. Gottlieb in their *Experimentelle Pharmakologie* and by L. Krehl in his *Pathologische Physiologie*. It is hoped that the comparison of abnormal findings as they present themselves in the course of a systematic clinical examination of a patient, and their classification by toxic causative agent or group of agents, will allow the elaboration of the toxicologic picture and thus lead to the diagnosis of the poisoning in question.

In accordance with this plan the actions of toxic agents are discussed with reference to their effect on organs and organ functions which are noticeable upon inspection, or which refer to the respiratory apparatus, the circulatory system, the gastrointestinal tract, its glandular structures, the urinary apparatus, glands of internal secretion, the central and peripheral nervous system, the sensory functions, and the blood-forming organs. This is followed by a discussion of changes in the blood chemistry and in the composition of the urine as produced by various poisons, and by a number of tests for the detection of toxic substances or their metabolites in blood and urine. In the last part a short synopsis of the clinical pictures of various poisonings is given, in order to summarize the various organ findings. In these descriptions the symptomatology is discussed somewhat summarily because the signs and symptoms produced by certain organ changes may vary in their frequency and intensity from case to case. It is felt that too detailed a description would run the danger of becoming a case history, and that a broader picture would emphasize certain definite characteristics whereas details may vary from case to case.

Similarly the management of poisonings is discussed from the point of view of organ functions rather than of individual poisons. In those instances where specific measures have been found of value they are discussed in the last part under the particular poison.

It is hoped that such an approach will facilitate the diagnosis of poisonings and allow an effective treatment even in those cases where the underlying cause has not been established.

2. Medico-Legal Responsibilities

As in any disease, it is the duty of a physician attending a case of poisoning to save the life of the patient, to make him comfortable, and to protect him against further injury.

Beyond this he has additional responsibilities. In certain cases the poisoning may indicate a danger to the general public, as in the contamination of drinking water or the pollution of the air by toxic gases or fumes. In such instances the public health authorities have to be notified at once so that steps may be taken to remedy the situation and to prevent further accidents. The occurrence of other poisonings, such as cadmium poisoning, may point to the appearance of cooking utensils and other food containers on the market which are plated with cadmium and it is obvious that the public should be warned against their use. The poisoning may result from the ingestion of food or food products contaminated with toxic materials used as preservatives, food dyes, and other agents added to make the food more appealing. Poisonings may result from the domestic use of insecticidal mixtures, dry cleaning agents, floor waxes, and similar materials which contain toxic substances. In all such cases it becomes the duty of the physician to notify the proper authorities so that appropriate action may be taken.

Similarly poisonings of an occupational character have to be reported in order to avoid accidents of similar nature and to secure for the patient the benefits provided by law.

The laws and regulations regarding the reporting of occupational poisonings and diseases vary with different states of the Union. The most recent compilation on this subject is that of V. M. Trasko (*Industrial Health Regulations: A compilation of state laws and regulations*. Division of Industrial Hygiene, Federal Security Agency, Public Health Service, Washington, D. C., July, 1950). According to this source industrial diseases including poisonings are reportable in 28 states as listed in Table 1. It will be noted that these have to be reported to different agencies in

different states. It is, therefore, advisable to familiarize oneself with the local state laws and regulations regarding this question.

TABLE 1. STATE AGENCIES TO WHICH OCCUPATIONAL POISONINGS SHOULD BE REPORTED

<i>State</i>	<i>Agency</i>	<i>Remarks</i>
Alabama	State board of health	
Arkansas	State health officer	
California	Division of labor statistics	within 7 days
Colorado	Local health officer	immediately
Connecticut	State department of health	within 48 hours
Georgia	State board of health	
Iowa	Department of health	
Kansas	State board of health	
Kentucky	Board of health	
Louisiana	Department of health	
Maine	Bureau of health	within 10 days
Maryland	State board of health	
Massachusetts	Labor department	
Michigan	State department of health	
Minnesota	Industrial commission	
Mississippi	State board of health, div. of industrial hygiene	
Missouri	State board of health	within 24 hours
Montana	State board of health, div. of industrial hygiene	
New Hampshire	State board of health	
New Jersey	State department of health, and department of labor	within 48 hours (lead)
New Mexico	Bureau of public health	
New York	Commissioner, dept. of labor	
Ohio	Commissioner of health and industrial commission	
Pennsylvania	Local health officer or State department of health	
Rhode Island	Dept. of public health	
South Carolina	State board of health	
Washington	Department of health	
Wisconsin	Industrial commission	

Some states publish lists of those occupational poisonings which have to be reported by the examining physician and may add to and change such lists as the necessity arises. Table 2 is compiled from the publication of W. Fowler (*The Reportable Diseases. Pub. Health Rep. 59:317, 1944*) and shows which occupational poisonings were reportable in certain states at that time. It should be pointed out that in some states the term "benzene poisoning" may also cover poisonings by nitro, hydroxy, and amino derivatives of benzene, and that in addition to poisonings from carbon tetrachloride, those by other organic halides or solvents are reportable in others.

