Practical Toxicology

for Physicians and Students

Kobert

W. R. Jenkins

PRACTICAL TOXICOLOGY

FOR

PHYSICIANS AND STUDENTS

BY

PROF. DR. RUDOLF KOBERT

MEDICAL DIRECTOR OF DR. BREHMER'S SANITARIUM FOR PULMONARY DISEASES AT GOERBERSDORF IN SILESIA (PRUSSIA); LATE DIRECTOR OF THE PHARMACOLOGICAL INSTITUTE, DORPAT (RUSSIA)

TRANSLATED AND EDITED BY

L. H. FRIEDBURG, PH. D.

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FROM THE PREFACE TO THE SECOND GERMAN EDITION.

This second edition is not offered as a great scientific achievement, neither was the first one. Nor is it intended to compete with the existing handbooks on toxicology.

"*The aim of the book shall be reached if it can be used to advantage by such physicians and students of medicine, who are not able to spend either much time or much money upon the study of toxicology.*"

Kösen, August 15th, 1887.

FROM THE PREFACE TO THE THIRD GERMAN EDITION.

The exceedingly sympathetic reception which the present unpretentious little book has met, has been shown by different facts.

The first edition originated from *Werber*. The second edition was entirely revised and changed, and ran rapidly out of print. The most diversified scientific journals criticised it favorably, and the right of translation into the Danish, Russian and Hungarian languages was asked for.

Such are the reasons which spurred the author's ambition to render the new edition, if possible, still more practical than the second one, which for years has been out of print. After

ample consideration, we believe we have achieved this by the introduction of *tables which, though occupying but little space, allow at a glance the observation of similarities and differences between poisons belonging to the same group.*

The detailed treatment of the individual poisons, which has but recently been furnished in an elaborate manner by the author in his "*Text-book of Intoxications*," (Stuttgart, 1893), has been condensed in this way.

Thus it has been possible to reduce the volume of this book, and at the same time to increase its contents, as compared with the last edition.

R. KOBERT.

Dorpat, 10-22 March, 1894.

PREFACE TO THE FIRST AMERICAN EDITION.

For several years I have followed "Kobert's Compendium of Practical Toxicology" in my lectures and demonstrations at the American Veterinary College, as well as at the Flower Hospital Medical College. The students were particularly anxious to obtain copies of the *tables*, or abstracts thereof, which gave to them "all they cared to know" about toxicology.

It seemed to me a matter of common politeness, if not justice, to translate the entire little book (permission for which was cheerfully granted by Dr. Kobert) and thus to put it into the hands of our medical students and lecturers alike. But it is at the same time the intention to aid the practicing physician, who wants "practical knowledge at a glance," not having the time to peruse the larger handbooks in each particular case of poisoning.

The language used in this book is intended to be without elocutionary pretentions. But, where the text has failed to clearly convey the idea of the fact involved, I ask for the criticisms of my colleagues, and shall be pleased to make use of them on a future occasion.

Some of the American poisonous plants have not been embodied in this compendium, for the simple reason that I have not been able to find reliable sources of information upon the same basis of treatment which plants have received in this book.

I shall highly appreciate any information furnished me in this regard, as well as communications upon toxicological cases from actual practice.

I have attempted to follow the chemical nomenclature according to the "*Rules for the Spelling and Pronunciation of Chemical Terms*," as adopted in 1891 by the American Association for the Advancement of Science, a copy of which has been embodied in this work. It may be, that here and there I have unwittingly failed to comply with these rules, but I have in only a single instance intentionally diverged from them, using the words "glycosid" instead of "glucoside," and "glycuronic acid" instead of "glucoronic acid," since I consider this more in conformity with the words "glycerol, glycol, glycocoll."

May the American edition of *Kobert's Compendium* find as many friends in this country as the German one so readily secured everywhere.

L. H. FRIEDBURG.

New York, September 17, 1897.

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PRACTICAL TOXICOLOGY

MOTTO: *Toxicology is the basis of morbid physiology and of therapeutics.*

GENERAL TOXICOLOGY.

I.—Definition—Source of Poisons.

The layman, the lawyer, and the physician have each his own conception of the meaning of the word "poison." We physicians regard as poisons such non-organized bodies—either inorganic or organic—that, by their chemical nature, under certain conditions, so affect one or more organs of living beings as to damage either temporarily or permanently the health, or comparatively healthy condition, of these organisms. These poisons may be developed within the organism, or may be introduced from without.

This definition excludes all mechanically acting irritants, such as boiling water, powdered glass, pins, etc., as well as injurious microbes and other organisms. It also avoids the mistake, made in a great many definitions, of regarding as poisons only such pharmacological agents as injure the healthy organism; for some agents which are well borne by the healthy organism, prove injurious in certain diseased conditions, and then are poisonous—*e.g.*, strong coffee. It is evident that it is erroneous to designate as poisons only such substances as may cause death. Every article of food taken, with a disordered stomach as the result, is a poison in that particular instance, even though at other times it has no injurious action.

We conclude, then, that for us no substances are "poisons in themselves"; and that it is only when the particulars are known that we are able to say, in any given case, whether the substance involved can be classed as a poison or not. Then, again, the above definition is so framed as to include substances which are poisonous to plant-life. Of course it includes the so-called endogenous toxins, viz., poisons which are produced within the human system.

From a pharmacological standpoint the above definition can be stated concisely, as follows: All pharmacological agents which, in a given case, do not act beneficially, but injuriously, are poisons.

It is naturally of interest to the physician, to know what position is held by the state or the country in which he lives, regarding the definition of the word "poison." Any physician may be called upon, or may be eventually forced, to express his opinion in court, as an expert, as to whether a given case is one of poisoning, or not.

The statutes of the State of New York, and those of the United States, do not define the word "poison." Words are there used to indicate their general meaning, unless something is found in the context to denote some special or restricted use.

According to its generally received meaning, we can say: In most cases a poison is a substance which, when given even in small doses, owing to its chemical constitution, is capable of destroying health or life.

Only a few poisons are derived from the animal and mineral kingdom. By far the largest number are produced from plants, not only of the higher, but also of the lower orders. The number of those prepared synthetically by the chemist grows steadily.

II.—Conditions of Poisoning.

It is quite clear that not all poisons act to the same degree; some act a thousand times—yes, even a million times—less powerfully than others. First of all, therefore, this depends upon the quality of the poison. Next, the quantity must be considered. All poisons, even the most powerful, may be given in doses so small as to have no toxic action. Starting from this inocuous amount, and gradually increasing it, we reach first the medicinal dose, viz., a quantity which, under certain conditions, has a beneficial action upon the sick; then the toxic dose, which is harmful to both the healthy and the sick, but which does not kill; and, finally, the fatal dose. It is a matter of great importance whether the poison be administered in substance or in diluted solution. For example, when *arsenic* is given in substance, the symptoms, which appear rather late, are referable to the intestines, whereas, when given in diluted solution, the general symptoms are the most prominent and appear very quickly. When given in substance, the character and rapidity of its action depend upon the solubility of the

poison, and also upon its physical state, viz., whether the poison is in lump or in the form of a loose powder. In case the poison is given in solution, its action is again modified according to the degree of concentration and the temperature of the solution. Hot poisoned draughts act more quickly than do cold ones. The vehicle must also be considered, for an alcoholic solution is more quickly absorbed than is an aqueous, and both of these more quickly than an oily solution. Age and method of preservation are important factors in modifying vegetable and animal poisons, as a great many of these decompose when kept for any length of time, more especially when their non-sterilized solutions are kept in a warm situation, exposed to light and in contact with oxygen.

It also makes a difference whether the organism to be poisoned is a plant, an animal, or a human being. The relative susceptibility to poisons, of the different classes of plants, has not been thoroughly investigated. There is, however, great diversity in this respect among animals.

Consequently, it is non-scientific and wrong to draw inferences regarding their effect upon man from the action of poisons in the frog —that pet of the toxicologist. The smallest snail will withstand more *strychnin* than an adult man. Many of the strongest *cardiac* poisons have no action whatsoever upon insects. Great care is necessary in thus reasoning from even the effects noted in experiments with warm-blooded animals approaching nearer to man. The rabbit can take more *morphin* than can a man of fifty times the animal's weight. Doses of *lead*, *nicotin*, *cytisin*, etc., sufficient to fatally poison man, do not injure the goat. *Amygdalin* does not affect dogs, but it kills rabbits. The hedgehog takes, with apparent enjoyment, a dose of *cantharides* that would kill several persons under excruciating pains. The bite of the most venomous snake does not harm him; he can even accommodate no inconsiderable quantity of *hydrocyanic acid*. Whereas the frog is extraordinarily susceptible to the *digitalis* poisons, they have no effect upon the toad.

Regarding the site of application, we must consider the various possible localities wherein a substance may demonstrate its poisonous quality, viz.: (1) The intact skin, for volatile and irritating substances, such as *nicotin* and *cantharidin;* (2) the inflamed or lacerated skin, even for substances which are non-volatile and non-inflammatory; (3) the subcutaneous tissue; (4) the readily accessible mucous mem-

branes of the eye, the nose, the ear in case of a perforated drum, the mouth, the pharynx, the larynx, the urinary passages, the vagina (cases of poisoning by *arsenic* administered *via* this source are on record), the rectum (death has been observed from rectal injections of tobacco and lupine alkaloids); (5) the stomach; (6) the cavities of the body (death has often resulted from irrigation of the pleural or peritoneal cavities with solutions of corrosive sublimate or carbolic acid); (7) the lungs (the inhalation of *arsin, prussic acid, hydrogen sulfid, carbon monoxid, carbon di-oxid,* has caused numerous deaths); (8) the circulatory system (ether, accidentally introduced into a small vein during hypodermic injection has proved fatal). Poisons act more powerfully when absorbed from the subcutaneous connective tissue than when administered internally, with the following exceptions:

The neutral *crotonolglycerid,* which is found in large quantities in the fresh seeds of *Croton Tiglium,* but which is often lacking in commercial croton oil, is inactive when introduced under the skin. It possesses, however, terrific action when taken into the stomach. *Myronic acid* of mustard, as an alkaline salt has no effect when it is injected under the skin; it has, on the other hand, a strong action when taken *per os* by herbivora; the same is true of *amygdalin.* In all three of the foregoing cases the apparent exception to the rule is explained by the fact that the substance, in itself not poisonous, is split up in the intestinal tract, giving off, amongst others, a toxic substance. In the instance first mentioned, *crotonolic acid* is the poison thus freed; in the second, ethereal *mustard oil;* and in the third, *hydro-cyanic acid.* Some substances, such as *salts of manganese, iron, tungsten,* have no poisonous action when introduced into the intestinal tract, because under these conditions only very minute quantities are absorbed; others are rendered inert because they are excreted almost as quickly as they are taken up, *curare* being an example; and yet others, such as *snake-poison, spider-poison, quillaic-acid, sapotoxin, ergotinic acid,* are converted into non-poisonous substances within the intestine.

