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POISONING

*A Guide to
Clinical Diagnosis and Treatment*

S E C O N D E D I T I O N

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U. S. Department of Health, Education and Welfare*

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BIOCHEMICAL RESEARCH

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DEDICATED TO
The Many Physicians Whose
Observations and Reports Have Enriched
Our Knowledge of Poisonings

Preface to the Second Edition

DURING the six years which have elapsed since the appearance of the first edition of "Poisoning" new types of poisoning have been reported, new methods for the detection of toxic materials in biological specimens have been devised, and new procedures for the treatment of poisonings have been proposed. The introduction of numerous new therapeutic agents, some with potentially dangerous side effects, has necessitated a more detailed description of these side actions which do not represent actual poisonings, but the knowledge of which is of vital importance to the correct diagnosis and the proper treatment of the patient. On the other hand the list of chemicals and poisons causing certain functional changes have been revised and an attempt has been made to restrict these to the more characteristic symptoms and to omit minor ones. It is hoped that these changes will increase the usefulness of the book.

It is again an agreeable duty of the author to thank the library staff of the National Institutes of Health for their untiring help in supplying the extensive literature. Last, but not least, thanks are due to the publisher, W. B. Saunders Company, for assuming the publication of this edition.

W. F. VON OETTINGEN

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Preface to the First Edition

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, finding 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 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 these 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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Glossary of the Abbreviations Used in the Lists of Poisons

acute pois.	= acute poisoning	occ. severe	= occasionally in severe poisoning
ac. high conc.	= acute poisoning with high concentrations	oral	= after oral doses
asp.	= aspiration	overdose	= after overdosage
chron.	= chronic poisoning	parent.	= after parenteral administration
chron. early	= early in chronic poisoning	pois.	= in poisoning
chron. late	= late in chronic poisoning	prol.	= in prolonged poisoning
conc. sol.	= concentrated solutions	prol. use	= after prolonged use
cont. expos.	= continued exposure	prot.	= in protracted poisoning
cont. use	= continued use	rare	= rarely observed
clon.	= clonic	rep.	= after repeated doses
exc. doses	= excessive doses	rep. oral	= after repeated oral doses
hypersens.	= hypersensitivity	sev.	= in severe poisoning
ind. expos.	= industrial exposure	sol.	= soluble or solution
inhal.	= by inhalation	subac.	= in subacute poisoning
i.v.	= with intravenous injection	sud.	= after sudden intravenous injection
i.v. rare	= rarely seen after intravenous injection	syst.	= with systemic poisoning
late	= late effect	trans.	= transitory phenomenon
lg. dose	= after large doses	tonic	= tonic convulsions
light	= in light poisoning	wom.	= observed in women

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PART ONE

Introduction



CHAPTER 1

Classification and Diagnosis of Poisoning

THE CORRECT diagnosis of poisonings or of toxic conditions has become increasingly 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.

As stated by many experts, the incidence of poisoning, especially among children, has increased during the past years as the number of substances to which modern man may be exposed is constantly growing. 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 cresol, were the prevalent cause of poisonings. The introduction of essence of vinegar for domestic use in certain countries 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, deodorants, 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 under

lying 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 to 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, had 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 dysfunctioning 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. 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

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Pathologische Physiologie. It is hoped that the comparison of abnormal findings as they present themselves in the course of a systematic clinical examination of patient, and their classification by toxic causative agent or groups 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 and side actions of drugs 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.

CHAPTER 2

Medico-Legal Responsibilities

AS IN ANY DISEASE, it is the duty of the 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 (Occupational Health and Safety Regulation. U. S. Dept. of Health, Education and Welfare; Public Health Service. P. H. S. Pub. No. 357, 1954). According to this source industrial diseases including poisonings are reportable in 29 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.

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 1. State Agencies to Which Occupational Poisonings Should Be Reported

STATE	AGENCY	REMARKS
Alabama	State board of health, county and state health officer	
Arkansas	State health officer	
California	Division of labor statistics	within 7 days
Colorado	Local health officer	immediately
Connecticut	State dept. of health	within 48 hours
Georgia	State board of health	
Iowa	Local health officer	
Kansas	State board of health	
Kentucky	City or county board of health	
Louisiana	Department of health	
Maine	Dept. of health & welfare	within 10 days
Maryland	State board of health	
Massachusetts	Dept. of labor & industry	
Michigan	State dept. of health	within 10 days
Minnesota	State dept. of health	within 5 days
Mississippi	County health officer	
Missouri	Division of health	within 24 hours
Montana	State board of health, div. of ind. hygiene	
New Hampshire	State board of health	within 48 hours
New Jersey	State dept. of health & dept. of labor	within 48 hours (lead)
New Mexico	Local health officer	
New York	Commissioner, dept. of labor	
Ohio	State commissioner of health	within 48 hours
Oklahoma	State dept. of health	
Pennsylvania	State dept. of health & dept. of labor & ind.	
Rhode Island	Dept. of pub. health	
South Carolina	State board of health	
Utah	State dept. of health	
Wisconsin	State dept. of health	

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.