TABLE 2. REPORTABLE OCCUPATIONAL POISONINGS

<i>Compound</i>	<i>Alabama</i>	<i>Colorado</i>	<i>Connecticut</i>	<i>Iowa</i>	<i>Kansas</i>	<i>Kentucky</i>	<i>Maine</i>	<i>Maryland</i>	<i>Minnesota</i>	<i>Missouri</i>	<i>New Hampshire</i>	<i>New Jersey</i>	<i>New Mexico</i>	<i>New York</i>	<i>Ohio</i>	<i>Pennsylvania</i>	<i>Rhode Island</i>	<i>South Carolina</i>	<i>Washington</i>	<i>Wisconsin</i>
Acids																				
Ammonia																				
Aniline		+	+	+	+	+	+	+	+	+	+			+	++	+	+	+++	+++	
Arsenic																				
Asbestos																				
Benzene		++		+		+	+			++			++					++	++	
Benzine (gasoline)		++		+		+				++			++					++	++	
Cadmium	+										+			+			+			+
Carbon disulfide		+			+	+				+			++		+	+		+	+	
Carbon dioxide	+	+		+	+	+			+	+			++		+	+		+	+	
Carbon monoxide		+		+		+							++		+	+		+	+	
Carbon tetrachloride				+		+			+				++					+	+	
Chlorine				+		+							+					+	+	
Chrome ulvers				+		+							+					+	+	
Chromic acid		+				+							+					+	+	
Cyanide				+									++					++	++	
Dinitrobenzene				+		+				+			++					++	++	
Formaldehyde	+				+								++					++	++	
Gasoline		+		+		+							+					+	+	
Hydrochloric acid						+							+					+	+	
Hydrocyanic acid						+							+					+	+	
Hydrofluoric acid						+							+					+	+	
Hydrogen sulfide						+							++					+	+	
Lead	+	+	+	+	+	+	+	+	+	+	+	+	++	+				+	+	+
Manganese		+		+		+				+			++					+	+	
Mercury	+	+	+	+	+	+	+	+	+				++	+				+	+	+

MEDICO-LEGAL RESPONSIBILITIES

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[illegible]

Other states, as, for instance, Massachusetts, may require every physician treating a patient whom he believes to be suffering from an ailment or disease contracted as a result of the nature, circumstances, or conditions of the patient's employment, to report such information. The department may issue a list of such diseases which shall be regularly reported on by physicians and may also add to and change such lists at any time.

Some states require the reporting of all cases of dermatitis from occupational exposure and others that of certain diseases connected with certain trades. So, for instance, Kentucky requires physicians to report petroleum workers' diseases, such as respiratory, gastrointestinal, nerve, and eye disorders. South Carolina requires reporting of poisonings from refrigerants such as methyl chloride, ethyl chloride, dichloroethylene, methyl bromide, ethyl bromide, methylene chloride, methylformate, dichlorodifluoromethane, dichloroethane, and sulfur dioxide.

Poisonings of suicidal or homicidal character and accidental poisonings should be reported to the county coroner or medical examiner in every jurisdiction of the United States.

It is obvious that these different responsibilities require considerable precaution and extreme thoroughness on the part of the physician in order to safeguard the interests of his patient and, in many instances, also of the community at large.

Pulmonary hemorrhages may be caused by severe cough as produced by irritant gases, especially in the presence of vascular injury, hypermia or stasis in the lungs. They may result in blood extravasations of different size or in typical hemorrhagic infarctions. Pulmonary hemorrhages may also be the sequelae of vascular injury, as produced by carbon monoxide and benzene, and of such changes of the blood and blood vessels which may result in thrombus formation in the pulmonary vessels as observed in poisonings from:

arsenic trioxide	gasoline
chlorate	quinidine
snake venom	

Gangrene of the lungs is usually of rapid onset and frequently the sequela of pneumonia and pulmonary infarctions. Although it may be difficult to differentiate between pulmonary gangrene and bronchiectasis, the former may be characterized by its characteristic foul odor. Pulmonary abscesses and gangrene have been observed in poisonings from poisons causing vascular injury, such as:

arsenicals	ergot
carbon monoxide	phosphorus

and from pulmonary irritants such as:

chlorine	mustard gas
hydrogen sulfide	nitrogen oxide
phosgene	

and after the aspiration of lye and other caustic agents.