The action of all poisons is rendered weaker when they are taken into a full stomach than when received by that organ when empty. It must not be forgotten that the organism can evacuate the poison from the stomach and intestines; whereas we are unable, even artificially, to rid the system of poisons introduced into the subcutaneous connective tissue. Failure to observe this fact has been the cause of a number

of fatal issues in the modern treatment of syphilis, by subcutaneous injections of mercurial preparations. Inunction of healthy skin acts less powerfully than when applied to the inflamed or lacerated areas. The healthy urinary bladder absorbs only very small amounts of substances which are not caustic and not volatile. Absorption takes place quite freely from the surface of all parenchymatous organs, as well as from the cavities of the body. In man, age, the condition of the stomach (as to whether filled or empty), constitution, the general condition of the patient, his habits, and even race and idosyncrasy, are important factors.

The Malays react differently to *opium* than do the Europeans: the former run about in an excited manner, whereas the latter are narcotized by the drug. Children are particularly susceptible to *opium* on account of their not being accustomed to intoxicants; just as old people are readily affected by vasomotor poisons, as *cytisin*, on account of the atheromatous condition of the arteries. Habit is a most important modifying factor. If a person daily indulges in *alcohol*, *morphin*, *opium*, *cocain*, *hashish*, *caffein*, *nicotin*, *piturin*, *kawa*, or *arsenic*, etc., he not only gradually accustoms himself to large quantities of his respective drug, but finally reaches that stage when he must have it, in order to remain in a pseudo-normal condition. He has now arrived at a stage wherein the suppression of the use of the accustomed poison has a deadly toxic effect; the system is unable to continue for a single day without the drug. In such a case, insomuch as we have a disturbance, caused by the withdrawal of the poison, it is therefore not a case of poisoning, in the limited sense of the term, although certainly a poisonous effect is produced. The influence of the general nutritive condition of the body has often been observed, more especially in the case of starving vagrants, who are killed by quantities of poisonous berries, putrid food, etc., which would in other and more normal individuals cause nothing more than a digestive disturbance speedily relieved by vomiting of the ingesta. The constitutional influence is seen to particular advantage in scrofulous children, in whom the reaction to poisons is stronger than in the healthy. By the term idiosyncrasy (lit., a peculiar mixing together of the juices) we understand the following two facts: 1st, that some people react in an abnormally strong manner to even minute doses of certain drugs; and, 2d, that some people are made seriously ill by partaking of, or even smelling, certain substances which to others are indifferent or even agreeable. Among such drugs we mention: calomel, morphin, turpentine; of the foods: lobster, raspberries, strawberries, mutton, fish, eggs, honey, cocoa, and beans; and of the odors: musk, sewer-gas, the scent of flowers, and the smell of toads. The drugs noted give rise to

symptoms such as would be caused by very large doses thereof. This observation applies especially to the cutaneous manifestations, principally eruptions; and sometimes excitation is present where we expect depression. The other substances give rise to astonishing symptoms, which we are unable to explain satisfactorily, *e. g.*, *urticaria*, following the ingestion of crabs, raspberries, strawberries, or other red dishes; attacks of sneezing, when in the presence of toads; *steno-cardic* attacks, after partaking of beans; colic, following a single cup of cocoa; fainting spells, illusions, hallucinations, and other nervous phenomena, caused by the exquisite scent of the rose, hyacinth, violet, lily, or auricula.

III.—Action and Localization of the Poisons.

We are still far from understanding the ultimate cause of action of a great many poisons; the attempt to regard every poisonous effect as a disturbed cellular isotony advances us no further in the appreciation of poisonous action. "The problem of the origin of a ganglion cell is no deeper than that of the decline of its functions from the action of morphin." In general, we distinguish two kinds of poisonous action: local and remote.

By local action we mean those symptoms and changes which the poison produces at the site of application. Some agents, such as *the salts of the heavy metals*, will readily combine with the protein substances, thereby causing their destruction, viz., necrosis of the tissues involved; others, as *concentrated acids* and *caustic alkalies*, act also as powerful irritants and cause a reactive inflammation; still others, as *strychnin*, *morphin*, *curarin*, and *muscarin*, cause an excitation or enfeebling of the nerves, muscles, or glands of the affected parts, without any marked apparent changes.

The remote effect is produced by the absorption of the poison into the lymphatics and into the blood, causing general symptoms, and diseases of other organs, *e. g.*, of the kidneys, following the administration of *cantharidin;* of the brain, after taking *opium;* of the intestine, after *quillaic acid.* Practically, the remote action is really a local one, produced by the poisoned blood circulating everywhere.

The poison as it circulates in the blood may either be decomposed, or it may enter into combination with the blood

constituents and thus change the composition of the blood, or it may reach the various organs in its original condition. Physiology teaches us that various endosmotic changes take place in these organs, depending upon their functions, upon the formation of their constituent elements, and upon the number and arrangement of the capillaries passing through them. The chemical constitution and physical properties of the poison will determine, to a varying degree, the rôle it plays in these changes upon participating in the interaction of the vessels of the tissues. The presence of this foreign substance sooner or later disturbs, to a greater or less degree, the healthy condition and function of the organs particularly affected; and, again, this cannot take place without a reaction upon the whole body. The animal organism, however, possesses four means of rendering partly or entirely harmless, poisons which have entered the system:

1. Rapid elimination. Under this head, naturally, we first mention vomiting, which, fortunately, occurs so promptly following the introduction of most poisons into the stomach that it generally saves the life of the patient, or at least has already materially lessened the danger to life before the physician puts in an appearance. We should call this vomiting, which takes place before the absorption of the poison, *primary* vomiting, in contradistinction to a secondary emesis, which takes place following absorption, and which latter is either exclusively a sign of disturbed cerebral activity, or is caused by the excretion of the poison from the blood into the stomach. In an analogous manner we must differentiate between a primary diarrhœa, which carries off the poison before absorption, and a secondary purging, which is a sign of disturbed intestinal innervation, or is caused by the excretion of the poison from the blood into the lumen of the gut. Some poisons are not removed by vomiting or by purging, but appear in the urine within a remarkably short time. Thus, for example, it is impossible to produce complete curarization by the administration of moderate though oft-repeated doses of *curare*, because the excretion of the poison through the kidneys takes place as rapidly as does absorption. The liver, pancreas, gastric mucous membrane (for *morphin*), intestinal mucous membrane (for *mercury*), salivary glands, mammary glands, and lungs, are other channels effective in assisting the excretion of various substances from the blood. Not nearly enough attention was formerly given to the excretion through the glands of the mucous membrane of the stomach. Finally, elimination takes place through the structures of the skin, especially through the sweat-glands.

2. The organism deposits and fixes poisons, in a manner not yet

sufficiently understood, in several organs, especially in the liver, which certainly must be regarded as a filter for poisons—so far, at least, as enzymes (*e.g.*, *emulsin*), metals (*iron*), metalloids (*e.g.*, *arsenic*), and alkaloids (*e.g.*, *strychnin*) are concerned. It is probable that, in the case of some substances, the biliary acids play an important part in the matter. We can hardly imagine that this disposition is accomplished in any other way than in the transformation of the readily soluble poisons into saline combinations, not freely soluble (bile-acid-alkaloids), or into albumen derivatives (metalalbuminates). But, since these combinations are in no case entirely insoluble, the beneficial action of the liver only consists in the fact that it gives the acute poisoning a more protracted and consequently a milder course.

3. The organism renders the poisons innocuous by phagocytosis. This destructive crusade carried on in the interest of the body by phagocytes, which has not yet been sufficiently inquired into pharmacologically, is applicable for certain toxalbumins (*toxopepton*, *enzymes*, as well as for heavy metals (*iron*, *silver*).

4. The organism transforms the poison into a comparatively harmless, though readily soluble, combination. Such a transformation may consist of neutralization, oxidation, reduction, coupling, splitting and peculiar changing of the chemical constitution. 1. As an example of poisons rendered inert by neutralization, we mention the *acids*, which are transformed as far as possible by the organism into the corresponding alkaline salts of less poisonous or absolutely non-poisonous properties. So far as the stomach is concerned, the organism attempts to balance any excess of *alkali* by the acids of the gastric juice, and does the same thing in the blood by the decomposition of an immense number of blood corpuscles, whereby glycerinphosphoric acid is formed from lecithin. *Caustic lime* is combined with *carbaminic acid*, and then excreted. 2. The best known example of inertia produced by oxidation is that of *phosphorus*, which is transformed into the phosphates. In an analogous manner the extremely poisonous *sulfids* are converted into sulfates which are relatively non-poisonous. The organic acids and their salts are oxidized to the ultimate degree, producing carbonates; and it is a prominent and important fact, that in the latter case the dangerous diminution of the alkalescency by means of these acids is transformed into an increase of alkalescency, since even the bi-carbonates are of alkaline reaction. 3. Examples of producing inertion by means of reduction are offered in the cases of *iodates*, *chlorates*, and *perchlorates*, which are excreted in the markedly less poisonous form of chlorids and iodids. 4. Inertion produced by coupling is one of the most remarkable facts in physiological chemistry. An intimate knowledge of this phenomenon is as imperative for the physician at the bed-side as for the chemist entrusted with the chemical analysis of the remains. A poison can unite by coupling: (a) with sulfuric acid (*e.g.*, *phenol* and *cresol*), (b) with glycuronic acid (*e.g.*,

camphor, *borneol*, *menthol*), (c) with glycocoll (*e.g.*, *benzoic acid*, *anisic acid*, a part of *salicylic acid*). 5. Inertion produced by splitting occurs with *tannic acid* of nut-galls, and with some glycosids (*e.g.*, *salicin*). 6. Examples of changes peculiar to themselves, as productive of inertion, are offered by the *salts of ammonia*, which are transformed into urea.

The liver is the most important organ in producing changes in poisons peculiar to themselves. Coupling occurs partly in the liver and partly in the kidney. Splitting processes take place mainly in the intestinal canal, although the liver must also be considered in this connection.

For a time it seemed that we were justified in supposing that organic substances could be divided into two well defined classes, according to their respective actions exhibited within the animal body: the substances of the fatty series were supposed to be destroyed, while those of the aromatic series were not. To-day, we know that this does not hold good for all substances: not even *Oxamid*, belonging to the fatty series, a trace of which is oxidized; and *Tyrosin*, a member of the aromatic series, which can be completely transformed into urea, carbon di-oxid and water.

This observation, therefore, can at the present time be stated only in the following form: organic substances containing annular linkage within the molecule are frequenly not oxidized to form carbon di-oxid, water and urea. It is immaterial whether or not they belong to the aromatic series proper. Substances not containing annular linkage, which are oxidized with difficulty, or not at all, are mainly certain *amids*.

One of the foremost tasks of scientific pharmacology is, to explain the relation between the chemical structure of a substance and its pharmacological action. Unfortunately, it can only be said at present that uniform laws, which would be of great service to the physician, have not yet been discovered. This is neither the time nor the place to dilate upon the many interesting fundamental structures of such laws.

IV.—Ætiology and Classification of Poisoning.