See pages 8 and 9 for Table 2.

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extract (2 cc. daily intramuscularly) and dimercaprol (BAL), 0.5 cc. every six hours for forty-eight hours and then 2 cc. daily for six 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. Arspenamine encephalitis appears to respond promptly to therapy with dimercaprol (BAL), and toxic hepatitis to acetylmethionine (Thiomedon) given orally or intravenously.

- Beattie, J., and Marshall, J.: Brit. M. J., 1:547, 1944.
Fisher, S., Holley, H., and Fein, G.: Arch. Dermat., 55:57, 1947.
Hanger, F. M., and Gutman, A. B.: Trans. Ass. Am. Physicians, 55:179, 1940.
Hattersley, P. G.: Ann. Int. Med., 28:452, 1948.
Shirbal, J. J., and Thurman, F. M.: Am. J. Syph., 19:17, 1935.
Zierz, P.: Arch. Dermat., 190:463, 1950.

Artane

Artane (trihexyphenidyl hydrochloride; alphacyclohexyl-alphaphenyl-1-piperidinepropanol hydrochloride) is a crystalline material which decomposes at 258.5° C. and which is soluble in water and slightly soluble in alcohol. It is used in the treatment of paralysis agitans. Dryness of the mouth and blurred vision are not uncommon side reactions and nausea, dizziness, and confusion may be complaints. Large doses may give rise to hallucinations.

- Doshay, L. J., Constable, K., and Zier, A.: J.A.M.A., 154:1334, 1954.
Porteous, H. B. and Ross, D. N.: Brit. Med. J., 2:138, 1956.

Asarum

Asarum europaeum (Häselwurz) is a small perennial herb which contains aserone (2,4,5-trimethoxy-1-propenylbenzene). The leaves of the plant were formerly used as a diuretic, diaphoretic, purgative and emetic. Decoctions of the root were used to induce abortion. Its ingestion produces nausea, vomiting, diarrhea, and hyperemia of the pelvis. At least one instance of successful use of the plant for abortion is reported.

- Von Oettingen, W. F.: Nat. Inst. Health Bull., No. 190:324, 1949.
Von Sury, K.: Münch. med. Wehnschr., 57:26, 1910.

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. Most patients show a reduction of the vital

capacity, a satisfactory respiratory volume, and hyperventilation with a considerable increase of the respiratory rate. 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. Several authors have reported cases of cancer of the lung in asbestos workers, but their number is too limited to allow a statistical evaluation.

Bastender, H., Denolin, H., DeCosta, A., and Englert, M.: *Ann. mal. prof.*, 16:546, 1955.

Behrens, W.: *Schweitz. Ztschr. allg. Path.*, 14:275, 1951.

Doll, R.: *Brit. J. Indust. Med.*, 12:81, 1955.

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

Smith, K. W.: *Arch. Indust. Health*, 12:198, 1955.

Aspidium

Aspidium (male fern, *Filix Mas*) is used as an 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.

Hall, M. C.: *J.A.M.A.*, 63:242, 1914.

Selander, P.: *Ann. Pediat.*, 173:259, 1949.

Asterol dihydrochloride

Asterol dihydrochloride (6-[betadiethylaminoethoxy]-2-dimethylamino-benzathiazole dihydrochloride; diamthazole dihydrochloride) is a crystalline material which decomposes at 269° C. It is freely soluble in water, methanol, and alcohol. It is used as a fungicide in the treatment of dermatophytoses

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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 Russel, 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 results 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 hypertrophy of the right side of the heart. Electrocardiographic records with chest-wall leads may demonstrate changes in the ST-segment and of the T-wave at a time when these are not demonstrable by the extremity electrocardiogram, and indicate an increased hemodynamic burden placed on the right ventricle. They may be observed in the absence of clinical symptoms of impaired cardiac action. Other electrocardiographic findings are indicative of right ventricular hypertrophy, but these are not so much related to the silicotic process as to the degree of emphysema, which affects the action and the position of the 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. Trauma caused by some silicious substances, such as glass fragments, may result after varying periods of time in the formation of granulomas at the sites of the original injury. They show a typical sarcoid reaction.

TREATMENT. This consists in discontinuation of the exposure and symptomatic and supportive measures as indicated.

Crossland, P. M.: A.M.A. Arch. Dermat., 71:457, 1955.

Giering, J. T. and Charr, R.: J.A.M.A., 113:574, 1939.

Hörmann, J.: Zeitschr. Kreislaufforsch., 39:624, 1950.

Sayers, R. R. and Jones, R. R.: Pub. Health Rep., 53:1453, 1938.

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