To what extent **cancer of the lungs** may be caused by toxic agents is at present still questionable, but it appears beyond doubt that this can be produced by the inhalation of:

chromates	radioactive substances
nickel carbonyl	tar products

Granulomatous changes may be produced by inhalation of certain beryllium compounds.

Pneumoconiosis is a chronic fibrous reaction in the lungs caused by prolonged exposure to excessive quantities of inorganic dust. Many of these pneumoconioses can hardly be considered as diseases since they may not cause impairment of the physical efficiency of the patient. This holds true for such pneumoconiotic processes as produced by the inhalation of coal dust (*anthracosis*), chalk dust (*chalicosis*), and iron dust (*siderosis*).

However, the type of exposure associated with silica, which results in the picture of *silicosis*, is a chronic condition of the lungs, characterized by the development of fibrous nodules in the lung tissue. In advanced cases *silicosis* will cause dyspnea upon exertion, although it is not associated with orthopnea unless aggravated by attacks of asthma. Only occasionally it is associated with dry or little-productive cough in the morning. A predominant cough is usually indicative of an existing tuberculous infection for which *silicosis* is a predisposing factor. In the initial stage the condition is difficult to diagnose without the aid of x-ray. Advanced cases may be complicated by hypertrophy of the right heart and dilatation of the pulmonary cone. Other pneumoconioses are *asbestosis* and *cryolithosis*; their prognosis is somewhat better than that of *silicosis*.

phate, with bile thrombi and cholangitis in the liver, the late type shows marked alterations of the liver cells ranging from swelling to necrosis. Severe damage of the kidney is a rare complication; nephrosis with purpura may be due to hypersensitivity; and in exceptional cases there may be nephritis with fatty degeneration. Occasionally the administration of arsphenamine may be followed by visual disturbances. These may be characterized by neuroretinitis with changes of the vitreous humor which are preceded by blurred vision, floating spots, flashes of light before the eyes, and some time later by such objective signs as haziness of the vitreous humor, alterations of the papilla, and in rare cases by optic neuritis, iritis, and paralysis of the oculomotor apparatus. In very rare cases the intravenous injection of arsphenamine has resulted in gangrene of extremities. This is preceded by shooting pain, swelling, and impairment of sensation and motor functions. Hemorrhagic encephalitis, one of the rarest complications of arsphenamine therapy, is relatively frequently seen in women during pregnancy, and it sets in 48 hours after the second or the third injection. The first symptoms are headache, malaise, dizziness, generalized pain, and later restlessness, moderate leukocytosis, coma, and convulsions. The disease usually takes a very rapid course, ending fatally as early as 24 hours after manifestation of the first symptoms.

Treatment: This has to be symptomatic. The nitritoid reactions can be promptly alleviated by epinephrine or antihistaminic drugs. For the treatment of agranulocytosis, pentnucleotide (10 cc. every 6 hours intravenously), liver extract (2 cc. daily intramuscularly) and dimercapto-propanol, or BAL (1.5 cc. every 6 hours, for 48 hours and then 2 cc. daily for 6 doses intramuscularly) have been suggested. This medication should be combined with general supportive therapy such as the intravenous administration of 5 per cent dextrose to maintain the water balance, vitamin therapy, and oral hygiene. Arsphenamine encephalitis appears to respond promptly to therapy with BAL.

Beattie, J., and Marshall, J.: *Brit. M. J.* 1:547, 1944.

Fisher, S., Holley, H. L., and Fein, G.: *Arch. Dermat. & Syphil.* 55:57, 1947.

Shirbal, J. J., and Thurmen, F. M.: *Am. J. Syph. Neurol.* 19:17, 1935.

Hanger, F. M., and Gutman, A. B.: *Tr. As. Am. Physicians* 55:179, 1940.

Asbestos

Asbestos (magnesium-calcium-silicate) forms fine, slender, flaxy fibers and is used as an insulating material. The inhalation of asbestos dust causes asbestosis, which differs from silicosis by its slower development and a certain type of pulmonary fibrosis. The earliest symptom is dyspnea upon physical exertion, later accompanied by a harsh, nonproductive

cough and fleeting pain in the chest. In more advanced cases anorexia and cyanosis may develop and there may also be emaciation and finger clubbing. Many asbestos workers show asbestos corns on their skin, caused by hyperkeratosis around asbestos fibers which have entered the skin. Asbestosis is characterized by a diffuse interstitial fibrosis of the lungs with areas of catarrhal changes and the appearance of asbestos bodies in the sputum. It is a progressive disease with a bad prognosis for which there is only symptomatic treatment.

Ellman, P.: *J. Indust. Hyg. & Toxicol.* 15:165, 1933.