In medical practice we ordinarily distinguish between acute and chronic poisonings, the former including cases in which a single large dose of poison was taken, whereas the latter are

such as are caused by the repeated exhibition of small doses. However, an extended experience at the bed-side reveals the fact that even acute cases may take a chronic course. For example, a case has been reported in which a single dose of *arsenic* produced in a student a trouble lasting for a number of years. Retention of the poison is not of necessity the cause of this chronicity, for it is possible that the poison may bring about long lasting pathological changes in an organ, which may endure even throughout life. Thus there are cases of stricture of the oesophagus after the exhibition of corrosive poisons, deafness or blindness after *quinin*, cerebral softening after *carbon monoxid*, cirrhosis of the liver after *phosphorus*, chronic nephritis following the use of *cantharides plasters*, or subcutaneous treatment with *aloin*, sloughing of various parts of limbs after the use of *ergot*.

The medico-legal classification considers only the impulse or motive which results in the introduction of poisonous substances into the organism, as in cases of murder and suicide, and in poisonings resulting from industrial pursuits, technical products, economy, and medicinal treatment.

Among the poisonings arising from certain industrial pursuits, we mention: those by lead, in the cases of potters and printers; by mercury, in the manufacturing of thermometers, etc., and of incandescent electric lamps; by phenol, in the case of surgeons who worked with the phenol spray; by phosphorus, in match manufactories, &c. By poisoning from technical products, we understand not those cases which are found amongst the workmen in the manufactory, but those arising in the consumers of the manufactured product. Such are cases of poisoning by wall-papers or upholstery containing *arsenic*, stockings containing *antimony;* sleeping in newly-varnished bedrooms, &c. Economical poisoning embraces those cases produced by partaking of food improperly prepared, and consequently rendered poisonous (insufficiently smoked sausages, which are stuffed into casings of intestinal membrane; fruit cooked in copper vessels, over-kept cheese, venison of haut goût. Medicinal poisoning includes those cases for which we physicians are responsible, and which afford opportunity for well-founded censure to the homœopathists and to those who adhere to cures by nature.

Physiological chemistry differentiates between exogenous and endogenous intoxications, according to whether the poison

is introduced from without or is formed within the organism. Endogenous intoxications are subdivided into retention-toxicoses and noso-toxicoses.

Retention toxicoses are developed when substances, which are normal products of metabolism, are for various reasons, not excreted, and, therefore, then accumulate in the organism in excessive quantities, and thus act like poisons. It seems to be a general law of nature, that all organisms generate products of metabolism which, in excessive quantity, bring about a diseased state or even the death of the mother organism. Such retention-toxicoses occur: (1) when the skin does not allow the passage of such products of metabolism as normally are excreted through it, and which represents the *perspiratio insensibilis*. Thus are explained the grave symptoms that appear in animals when their skins are varnished, or the coma, pathological euphoria, and subnormal temperature in man, in case of extensive burns; (2) when the intestine does not conduct *ad anum* those substances which it properly should excrete, a condition which not infrequently obtains in: incarcerated hernia, volvulus, compression from tumors, clogging by fæcal masses or obstruction from stricture. The more important substances causing toxic action under the foregoing conditions are : scatol, indol, methyl-mercaptane and hydrogen sulfid; (3) when the respiratory tract ceases to properly eliminate the carbon di-oxid, and there are produced as a result of poisoning thereby, cyanosis, slow pulse, dyspnœa, and spasms; (4) when the uropoëtic system, especially the kidney, does not secrete and void the urine in a satisfactory manner. The clinical picture that follows in consequence of this has long been known as uræmic intoxication, and it is probably caused by an increase of the potassium salts, creatinin, xanthin substances, acetene, fatty acids, &c.

Nosotoxicoses (from $\nu o \sigma o \varsigma$=meaning disease) are produced: (*a*) when in diseases not caused by a living contagium, qualitative and quantitative disturbances of metabolism occur, which cannot be sufficiently brought into equilibrium despite the fact that all avenues of egress are open. Into this class we put the so-called aut-intoxications (by an excess of hydrogen sulfid, excess of lactic acid, by substances of the character of acetone, oxybutyric acid, &c.); also the poison of the so-called coma of carcinoma, will belong here, provided this disease is not in the future found to be of bacterial origin; then again, oxaluria, if this really exists as a disease *sui generis*, &c.; (*b*) when in diseases recognizing a *contagium vivum* as their cause, poisonous products of metabolism of these microorganisms are produced (in diphtheria, tetanus, rabies, tuberculosis), or in conditions where abnormal waste products are formed from the normal constituents of the organism or from endproducts of metabolism (ammonia from urea). We shall subdivide the intoxications into such which, as a rule, cause gross anatomical changes, and such which may

act fatally, without producing gross anatomical changes. Between the two we place the group of blood-poisons. With these, the blood only is found to have changed, or at least *primarily* so, and the changes within the other organs are brought about in consequence.

V.—The Diagnosis of Poisoning.

At times the recognition of a case of poisoning is comparatively an easy matter, while again, it may be very difficult, or even impossible. We can readily understand this when we consider the large number of poisons, how different their mode of action, and how the symptoms of one and the same poison may vary, according to the size of the dose and the form of administration. This fact becomes all the more apparent when we remember that the various symptoms (because they are only symptoms) may appear similarly in other diseased conditions. The following are, in general, the signs which are of importance in making a diagnosis:

1. *The Symptoms.*—For a great many acute cases, it is true, as the old saying has it, that poisoning, caused by the introduction of a poison from without, is probable when marked and violent symptoms suddenly appear in an individual previously perfectly healthy. But, on the one hand, people who have already been ailing may become poisoned; and, on the other hand, this statement excludes the frequent cases of chronic poisoning. Then, again, death can be caused suddenly by such disease as cholera, rupture of the heart, apoplexy, embolism, intestinal obstruction, peritonitis due to perforation of the intestine, uræmia. The suspicious symptoms become more conclusive when they appear shortly after the partaking of any food, and especially when they appear simultaneously in several persons, and even in domestic animals.

The symptoms may affect all the organs; the most important are vomiting, diarrhœa, convulsions, paralyses, cyanosis, dyspnœa and disturbance of the pulse. The following table seems to be of practical value in making a diagnosis:

TABLE OF THE MOST IMPORTANT SYMPTOMS OF ACUTE ACTION OF THE MORE COMMON POISONS.

NO.	IN THE PRESENCE OF THE FOLLOWING SYMPTOMS:	WE SHOULD FIRST THINK OF:
1	Death within a few moments	Hydrocyanic acid, potassium cyanid, carbon di-oxid, carbolic acid

NO.	IN THE PRESENCE OF THE FOLLOWING SYMPTOMS:	WE SHOULD FIRST THINK OF:
2	Deep coma	Alcohol, morphin, opium, chloralhydrate and its analogues, sulfonal, chloroform and its analogues, carbonmon-oxid, anilin oil, oxybutyric acid
3	Collapse	Corrosive acids, corrosive alkalies, nicotin, arsenic, preparations of antimony, colchicin
4	Febrile rise of temperature	Phosphorus, cocain; under certain conditions, all powerful convulsants, enzymes
5	Raving mania, furibund delirium, psychical excitation	Chronic alcoholism, atropin, cannabinon, camphor, physostigmin, veratrin, lead (in animals)
6	Mental depression	Morphin habit, cocain habit, maydism, ergotism, etherism, saturnism, mercurialism, alcoholism, iodoform, carbon disulfid
7	Severe convulsions, even tetanus	Strychnin, toxin of the tetanus bacillus, salts of ammonia, cytisin, cornutin, picrotoxin, cicutoxin, digitaliresin, cocain, santonin, guanidin, aconitin, condurangin, corydalin, gelsemin, filicic acid
8.	Paralysis generally ascending	Coniin, curare, guachamacá-poison, colchicin, ergotinic acid
9	Dilated pupils	Atropin, hyoscyamin, scopolamin (hyoscin), homatropin, cocain (contracted later on), ephedrin, aconitin, coniin, gelsemin
10	Contracted pupils	Muscarin, pilocarpin, nicotin, morphin, codein, opium, physostigmin
11	Moist skin	Opium, morphin, aconitin, muscarin, pilocarpin, nicotin, physostigmin, lobelin, antimony
12	Markedly dry skin, even in heated bed; mouth and pharynx also dry	Atropin, (parts of the deadly nightshade, thornapple, henbane), hyoscyamin, scopolamin, hyoscin, allantotoxin (a poison found in decaying fish).
13	Urticaria. or scarlatinal rash	Atropin, hyoscyamin, antipyrin, quinin, balsam of copaiba, cubebs, chloral hydrate, iodin, morphin

NO.	IN THE PRESENCE OF THE FOLLOWING SYMPTOMS:	WE SHOULD FIRST THINK OF:
14	Eczematous eruption	Croton oil, curcas oil, cardol, poor kinds of vanilla, dust of the bark of cinchona, carbolic acid, tar
15	Acne pustules	Bromids, compounds of/or substances containing antimony, arsenicals, emetin
16	Clear vesicles on the skin, or even in the mouth	Preparations of cantharides, Ranunculus acris, R. sceleratus, etc.
17	Skin having a dark, muddy discoloration, but not cyanotic	Argyria, poisoning by mercury, lead, copper, arsenic, (arsenical-melanosis)
18	Dark border on the gums	Lead, silver, mercury, bismuth, copper
19	Discoloration of the tongue and of the mucous membrane of the mouth	*Reddish-yellow:* salts of chromic acid and bichromates, *Yellow:* nitric acid, picric acid, *Brown:* iodin, bromin, *Greenish-blue:* salts of copper, Paris green, *White:* corrosive alkalies, corrosive acids, corrosive metallic salts
20	Specific odor of the breath	Opium, alcohol, paraldehyde, amylene-hydrate, pental, chloroform, ether, brominated ether, creasote, iodin, iodoform, bromin, bromoform, phosphorus, nitrobenzene, hydrocyanic acid, all ethereal oils, salts of tellurium, amylnitrite, ammonia
21	Salivation	Pilocarpin, muscarin, nicotin, cornutin, physostigmin, cytisin, mercury, ammonia, arsenic, antimony, cantharidin, saponin substances, etc., etc.
22	Barking cough and aphonia	Atropin, hyoscyamin, scopolamin, allantotoxin
23	Oedema of the glottis	All corrosive poisons
24	Pulmonary oedema	Morphin, muscarin, pilocarpin, ammonia
25	Phosphorescence of the breath, of the vomit or of the urine	Phosphorus