Aspidium

Aspidium (male fern, *Filix Mas*) is used as anthelmintic against tape worms. Toxic effects may be observed even with moderate doses and are characterized by irritation of the gastrointestinal tract or symptoms from the central nervous system. In light cases there is vomiting, colic, and diarrhea, followed by headache, dizziness, more or less marked dyspnea, yellow vision, and amblyopia. In severe poisonings the pulse is rapid and the patient may suffer from fainting spells and may pass into collapse. He may become delirious, suffer from muscle cramps, trismus, clonic and tonic convulsions, become comatose, and in many instances develop visual disturbances such as nystagmus, hemianopsia, amblyopia, and amaurosis. Psychosis may be observed occasionally. The liver may be affected, as indicated by the presence of jaundice, and there may be some irritation of the kidney as indicated by the presence of albumin, casts, and sugar in the urine.

Treatment: This consists of gastric lavage, saline cathartics, and enema. The irritation of the intestinal tract may be alleviated by demulcents. Otherwise the treatment has to be symptomatic.

Schlier: *Therap. Monatschr.* 4:465, 1890.

Westphal, A.: *Samml. Vergiftungsfällen* 1:71, 1930.

Aspirin

Aspirin (acetylsalicylic acid) forms white odorless crystals or a crystalline powder which decomposes readily into salicylic and acetic acid. It is used very extensively as an antipyretic and analgesic agent. The ingestion of large doses will cause toxic symptoms similar to those seen in salicylate poisoning (q.v.). The patients suffer from tinnitus and impaired hearing, nausea, vomiting and diarrhea, profuse perspiration, severe thirst and dehydration. Later there is dizziness, anxiety, and great fatigue, passing into somnolence; in severe cases this may be followed by delirium, hallucinations, convulsions, and coma. The vision may be impaired and there may be flickering before the eyes, and in severe cases the pupils are dilated

- Smith, M. I., Franke, K. W., and Westfall, B. B.: *Pub. Health Rep.* 51:1496, 1936.
Lemley, R. E., and Merryman, M. P.: *Journal Lancet* 61:435, 1941.
Middleton, J. M.: *Arch. Ophth.* 38:806, 1947.
Manville, I. A.: *Am. J. Pub. Health* 29:709, 1939.

Shellac

Shellac (lacca) is a resinous exudate of the bark of various resiniferous species of *Schleicheria*, *Butea*, *Ficus*, and others, caused through puncture by the insect *Coccus lacca*. It is one constituent of lacquers and varnishes. The prolonged inhalation of the mist of such lacquers may lead to chronic exudative pneumonia with induration.

- Hirsch, E. F., and Russell, H. B.: *Arch. Path.* 39:281, 1945.

Silica

Silica (silicon dioxide) occurs in quartz, sand, flint, chalcedony, opal, agate, and Kieselguhr. It forms colorless, transparent, and tasteless crystals or a white amorphous powder. It is used in the ceramics industry, and it is one of the hazards in stone cutting, sand blasting, mining, casting, and other trades. Inhalation of silica will result in silicosis which is essentially a fibrosis of the lungs. Its onset is slow and insidious. In the beginning of the disease symptoms may be absent except for changes revealed by x-ray examination. After the process has fully developed, shortness of breath upon exertion, cough, and pain in the chest are the most common subjective signs. The dyspnea is not affected by the patient's position. The objective signs are shortness of breath, cough, prolonged expiration, altered breath sounds, and rales, impaired resonance and the characteristic roentgenographic appearance of the lung fields; however, there is usually neither cyanosis nor clubbing of the fingertips. The blood pressure shows no appreciable rise, but there may be a hypertrophy of the right heart. The silicotic process may arise and continue after discontinuance of the exposure to silica, and there is no therapy to stop the process. Persons suffering from silicosis are prone to contract tuberculosis.

- Dreesen, W. E.: *J. Indust. Hyg. & Toxicol.* 15:66, 1933.
Seevers, M. H., Enzer, N., and Becker, T. J.: *J. Indust. Hyg. & Toxicol.* 20:593, 1938.
Sayers, R. R., and Jones, R. R.: *Pub. Health Rep.* 53:1453, 1938.
Giering, J. T., and Charr, R.: *J.A.M.A.* 113:574, 1939.
Farrell, J. T., Jr., Sokoloff, M. J., and Charr, R.: *Am. J. Roentgen.* 44:709, 1940.

Silver

Silver is a white, malleable, and ductile metal and the most important of its salts from the toxicologic point of view is silver nitrate, which forms colorless, odorless, large, transparent crystals or white small crystals which darken upon contact with organic matter and melt at 212° C.

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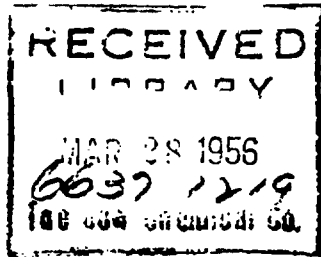
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