NO.	IN THE PRESENCE OF THE FOLLOWING SYMPTOMS :	WE SHOULD FIRST THINK OF:
26	Vomiting and purging	Antimony and substances containing it, arsenic and substances containing it, substances of the digitalin group, pilocarpin, nicotin, muscarin, colchicin, corrosive poisons, colocynths, emetin, croton oil, etc., etc.
27	Vomiting without diarrhoea	Apomorphin, lobelin, cytisin, Narcissus poisons
28	Diarrhoea without vomiting	Jalap, podophyllotoxin
29	Markedly slow pulse	Opium, morphin, muscarin, substances of the digitalin group (later pulse becomes accelerated), pilocarpin (later accelerated and irregular), nicotin (acts like pilocarpin), physostigmin, lead, baryta, all narcotics
30	Greatly accelerated pulse	Atropin, hyoscyamin, scopolamin, etc.
31	Wiry pulse	Substances of the digitalin group, baryta, lead
32	Peripheral parts of the body becoming black and blue	Gangrenous ergotism
33	Cyanosis	Nitrobenzene, benzocoll, anilin, toluidin, antifebrin, exalgin
34	Icteric or pseudo-icteric, yellowish-brown discoloration of the conjunctiva, or of the skin	Phosphorus, helvellaic acid, phallin, solanin, saponin substances, potassium chlorate, sodium nitrate, amyl nitrite, pyrogallol, arsin, ictrogen (in animals)
35	Icteric urine	Phosphorus, toluylene diamin, phallin, cephalanthin, ictrogen (in animals)
36	Yellow skin and reddish-yellow urine	Picric acid and its salts
37	Urine the color of claret from haematoporphyrin	Sulfonal, trional
38	Urine contains blood coloring matter in solution	Cyclamin, solanin and other saponin substances, phallin, helvellaic acid

NO.	IN THE PRESENCE OF THE FOLLOWING SYMPTOMS:	WE SHOULD FIRST THINK OF:
39	Urine contains methaemoglobin with or without haematin	Arsin, potassium chlorate, sodium nitrite, amyl nitrite, pyrogallol, chrysarobin, all corrosive poisons
40	Urine that reduces Fehling's solution	Carbon monoxid, chloralhydrate, chloroform, pyrogallol, oxalic acid and its salts, formic acid, phloridcin, uranium salts, benzaldehyde (oil of bitter almonds)
41	Urine becoming dark-green when exposed to the air	Carbolic acid
42	Urine which upon access of air slowly turns scarlet	Santonin
43	Acid urine, containing colorless crystals	Oxalic acid and its salts
44	Urine with a marked odor	Oil of turpentine (violets), other ethereal oils, methylmercaptane, ammonia, tellurium, asparagus
45	Anuria	Oxalic acid, potassium oxalate, oxaminic acid, oxamid, cantharidin, corrosive sublimate and other preparations of mercury.
46	Priapism	Cantharidin, poison of Gyrinus natator
47	Abortion	Juniperus Sabina, Ruta graveolens, Mentha pulegium, phosphorus, cornutin
48	Where poisoning is first apparent from 12 to 24 hours after the meal	Mushrooms containing phallin, arsenic not in solution

2. *Exact history of the case.* It is often necessary to inquire into family, social and physical conditions as well as into the history of the disease itself. If there is no reason to suspect murder or suicide, and the symptoms follow the partaking of any food or drink, we would then suspect an accidental poisoning by poisonous food. At such times there are often others than the immediate victims who have partaken with them of the same food, but who have not sought medical

aid because of the insignificance of the symptoms experienced in their cases.

3. *Morbid anatomical changes* may sometimes be noticed during life, if the mouth, pharynx, anus, faeces, urine and vomit are carefully examined. A *post mortem* examination decides the diagnosis.

4. *The finding of poison* in the sick-room, *extra corpus*, or, the determination of its presence in the cadaver and in its excretions by means of pharmacological, physical or chemical examination. This, naturally, affords the most reliable information, although even then we are not always furnished with conclusive proof, since a number of poisons are medicines, or even foods. Then, again, there may be accidentally brought near the cadaver such poisons as artificial flowers, colored with *arsenical* preparations, earth of the churchyard containing *arsenic*, or, for the purpose of deception, a poison may even have been applied on or into the cadaver.

In most cases, however, the due consideration of all these points will enable even the physician, who possesses but a moderate medical experience and knowledge of human nature, to decide either for or against poisoning. In cases in which the medical attendant is unable to make a positive diagnosis, he may say, that while as a physician he cannot assert positively that poisoning obtains, however, as a man, he is satisfied that such is the case.

VI.—Prognosis in Poisoning.

Since the chief factor of prognosis, the nature and strength of the particular poison, comes under the subject of Special Toxicology, we will here mention only a few points, calling attention to those which are of importance in all cases. In general the prognosis is more unfavorable in proportion to the size of the dose received. However, this is not always the case, as the vomiting that follows large doses, taken internally, can rid the system more or less completely of the poison; the solubility and concentration also influence the prognosis. The danger to life is increased when the poison has been introduced into an empty stomach or given to debilitated persons, to children and to old persons. The time that elapses before medical treatment is instituted, is also of great importance.

The prognosis is graver when the poison is administered subcutaneously, because in this case it cannot be removed even partially. Many cases of poisoning, particularly from *mercury*, could be avoided if physicians would bear this well in mind.

In cases of poisoning, it is more necessary even than in disease to make a clear distinction between the prognosis *quoad vitam* and the prognosis *quoad valetudinem*. It is by no means necessary to make a grave prognosis as to life even if the dose received be the usually fatal one. The outcome depends more upon the question whether by artificial means, it is possible to remove from the stomach a large part of the poison. The hope of being able to do this is the more justified the shorter the time that has elapsed since the introduction of the poison. In the case of *morphin* the prognosis need not be a grave one even when the fatal dose has been injected, because a rapid excretion of the poison takes place through the gastric mucous membrane and thereby making successful treatment possible.

Of the various symptoms having an important bearing upon the prognosis, *Facies hippocratica* and *œdema* of the lungs are considered the most unfavorable. Yet it must be remembered that these symptoms may often be relieved. Cold sweat is generally a very unfavorable sign, whereas convulsions and spasms are very often borne better than would be supposed. Coma may be made to disappear even if it has existed for hours. Cessation of the pulse, when respiration continues, even though much weakened, and, *vice-versa*, the cessation of respiration, with continuance of the pulse, are not absolutely unfavorable signs, for there have been reported cases of recovery where artificial respiration had to be practiced for from one to two hours, as well as other instances in which the pulse could be neither felt nor heard for from 30 to 60 minutes. A prognosis favorable as to health, should never be given early in the case, for a great many intoxications from *carbon monoxid*, *arsenic*, *lead*, *phosphorus*, *cantharides*, etc., are by no means entirely cured when the acute symptoms have been relieved and the patient already considers himself entirely recovered. These poisons may give rise to very severe later complications.

VII.—Treatment of Poisoning.

The simplest and most natural classification of the means concerning us here, is into physical (mechanical), antidotal and symptomatic treatment. Prophylaxis may, in a certain sense, be considered as an additional measure.

I. The Physico-mechanical treatment comprises the following manipulations:

1. Removal of the poison from wounds by pressure, suction, washing, cauterization.

2. Removal of the poison from the stomach by means of emetics, syphon, or stomach pump, followed by washing of the stomach.

3. Removal of the poison from the intestine by means of catharsis, or rectal flushing.

4. Reduction of the absorptive power by ligature (in bites and stings of poisonous animals) and immobilization of the extremity.

5. Artificial respiration in cases where the breathing ceases, but the upper air passages remaining free.

6. Intubation or tracheotomy in cases where the laryngeal entrance is swollen, as often occurs after drinking corrosive sublimate, acids and alkalies.

7. Electric excitation of the phrenic nerve to stimulate breathing, or irritation of the soles of the feet with the electric brush to restore lost reflexes.

8. Application of cold in the form of the ice bag, cold compresses, douches, etc.

9. Application of heat in the form of hot water bags and warm baths, in cases of poisons, such as narcotics, which greatly reduce temperature.

10. Passive motion of the extremities to stimulate circulation.

11. Massage, particularly in the form of stroking in the direction of the venous flow to stimulate circulation.

12. Brushing and rubbing the skin to raise its temperature and to excite reflex activity.

13. Low position of the head when there is a tendency to fainting.

14. Administration of ice to alleviate the symptoms of inflammation caused by corrosive poisons.

15. It is often necessary to catheterize the bladder, especially in *morphin* poisoning; in many cases this is desirable in order to confirm the diagnosis.

16. Blood-letting, followed by transfusion of human blood or infusion of salt-sugar solution (7.5 grms. of sodium chlorid and at least 20.0 grms. of cane sugar to a liter of boiled water). In cases where there is reason to suppose that a diminished alkalescency of the tissue-juices and of the blood obtains, we can add to this solution some alkali (*e. g.*, 1.0 grm. sodium carbonate).

17. Hot iron, Paquelin's cautery, and the galvano-caustic apparatus, for burning out poisoned wounds.

II. Unfortunately the **antidotal treatment** is but too often nothing but symptomatic, as the poison is by no means rendered completely inactive by the administered antidote. Nevertheless, the physician should thoroughly acquire the contents of the following table, by virtue of which knowledge he will be often enabled to do good. Every medical man should have always prepared, in a case by themselves, everything necessary for the antidotal treatment of poison cases. Such an outfit will be discussed at the end of the present chapter.

I.—TABLE OF THE MOST IMPORTANT ANTIDOTES.

NO.	ANTIDOTES	SUITABLE FOR THE FOLLOWING POISONS:
1	Ferric hydrate and (or) magnesic hydrate	Arsenious oxid, arsenic compounds in general
2	Inactive oxygen, inhaled under pressure	Carbon monoxid, hydrocyanic acid, potassium cyanid, potassium chlorate, cairin, anilin, phosphorus, arsin, nitrobenzene, pyrogallol, chloral hydrate, morphin
3	Hydrogen peroxid, given in solution or injected subcutaneously	Phosphorus, hydrocyanic acid, carbon monoxid
4	Egg albumen stirred under water	Corrosive acids, corrosive salts, corrosive alkalies

NO.	ANTIDOTES	SUITABLE FOR THE FOLLOWING POISONS:
5	Olive oil	Lead colic, corrosive poisons
6	Milk	Corrosive salts
7	Milk, gelatine, glue, vegetable mucilaginous drinks of agar-agar, gum arabic, tragacanth, althea, saloop, mallow, linseed, poppy seeds, starch	Corrosive substances
8	Animal charcoal (better than vegetable charcoal), tannic acid, decoction of oak bark, walnut leaves, etc.	Alkaloids, glycosids, salts of metals
9	Infusion of black coffee or Chinese tea	Alkaloids, especially morphin, glycosids and salts of metals
10	Iodin in solution of potassium iodid	Alkaloids, glycosids
11	Potassium iodid	Lead (chronic poisoning)
12	Sodium hyposulfite	Iodin or potassium iodid, chlorin, Javelle water
13	Chlorin in the form of aqua-chlori, calcaria, chlorata and natrium hypochlorosum	Externally in poisoned wounds, against alkali-sulfids in gaseous form (exceedingly dangerous)
14	Chromic acid	Externally in poisoned wounds
15	Potassium permanganate	Phosphorus, hydrocyanic acid, opium, morphin, strychnin, physostigmin, coronillin; externally in poisoned wounds
16	Sugar-lime (viz., a solution of slaked lime and sugar, in water)	Oxalic acid, acid potassium oxalate ($C_2O_4HK + C_2O_4H_2 + 2\ H\ O$), carbolic acid
17	Glauber's salt	Carbolic acid, baryta, lead salts (acute)
17	Blue vitriol	Phosphorus, baryta
18	Atropin sulfate	Muscarin, pilocarpin, arecolin, physostigmin, morphin, lead (colic), baryta (colic)

NO.	ANTIDOTES	SUITABLE FOR THE FOLLOWING POISONS:
19	Morphin hydrochlorid	Atropin (in cerebral irritation)
20	Pilocarpin hydrochlorid	Atropin, hyoscyamin, scopolamin, homatropin, poison of uraemia
21	Chloral hydrate, curarin	Strychnin, picrotoxin, cicutoxin, digitaliresin, toxiresin, poison of the tetanus bacillus, ammonia
22	Strychnin	Chloral hydrate, alcohol
23	Sodium sulfanilate	Iodin
24	Sodium carbonate	Iodin, corrosive acids, acid metal salts
25	Mixture of sodium carbonate and bicarbonate injected into vein	Beta-oxy-butyric acid (diabetic coma), potassium chlorate, cairin, anilin, arsin, nitrobenzene, phosphorus, any acid
26	Tartaric acid, malic acid	Corrosive alkalies and corrosive alkaline earths
27	Magnesia carbonica and usta	Corrosive acids, acid salts
28	Inhalation of ammonia	Morphin, chloral hydrate, sulfonal, alcohol
29	Inhalation of chloroform	Strychnin, picrotoxin, cicutoxin, poison of tetanus bacillus, ammonia
30	Injection of ether. Camphor, digitalein	Chloral hydrate, morphin, sulfonal
31	Potassium ferrocyanid	Copper, strychnin
32	Scopolamin	Cannabinon, delirium tremens

II.—TABLE OF THE MOST IMPORTANT ANTIDOTES ARRANGED IN THE ORDER OF POISONS TO BE COUNTERACTED.

NO.	POISONS.	ANTIDOTES.
1	Acid metal salts, corrosive acids	Sodium carbonate
2	Acid salts, corrosive acids	Magnesia carbonica and usta

NO.	POISONS.	ANTIDOTES.
3	Acid potassium oxalate ($C_2O_4HK + C_2O_4H_2 + 2H_2O$)	Sugar-lime, viz., sugar + slaked lime dissolved in water
4	Alcohol	Strychnin, inhalation of ammonia
5	Alkaloids, glycosids, salts of the heavy metals and of alkaloids	Animal charcoal, better than vegetable charcoal; tannic acid; decoctions of oak bark, walnut leaves, etc.; infusion of black coffee or Chinese tea (especially for morphin alkaloids, etc.)
6	Alkaloids, glycosids	Iodin in solution of potassium iodid
7	Alkali-sulfids	Chlorin in the gaseous form, for inhalation (very dangerous)
8	Ammonia	Chloral hydrate, curarin, inhalation of chloroform
9	Anilin, arsin	Inactive oxygen administered under pressure; mixture of sodium carbonate and bicarbonate injected into vein
10	Any acid	Mixture of sodium carbonate and bicarbonate injected into vein
11	Arecolin	Atropin sulfate
12	Arsenious oxid, etc.	Ferric hydrate and (or) magnesium hydrate mixed with water
13	Arsin	Inactive oxygen administered under pressure, mixture of sodium carbonate and bicarbonate injected into vein
14	Atropin	Morphin hydrochlorid (in cerebral irritation), pilocarpin hydrochlorid
15	Baryta	Glauber salts, blue vitriol, atropin sulfate (colic)
16	Beta-oxy-butyric acid	Mixture of sodium carbonate and bicarbonate injected into vein
17	Cairin	Inactive oxygen administered under pressure, mixture of sodium carbonate and bicarbonate administered into vein
18	Cannabinon, delirium tremens	Scopolamin

NO.	POISONS.	ANTIDOTES.
19	Carbon monoxid	Hydrogen peroxid, given in solution or injected subcutaneously
20	Carbolic acid	Sugar-lime, Glauber salts
21	Chloral hydrate	Strychnin, camphor, digitalein, inhalation of ammonia, injection of ether
21A	Chloral hydrate, carbon-monoxid	Inactive oxygen administered under pressure
22	Chlorin	Sodium hyposulfite
23	Cicutoxin	Chloral hydrate, curarin, inhalation of chloroform
24	Copper	Potassium ferrocyanid
25	Coronillin	Potassium permanganate
26	Corrosive substances—corrosive acids, alkalies and salts	Vegetable mucilaginous drinks of starch, agar-agar, gum arabic, tragacanth; althea, saloop, mallow, linseed, poppy seeds, gelatin, glue, egg albumin stirred under water; olive oil, milk (for corrosive salts); sodium carbonate, magnesia carbonica and usta (for corrosive acids); tartaric and malic acids (for corrosive alkalies and alkaline earths)
27	Delirium tremens, digitaliresin	Chloral hydrate, curarin
28	Diabetic coma	Mixture of sodium carbonate and bicarbonate injected into vein
29	Hydrocyanic acid	Inactive oxygen administered under pressure, hydrogen peroxid given in solution or injected snbcutaneously, potassium permanganate
30	Hyoscyamin, homatropin	Pilocarpin hydrochlorid
31	Iodin	Sodium sulfanilate and sodium carbonate, sodium hyposulfite
32	Javelle water	Sodium hyposulfite

NO.	POISONS.	ANTIDOTE.
33	Lead	Atropin sulfate, olive oil (colic), potassium iodid (chronic); Glauber salts (acute from salts)
34	Morphin and opium	Inactive oxygen administered under pressure, potassium permanganate, atropin sulfate, inhalation of ammonia, injection of ether, camphor, digitalein
35	Muscarin	Atropin sulfate
36	Nitrobenzene	Inactive oxygen administered under pressure, mixture of sod. carb. and bicarb. intravenous
37	Oxalic acid	Sugar-lime
38	Physostigmin	Potassium permanganate, atropin sulfate
39	Phosphorus	Inactive oxygen administered under pressure, hydrogen peroxid given in solution or injected subcutaneously, potassium permanganate, blue vitriol, mixture of sodium carbonate and bicarbonate injected into vein. (The old antidote was oil of turpentine.)
40	Picrotoxin	Curarin, chloral hydrate, inhalation of chloroform
41	Pilocarpin	Atropin sulfate
42	Poisoned wounds	Externally: potassium permanganate, chlorine in the form of aqua-chlori, calcaria, chlorata and natrium hypochlorosum, chromic acid
43	Potassium chlorate	Inactive oxygen administered under pressure, mixture of sodium carbonate and bicarbonate injected into vein
44	Potassium cyanid, pyrogallol	Inactive oxygen administered under pressure
45	Potassium iodid	Sodium hyposulfite
46	Scopolamin, uraemic poisoning	Pilocarpin hydrochlorid

NO.	POISONS.	ANTIDOTES.
47	Sulfonal	Inhalation of ammonia, injection of ether, camphor, digitalein
48	Strychnin	Potassium permanganate, chloral hydrate, curarin, inhalation of chloroform, potassium ferrocyanid
49	Tetanus bacillus	Chloral hydrate, curarin, inhalation of chloroform
50	Toxiresin	Chloral hydrate, curarin

III. The prophylactic treatment is very largely that of hygiene and is the province of the Board of Health.

1. Articles of food, which are either decayed, or adulterated with poisons, should be confiscated.

2. Poisonous plants and animals should be exterminated, and a thorough knowledge of them taught in the schools.

3. Manufacturing, which is likely to lead to the poisoning of the employed, should be prohibited, or at least the surroundings of the men who work at such industries rendered as healthful as possible.

4. The use of poisonous dyes for wall-paper, clothes, toys, food-stuffs and all sorts of utensils, should be prohibited.

5. The entrance into dwelling places of poisonous gases from sewers, water-closets, gaspipes, stoves, etc., should be prevented by sanitary regulations.

6. The dangers of certain articles of diet should be kept constantly in the mind of the public by frequent appropriate illustrations in the churches, schools, at home, &c.

7. Antidote cases, ready for use, should be kept in various quarters of the town, in village hospitals, &c.

The Antidote Case.

Next to the choice of the proper remedy, the success of the treatment of a case of acute poisoning depends chiefly upon *how promptly the antidotal treatment is begun.* Hence, it is the duty of every conscientious physician to have in constant readiness, a case containing everything necessary for coping

with accidents &c., caused by poisoning. This will enable him to hurry to the scene of accident at a moment's notice. However, this is only possible when there has been made a careful, compendious selection of the necessary articles, as otherwise the volume and weight of the armamentarium would not admit of ready and comfortable transportation. For each physician to prepare such a case according to his own ideas, would necessitate the expenditure of a great deal of thought, time and money. A much more economical plan would be for a number of physicians to arrange at the same time to supply themselves with such cases, and for this reason the author has described an outfit which is prepared and put upon the market by Ehrhardt & Metzger, of Darmstadt. Whether the physician uses this or any other case, the contents should be practically the following:

In the first place, a small book on poisoning, such, *e.g.*, as the present, or a better one.

Apparatus for chloroforming, for subcutaneous injection, for washing of the organism (salt-sugar transfusion), for washing the stomach, for opening the mouth and holding it so, for retracting the tongue, for auscultation, for external friction (Luffa-sponge), for catheterizing; and for performing a few simple chemical tests (sugar in the urine, Froehde's reaction for morphin, etc.), a medicine glass, graduated cylinder and filter paper are necessary.

As for *instruments*, it is desirable to have a few forceps, including artery-forceps, a scalpel, for blood-letting, etc., and a few needles; and in the line of *bandaging material*, silk, sponges, cotton and gauze bandages.

Of *medicines* and *reagents*, some should be in solution and some in solid form.

As regards the solutions we need, first, sterilized solutions for subcutaneous injection, in small, sealed glass tubes, especially of apomorphinum hydrochl. 0,01 : 1,0, atropinum sulfuric 0,001 : 1,0, curarinum 0,01 : 1,0, digitaleinum 0,01 : 1,0, diuretinum lithio-benzoic, 0,2 : 1,0, morphin hydrochl. 0,02 : 1,0 and 0,03 : 1,0, pilocarpinum hydrochl. 0,01 : 1,0, scopolaminum hydrobrom. 0,0005 : 1,0, strychninum nitricum 0,005 : 1,0.

Of other fluids necessary, we mention liquor ferri sulfurici oxydati (for the preparation of the antidote for arsenic), chloroform, olive oil, cognac or whiskey, ether, ether aceticus, saturated hydrogen peroxid solution, liquor ammonii caustici, liquor ammonii acetici, spirits of turpentine, spirits of camphor, acetic, hydrochloric, and nitric acids, concentrated sulfuric acid, liquor ferri sesquichlorati, phospho-tungstic acid, phospho-molybdic acid, mercuric-iodid in potassium-iodid, tincture of guaiac, Fehling's solution, solution of iodin in potassium iodid (1,0 pot. iod. + 0,1 iodin + aq. dist. ad. 10,0), liquor plumbi subacetici, solution of argentic nitrate, solution of sodium sulf-anilate, sugar-lime solution and distilled water.

Of substances in the form of powder, there should be on hand calcium carbonicum praecipitatum, magnesium sulfuricum, natrium sulfuricum, barium chloratum, tragacanth, agar-agar, starch, citric acid, tartaric acid, malic acid, magnesia usta ponderosa, magnesium carbonicum, ground roast coffee, cuprum sulfuricum, acidum picro-nitricum, potassium ferrocyanid, sodium molybdaenicum, plumbic acetate, powders of tartar emetic 0,1 + rad. ipecac. 1,0, others of calomel 0,5 + tub. jalap. 1,0, and lastly a mixture of tub. jalap 10,0 + cream of tartar 20,0.

In the line of aseptic, compressed tablets for the purpose of preparing solutions for subcutaneous injection, it is preferable to have those which correspond to the solutions mentioned above. Of other compressed powders and the like we mention such of charcoal, salt, potassium permanganate, sulfonal, potassium chlorate, tannin, soda, sodium bicarbonate, cathartic acid; cough powders, opium with extr. ligni. camp.; sublimate and salt; calomel and jalap; opium and pulvis gummosus; ammonium-chlorid and licorice; and then, for washing the organism, sugar 2,0 + salt 0,75 + potassium carbonate 0,05, + sodium carbonate 0,05. Ten of these powders in a liter of boiled water, make a solution suitable for introduction into the veins, which, however, must be filtered before using.

Sundry other substances to be considered are: suppositories for checking diarrhœa, for purging and for producing a

sedative effect; adhesive plaster, &c., mustard paper, litmus paper, chloral capsules, lanolin salve, paraffin salve, &c.

In addition, the larger hospitals should keep in readiness means for stimulating the phrenic nerve, for performing tracheotomy, for inhalation of oxygen, a spectroscope (for blood, &c.), a laryngoscope (in case of œdema of the glottis), as well as larger handbooks on poisons.

VIII.—Pathological Demonstration of Poisoning.

There are many cases of poisoning which cannot as yet be pathologically demonstrated, because the changes are not such as to be seen at a glance, but where resort must be had to the microscope. For years we have endeavored to bring this science, "pharmacopathology," into renown with the medical profession. We have been able, partly by ourselves and partly in conjunction with the gentlemen working in our institute, to show the pharmaco-pathological changes in poisoning with sodium oxalate, iron, manganese, chromium, silver, copper, quillaic acid, sapotoxin, cyclamin, senegin, sodium crotonolate, podophyllotoxin, ergot, &c., or to study more closely those changes already known, and to represent them by diagram. The characteristic changes from which we are often able to conclude as to the nature of the poison, are found in the kidneys, liver, spleen, heart, blood, intestinal canal and lungs. But all of these changes are characteristic only when an early autopsy is made. Unfortunately, the physician has no right to make a post-mortem examination in a case of poisoning directly after death; he is obliged to wait until an inquest is held. In this way the most valuable time is generally lost, so that the various organs are usually not fit for microscopical examination. The physician should, however, do all in his power to have the autopsy held as soon as possible.

Coroner's Inquest.—The conduct of a coroner's inquest, the manner of holding the same, taking testimony, procuring services of experts, &c., is, in the U. S. of America, entirely in the discretion of that official. Chemical analyses may be performed at such time and place as the chemist chooses. All the world may attend the inquest.

Regarding the bacteriological examination, it must not be forgotten, that even "normal" cadavera contain micro-organisms other than those in the intestinal canal; of these we mention bacillus lactis aërogenes, bacillus coli communi, proteus vulgaris, proteus Zenkeri, bacillus albus cadaveris, bacillus citreus cadaveris, &c. It is not yet known whether poisons favor the development of certain microbes of importance in diagnosis.

With reference to the chemical examination, it is important to remember that the cadavera of man and of all other mammals first enter a stage of acid reaction, and then, by disintegration of the nitrogen-containing component of albumin into bases, undergo alkaline reaction. The latter makes itself known by the ammoniacal odor. Independently, the sulfur of the albuminous substances is gradually transformed into simpler combinations, and finally is converted into hydrogen sulfid or ammonium sulfid, and is then detected by the odor. Some poisons favor this change, others retard it. Hydrogen sulfid converts the haemoglobin of the blood into sulf-methaemoglobin and, finally, ferrous sulfid is produced. Both of these give to the corpse a green color. Mummification and the formation of adipocire are two remarkable changes which may take place in normal corpses as well as in those due to poisoning. The chemistry of these changes has, as yet, not been sufficiently investigated. By decay, we understand a transformation which takes place in corpses resting in a soil containing atmospheric air. This transformation, which takes place in corpses of non-poisoned as well as in those of poisoned individuals, does not give rise to bad odors, since it is combined with oxidation, the final products being: water, carbon di-oxid, nitrates, sulfates and phosphates.

The following table contains some of the most conspicuous changes in the cadaver. It will facilitate the recognition of conditions in the autopsy:

TABLE OF PATHOLOGICO-ANATOMICAL FINDINGS, CHARACTERISTIC IN CERTAIN CASES OF POISONING.

NO.	FINDING:	POINTS TO POISONING BY:
1	Defluvium capillorum in non-syphilitic individuals	Arsenic, when the course is not very acute
2	Green discoloration of hair	Copper, in chronic cases
3	Contraction of pupils still present at time of autopsy	Physostigmin
4	Marked dilation of pupils still present during autopsy	Atropin, scopolamin, hyoscyamin
5	Extensive papular, pustular, or ulcerative changes of the skin	Corrosive acids, alkalies, chromates, carbolic acid, iodin, bromin, ergot
6	Atrophy of muscles	Lead, arsenic, ergot, poison of Lathyrus
7	Griffin-claw (Greifenklaue)	Ergot, administered some time previous to death
8	Icteric and pseudo-icteric discoloration of the skin	See page 17, No. 34
9	Skin shows black patches	Silver, arsenic
10	Post-mortem spots of markedly bright red color	Hydrocyanic acid, potassium cyanid, carbon monoxid
11	Multiple hæmorrhages into the skin	Phosphorus
12	Cadaver markedly well preserved, or mummified	Arsenic
13	Dry gangrene of some of the limbs	Sphacelinic acid
14	Corrosion of lips and corners of mouth	Corrosive alkalies, corrosive acids, free haloids, corrosive salts
15	Perforation of nasal septum in non-syphilitic individuals	Chromates (protracted action in the form of dust)
16	Perforation of frenulum praeputii in persons not suffering from genito-urinary diseases	Chromates (protracted action on the epidermis)

NO.	FINDING:	POINTS TO POISONING BY:
17	Dark line on the gums	Lead, mercury, silver, bismuth
18	Stomatitis, glossitis and loose teeth	Mercury, bismuth
19	Necrosis of jaw	Phosphorus (inhaled)
20	Gastro-adenitis	Phosphorus, arsenic, antimony
21	Mineral particles in gastro-intestinal canal	Arsenious oxid, metallic arsenic, arsenious sulfid, antimony, antimonious sulfid, calomel, mercuric oxid, mercuric iodid, chromium preparations
22	Green shining particles	Cantharides
23	White crystals of the appearance of flour, solidly attached to the walls of the intestine	Oxalic acid, acid potassium oxalate
24	Small pieces of wood in the gastro-intestinal canal	Phosphorus (matches)
25	Leaves of a dicotyledon in the gastro-intestinal canal	Hyoscyamus, Datura stramonium, Atropa Belladonna, Nicotiana tabacum, Aconitum napellus
26	Fragments of hair-coated seeds in the gastro-intestinal canal	Strychnos nux vomica (bachelor-buttons)
27	Seeds or fragments of non-hair-coated seeds in gastro-intestinal canal	Cytisus-laburnum, Hyoscyamus, Datura stramonium, Ricinus
28	Particles of plant belonging to the coniferae in the gastro-intestinal canal	Sabina, Thuja, Taxus
29	Particles of mushrooms in the gastro-intestinal canal	Amanita muscaria, Agaricus phalloides, Helvella
30	Cavities of the body and entrails presenting the odor of garlic	Arsenic, tellurium, ether bromatus (ethyl-bromid or hydro-bromic ether), phosphorus
31	Cavities of the body and entrails pervaded by odor of bitter almonds	Hydrocyanic acid, potassium cyanid, nitrobenzene

NO.	FINDING:	POINTS TO POISONING BY:
32	Cavities of the body and entrails smell like spirits of turpentine	Sabina, Thuja, Taxus, Ruta
33	Cavities of the body and entrails have a pleasant odor	Eau-de-cologne, perfumed cordials, amyl-nitrite
34	Cavities of the body and entrails smell of acetone	Ethyl-di-acetic acid (ethyl-aceto-acetic ester), formed in diabetes
35	Cavities of the body and entrails have other peculiar odors	Alcohol, ether, amylene, amylene-hydrate, chloroform, camphor, anilin, carbolic acid, nicotin, iodin, bromin, chlorin, hydrogen sulfid, ammonia, hydrochloric acid, acetic acid, opium
36	Contents of stomach and intestine luminous in the dark	Phosphorus
37	Walls and contents of gastro-intestinal canal markedly acid	Acids, acid-salts
38	Walls and contents of gastro-intestinal canal markedly alkaline	Potassium cyanid, caustic alkalies, alkaline earths
39	Walls and contents of the gastro-intestinal canal turn black on touching with ammonium sulfid, or on exposure to its vapors	Lead, bismuth, mercury, copper
40	Villi of small intestine turn black spontaneously	Silver
41	Walls and contents of the upper intestinal canal of a yellowish color	Nitric acid, picric acid, picrates, plumbic chromate
42	Walls and contents of the upper intestinal canal of a greenish color	Scheele's green, Schweinfurt green (Paris green), cupric sulfate, verdigris
43	Walls and contents of the upper intestinal canal of a brownish color	Iodin, bromin, potassium chromate, phosphorus (icteric)
44	Walls of large intestine dark colored	Bismuth

NO.	FINDING:	POINTS TO POISONING BY:
45	Lower portion of small intestine and the entire large intestine dysenteric	Mercury, arsenic, antimony
46	The entire intestine dysenteric	Castor beans (Ricinus)
47	Multiple hæmorrhages into the gastro intestinal canal	Phosphorus, arsenic, baryta
48	Fatty liver	Phosphorus, phallin, Poley-oil, arsenic, antimony, ammonia, iodin
49	Fatty degeneration of heart, kidneys, and muscles	Phosphorus, phallin, arsenic, antimony, ammonia, iodin
50	Kidney produces a grating sound on cutting	Mercury, oxalic acid, oxamid, lead (in chronic cases)
51	The glomeruli appear as black spots upon the cut surface of the kidney	Silver (in chronic cases)
52	Glomerulo-nephritis	Cantharidin, iodin, osmic acid and its salts
53	Even macroscopically, the kidney shows reddish brown cylinders	Corrosive acids, corrosive alkalies, corrosive salts, phallin, cyclamin, helvellaic acid, potassium chlorate, etc.
54	Liver, on cut surface, shows black discoloration of the connective tissue and vessels	Silver (in chronic cases)
55	Pulmonary oedema	Muscarin, pilocarpin, nicotin, morphin
56	Diseased condition of the spinal cord	Ergotism, lathyrism, pellagra

When preserving blood for spectroscopic examination, we must remember that some blood changes, as, for example, the formation of methaemoglobin, are very transient, therefore, this examination must be made at once, if, indeed, we wish to draw conclusions from methaemoglobin formation. Other changes, such as the presence of carbon monoxid-haemoglobin,

which can be detected by the spectroscope, may be preserved by excluding the air from the blood as completely as possible. This result is most readily obtained by sealing the blood in glass tubes.

The more detailed examination of particles of poison and of the suspicious contents of the gastro-intestinal tract, is left in the hands of the chemist. However, the coroner can order that the examination be made under the supervision, or with the assistance, of a physician. But, as a rule, this is not done. Nevertheless, the physician is often enabled to draw important conclusions as to the nature of the poison at the time of the autopsy, even when there are present no extensive anatomical changes and without the aid of finer chemical tests. To this end he must direct particular attention to the points given in the above table, during the examination of the vomit, and of the remaining particles of food-stuff, and during the autopsy.

IX.—Chemical Detection of Poisoning.

I.—GENERAL REMARKS ON THE CHEMICO-LEGAL EXAMINATION.

The detection of a poison by chemical means has, naturally, great weight as evidence, since the actual finding of the "*corpus delicti*" more completely establishes the fact of introduction of the poison into the body than could anything else. However, this actual finding of the poison does not prove, by any means, that death, in any particular case, was caused by it. For, in the first place, we have to consider that most poisons serve as medicines; secondly, the possibility of introduction of the poison post mortem, with the intention of exciting suspicion. In this latter case, the anatomical changes, peculiar to the poison in question, will be wanting. Besides, in such a case, the poison will have penetrated through the walls of, and into neighboring organs, in obedience to physical laws, and will not have been carried by the blood to remote organs. However, in cases where artificial respiration had been practiced for some length of time, or where the corpse had been moved about to a great extent, the poison, introduced post mortem, may reach the most remote organs within a comparatively short time. Therefore, as a

rule, the detection of a poison is of value as conclusive proof only, when the symptoms during life, and the change found in the autopsy correspond to those, typical of this particular poison. On the other hand, this detection is not absolutely necessary; that is to say, we are by no means justified in considering the question of poisoning as dismissed, because the expert failed to find any poison at all. The poison may have been voided by vomiting or by the organs of excretion; it may have decomposed or oxidized (*e. g.*, phosphorus). In such instances we would find nothing, or merely chemically altered products, none of which would be of much value as conclusive evidence (*e. g.*, phosphoric acid, which occurs normally in every part of the body). We mention further the difficulty of research, which obtains in case of some organic substances, used in minimal quantities, which call for the exercise of the greatest caution and skill upon the part of the expert. He confronts the utmost difficulties. He must separate the poison from a large bulk of organic material, in which he must search for it, and obtain it in a pure condition, to be valuable as proof.

An especial difficulty is caused by the fact, that in corpses of non-poisoned individuals, substances may spontaneously form, which are of greatest chemical similarity to our most poisonous alkaloids. This is true to such an extent, with reference to the general group reagents, that those poisons have not only been mistaken for true alkaloids in the past, but this error is scarcely avoidable even at present. These substances we call, following the procedure of the late Italian chemist, SELMI, "ptomaines." (The word should rather be: "ptomatines," as grammatically the correct, from πτῶμα, πτώματος). The difficulty is still further increased, since these substances may give rise to errors due, not only to their chemical behavior, but also concerning the pharmacological action, as some of them are exceedingly poisonous, and in action closely resemble vegetable poisons. According to BRIEGER, one of them, the cadaver-muscarin, is identical with the muscarin of Amanita muscaria, both in chemical composition and in its action upon the glands, heart and pupil.

In doubtful cases of poisoning by vegetable poisons, it is consequently imperative to prove the presence of the poison,

not merely by chemical means, but additionally, by the pharmacological experiment.

Here the situation obtains, for which the German law provides, that the chemical investigation should be made in conjunction with a physician (specialist in pharmacology) for the purpose of physiological detection of the poison. It is possible to conclude with certainty, that the deceased succumbed to a particular poison, only, when the chemist and the pharmacologist arrive at the same diagnosis, and when this diagnosis corresponds with the symptoms, observed during life, by the attending physician.

It cannot and it should not be considered the duty of the physician to make the purely chemical investigation of remnants of food, of the vomit, of ejections, of the contents of the stomach and intestine. But he should know the course of analysis so as to be able to understand and, possibly, to criticise the testimony of the chemist. We shall, therefore, discuss this important topic somewhat minutely in a later chapter.

The *vessels* in which the substances are handed over to the expert chemist, must be well closed and also sealed. The examination must be made in a room used, for the time being, for this analysis exclusively, and accessible only to the chemist. All necessary *apparatus* and *utensils* must be chemically pure. It has frequently happened that poison, chiefly *arsenic*, was introduced into the material by the reagents used. All reagents must therefore be tested particularly in regard to their purity. The assurance that they were purchased as "chemically pure" does not suffice. The precautions observed to prevent such an introduction of poison during the course of analysis, must be especially mentioned in the official report of the chemist.

Fortunately, we have at our disposal means and ways of forensic chemical analysis for the detection of all conceivable poisons. It is desirable, in order to test for all of these, to divide the suspicious material into five portions. I, serves for the detection of volatile substances; II, for acids and alkalies; III, for alkaloids and glycosids; IV, for metals; and V, is reserved in case of accidents or need. One portion may be used for different purposes, *e. g.*, detection of volatile matter

and metals. Lastly, we should also have a sixth portion to use for the purpose of making so-called preliminary tests.

It is desirable, and the law of certain countries prescribes for all cases of forensic chemical research, that the poison be presented to the judge and the jury in a permanently stable condition, capable of impressing the senses. Forensically this is called the *corpus delicti.* Of such we mention the platinum double-salts of *ammonia* and of *alkaloids,* Prussian blue from *hydrocyanic acid,* mirrors and spots of *arsenic* and *antimony, phosphorus* as such, *mercury* in the form of red mercuric iodid, *oxalic acid* as calcium oxalate, fragments of the elytra of *cantharides,* seeds of *Hyoscyamus,* hulls of berries of *Atropa Belladonna,* leave-fragments of *Digitalis purpurea,* &c. We will never be able to obtain the total quantity of the poison from the cadaver, for the reason that this would necessitate working the latter up in its entirety.

II.—THE SO-CALLED PRELIMINARY TESTS.

The table p. p. 33-36 contains some points to be considered here, such as: reaction, identification of vegetable fragments, of elytra of cantharides, fragments of *arsenic,* &c. Without detriment to the material, the physician in charge of the case may establish to a degree some of the same facts that will be afterwards ascertained by the chemist. It is left to his good will as to whether or not he will make a test by applying his tongue to some of the material; the chemist must do this.

A further test with reagent-paper, applicable by the physician at the time of death, consists in exposing to the vapors arising from the contents of the stomach, a piece of reagent-paper moistened with newly prepared guaiac-tincture and cupric sulfate, in a closed bottle, at the temperature of the room. If this is turned blue, the suspicion of the presence of *hydrocyanic acid* is justified. A strip of plumbic acetate-paper and another one of argentic nitrate are consecutively introduced into the bottle and fastened between the neck and stopper. The bottle is gently warmed by dipping it into warm water. If both strips are blackened, *hydrogen sulfid* or *ammonium sulfid* are present, which of course may have been

produced by putrefaction. If only the silver-paper is blackened, *phosphorus* is present.

There are two additional preliminary tests to be considered by the chemist, that by means of metal strips and the dialytic one.

The Test by Means of Strips of Metal.—The contents of the stomach and intestines, or the disintegrated organs are finely distributed in water, and the faintly acidulated magma is introduced into four vessels. Into these we put bright plates of the following kind: into the first, *zinc*; second, *iron*; third, *copper*; fourth, a *zinc-platinum*-couple. The zinc turning black indicates a likelihood that a *metal* is present; if the iron turns red, *copper* is indicated; if the copper receives a silvery white coating, this shows the presence of *mercury*. If the platinum turns black, *antimony* is present. If the copper did not change and we proceed to heat it in the liquid, acidulated with hydrochloric acid, it may turn gray; this shows the presence of *arsenic*. The gray film dissolves on warming in ammonia, giving a solution that allows of the detection of copper-arsenid.

The Test by Dialysis, invented by GRAHAM, depends upon the fact that all crystalloids will pass comparatively readily through parchment-paper into distilled water, and thus allow of a separation from albumin, mucin, glue, blood-coloring matter, &c. Poisons, as *strychnin*, *morphin*, *atropin*, have thus frequently been found, quickly and surely.

III.—SUBSTANCES THAT MAY BE DETECTED BY MEANS OF DISTILLATION.

In case the material to be examined is strongly alkaline from the outset, the substances are directly distilled, furnishing *ammonia* and the so-called *higher ammonias* (*methylamin*, *ethylamin* and the *diamin-bodies*, &c). *Nicotin* and *coniin*, which under these circumstances may also distil off, at higher temperature, we generally test for when approaching the alkaloids. We shall later refer to *anilin*, which may be found at this stage. If the alkaline reaction was caused by *potassium cyanid*, the decomposing influence of carbon dioxid of the air will cause the passage of some *hydrocyanic acid* into the distillate.

If chloral hydrate was present, the decomposing action of strong fixed alkalies will give us chloroform in the distillate.

The following table contains a sketch of the most important substances which can be separated by means of distillation. (The abbreviation ppt. signifies: precipitate.)

TABLE OF VOLATILE POISONS SEPARATED BY DISTILLATION.

I. From alkaline solutions we obtain: *ammonia*, the frequently occurring cadaver poisons, such as *amins*, *diamins*, etc., and *volatile alkaloids*.

II. From acid solutions, according to DRAGENDORFF, the following are obtained:

1. Vapors which pass over, colorless; distillate *neutral*.

(*a*) EASILY VOLATILE SUBSTANCES; ODOR OF ALCOHOL, ETHER, etc.

Vapors contain chloral. They furnish $CaCl_2$ when passed over glowing lime in a current of steam.	1. Distillate warmed with alcoholic KOH and anilin, productive of iso-nitril-reaction: **Chloroform.** 2. Distillate does not furnish isonitril: **Ethylene chlorid,** boiling point + 85° C., or **Arans Ether,** volatile at + 105° C.
The filtrate upon addition of iodin + KOH gives the iodoform reaction.	1. It has the odor of: **Alcohol.** 2. It has the odor of: **Ether.** 3. When heated with $Ba(OH)_2$ it furnishes barium-acetate: **Acetic ester.** 4. The iodoform precipitate is amorphous: **Acetone.**
The distillate smells of Benzene, Petroleum, etc. It consists of water and oily drops on top of it.	1. These furnish with HNO_3 Nitro-benzene: **Benzene.** 2. They furnish nothing: **Petroleum.**
The distillate smells of rotten eggs.	With acid solution of plumbic acetate, black ppt. **Hydrogen sulfid.** Will rapidly discolor a 1 p. c. blood solution.
The distillate is of fetid, alliaceous odor.	With NH_3 and sugar of lead solution it produces a black ppt. **Carbon-disulfid.** It will not change a 1 p.c. blood solution.

(*b*) DIFFICULTLY VOLATILE BODIES.

A larger amount must be distilled off; then agitate the distillate with ether.

Ether residue:

Smells of ethereal oil, and is **Liquid.**	1. Smells of: **Spirits of Turpentine, Oil of Rue, Oil of Ledum, Oil of Savin.** 2. Smells of: **Oil of bitter almonds.** (a) It will produce anilin when treated with Zn and H_2SO_4 **Nitrobenzene.** (b) It will not give this reaction, but when oxidized at the air produces benzoic acid: **Benzaldehyde (oil of bitter almonds).** 3. It has a pungent odor causing inflammation of eyes and nose: **Mustard-oil, garlic-oil.**
Smells of ethereal oil, and is **Solid.**	1. H_2SO_4 will color red, brown to violet: **Ledum-camphor.** 2. H_2SO_4 will produce a *red* color, only upon addition of acetic acid containing a trace of *iron:* **Thymol.** 3. H_2SO_4 produces *no* color: **Camphor.** 4. Of intensely yellow color, hexagonal plates; specific odor of: **Iodoform.** (*Bromoform*, in contradistinction to iodoform, is liquid, and can be distilled).
Smells of creasote...	1. With ferric chlorid + water, *blue violet:* free **Phenol.** 2. With Fe_2Cl_6 + alcohol, *green:* **Creasote.** Both substances, when applied to the epidermis, produce white spots and deaden the sensibility. The coupled phenol, *e.g*, of the liver, can not be directly obtained by this method; see p.
Is solid.....	It smells of chloral, and produces with KOH chloroform: **Chloral hydrate.** The main quantity of chloral, however, does not distil off.
Smells of fusel oil....	1. With H_2SO_4 *red:* **Amylic alcohol.** 2. *Not red* with H_2SO_4, but the odor persists: **Aroma of Rum, Arac, Cognac, &c.** 3. H_2SO_4 destroys the aromatic odor: **Essences of Rum, Arac, Cognac, &c., imitations.**

2. Distillate colorless, of *acid* reaction.

Paper moistened with guaiac tincture + Cu SO_4 is turned blue; curdy, white ppt. with $AgNO_3$	No blue coloration of guaiac paper, ppt. with $AgNO_3$:	Shaken with ether, furnishing crystals; these turn with ferric chlorid		Exactly neutralized, it will color ferric chlorid *brownish-red.* Silver solution	
		violet:	*yellow:*	is reduced	is not reduced
Hydro-cyanic acid.	**Hydrochloric acid.**	**Salicyclic acid.**	**Benzoic acid.**	**Formic acid.**	**Acetic acid.**

3. Distillate bleaches litmus, turns potassium-iodid-starch *blue:* **Chlorin, hypochlorous acid.**

4. Colored vapors—*Blue-violet:* **Iodin**; *orange:* **Bromin.**

5. Luminous vapors: **Phosphorus.**

IV.—ANALYTIC COURSE FOR THE ISOLATION AND IDENTIFICATION OF ALKALOIDS, GLYCOSIDS, BITTER PRINCIPLES, ETC.

For this purpose we have a series of methods, with which the physician should be familiar if he wants to get a proximate idea of the detection of the pertaining substances. In general, all of these methods have (*d.*) a desirable and (*n.d.*) a non-desirable collateral:

(*d.*) We find besides the alkaloids and glycosids in question, some substances, as *cantharidin*, *digitoxin*, *picrotoxin*, which do not, strictly speaking, belong here, but:

(*n.d.*) Frequently, however, we find most of the ptomaines, which is disagreeable to such an extent, that the detection of the poison really in question is often made a task of extraordinary difficulty.

1. Methods of Extraction. Before the detection proper of a poison is possible, it has to be extracted from solid masses or from mixtures forming a mash. Extractions of such a kind obtain in the research of leaves, barks, roots, poisonous bread, poisonous meat, and of organs, or of the contents of the intestines of poisoned individuals.

(*a*) **Boiling by water** is at the same time the simplest and most imperfect method, for the reason that many substances are insoluble in water, while a great number of others are decomposed by it; and furthermore, under this treatment human or animal organs are transformed into a pulp. However, as far as parts of plants are concerned, this method forms a satisfactory preliminary procedure.

(*b*) **Extraction by acidulated water,** cold or warm, is applicable if an alkaloidal poison is suspected, many free alkaloids being insoluble in water, while their salts are soluble. The acids most frequently used for this purpose are *hydrochloric-*, *sulfuric-* and *tartaric-acid.*

(*c*) **Extraction by alkaline water** is of use for obtaining a poisonous organic acid, since the alkali-salts of many acids are more soluble than these. Ammonia is used as a rule.

(*d*) **Extraction by alcohol,** warm or cold, is commendable mainly for vomit, contents of intestines and animal organs, since gelatin, albumin, coloring matter and many inorganic salts are the more insoluble in alcohol in proportion as this is stronger. The alkaloids are, as a rule, well dissolved by alcohol. An important advantage of alcoholic extraction is offered in the facility of ready separation of the extract from the alcohol by distillation.

(*e*) **Extraction by ether,** upon alkaline reaction, is an excellent method for alkaloids, which in the free state are, as a rule, readily soluble in ether. A type, such as SOXHLET'S apparatus is preferably used in this case.

(*f*) **Extraction by chloroform** or mixtures of chloroform and ether (with and without alcohol) must be considered in the case of substances which are insoluble in ether (*sapotoxin, quillaic acid, curarin*), and it is performed in the same manner as with ether. However, we have to consider that in order to evaporate the chloroform entirely, the extract has to be heated to a higher temperature than is the case when ether is used. The same apparatus may be here used as for ether.

2. Methods of Agitating (or Shaking) are to be considered in case we want to separate the poison from liquids. The advantage of these methods consists in obtaining the substances in a very pure state.

(*a*) **Method of Stas.** This refers to most of the alkaloids known thirty years ago, and to some glycosids (*digitalin*) and some indifferent substances (*picrotoxin*). STAS applied this method for the first time in 1851, at the occasion of the famous trial of "BOCARMÉ," and he succeeded by its use in detecting *nicotin* in the organs.

In short, this method is based upon the fact, that many alkaloids can be obtained from alkaline solutions, by agitating (shaking) these with ether, from which latter they can then be separated by evaporation or by shaking with acidulated water. Of substances incapable of isolation in this manner, we mention *morphin, narcein, muscarin, curarin, cytisin.* In case the mass to be analyzed consists of a pulp or mash (contents of intestines), or of an organ (liver), we must first extract the substance with *alcohol* containing *tartaric acid* and then evaporate the alcohol at + 35° C.

Hilger and **Kuester** have proposed to extract the masses to be analyzed, with water acidulated with *tartaric acid*, at + 50°-60° C, then to concentrate the filtrate, to add gypsum and desiccate the mass. The acid gypsum powder is extracted by means of ether in SOXHLET'S apparatus, rendered alkaline by sodium carbonate, dried again and extracted again with ether. It is claimed that under these conditions even *morphin* will pass into the solution and after some time deposit in crystalline form at the walls of the apparatus.

(*b*) **Method of L. von Uslar and I. Erdmann.** This is very similar to that of STAS, amylic alcohol being used instead of ether, the former dissolving alkaloids and glycosids in larger proportion.

(*c*) **Method of Fr. Julius Otto and Robert Otto.** This is an improvement upon and combination of the two preceding methods. In general, ether is used for shaking and afterwards warm, amylic alcohol for the isolation of *morphin* and *narcein*. This is a decided improvement as compared to method (*b*), since amylic alcohol is not particularly commendable for shaking purposes, in the first place because it strongly irritates the mucous membranes of the chemist; secondly, it contains, from the start, poisonous bases, and thirdly, it dissolves to a great extent ptomaines, which we do not want at all in the analysis.

(*d*) **The methods of Prollius, of Husemann and of Thomas** use chloroform as a means of shaking. If the extraction by means of chloroform was applied to parts of plants containing chlorophyll, this coloring matter would also be dissolved. In order to avoid this, the alcoholic solution, frequently of intense green color, is treated, according to BECKURTS, with barium hydrate water in excess. This precipitates all chlorophyll, as barium-cyanphyllinate (according to TSCHIRCH barium-phyllocyaninate), which is entirely insoluble in alcohol. Alkaloids are not precipitated at the same time. Filter and remove the excess of baryta by means of carbon di-oxid.

(*e*) In the **method of A. Seyda,** ether, chloroform and amylic alcohol are used.

(*f*) **Method of Dragendorff.** Originally it applied merely to *strychnin* and *brucin*, but gradually DRAGENDORFF and his numerous pupils have extended its applicability to a great number of substances. Proceed as follows: Extract the masses in question by means of *very diluted sulfuric acid at* + 50° *C*, concentrate to syrup consistency, add much *alcohol* and filter after 24 hours. Now all organic substances in question are contained in the solution, from which distil off the alcohol. The clear *acid* solution (eventually filtered again) is successively shaken with *Petroleum-ether*, then twice with *benzene* and finally with *chloroform*. After removal of the chloroform, render the residue *alkaline* by means of ammonia, and again proceed to shake successively with petroleum-ether, benzene and chloroform, and finally, in

addition, with *amylic alcohol* and in a warm condition. Thus we obtain seven or eight portions. Of the numerous substances which these may contain, we merely mention a few for each portion, remarking in advance, that certain substances will have to be mentioned several times, for the reason that they pass into a number of different extracts.

Portion I, obtained by treating the **acid solution with petroleum ether,** may contain : *piperin, picric acid, salicylic acid, benzoic acid, carbolic acid, camphor, cardol,* etc., as far as these substances had not partly been removed with the alcohol distilling off.

Portion II, obtained by treating the **acid solution with benzene,** may contain: *coffein, cantharidin, anemonin, santonin, colocynthin, colocynthein, colchicin, colchicein, digitalin, gratiolin, cascarillin, ericolin, bitter principles* from *Quassia, Artemisia Absinthium, Menyanthes, Cnicus benedictus,* etc.

Portion III, obtained by treating the **acid solution with chloroform,** contains, according to DRAGENDORFF: *papaverin, narcein, digitalin, digitalein, convallamarin, helleborein, saponin, senegin, solanidin, picrotoxin, lycaconitin, myoctonin, colchicin, colocynthin,* etc.

Portion IV, obtained by treating the **ammoniacal solution with petroleum ether,** may contain: *strychnin, brucin, veratrin, gelsemin, aconitin, delphinin, sabadillin, emetin, coniin, methylconiin, conhydrin, lobelin, nicotin, piperidin, spartein, quinin* and many different *ptomaines.* We may advantageously separate volatile alkaloids, such as *nicotin* and *coniin,* without shaking with any solvent, by rendering the extract alkaline with potassium hydrate solution from the start, and subsequeht distillation in a current of hydrogen.

Portion V, obtained by treating the **ammoniacal solution with benzene,** may contain: *strychnin, brucin, emetin, quinin, conquinin, cinchonin, codein, thebain, narcotin, atropin, hyoscyamin, cocain, physostigmin, aconitin, nepalin, lycaconitin, myoctonin, delphinin, delphinoidin, veratrin, sabatrin, sabadillin, pilocarpin.*

Portion VI, obtained by treating the **ammoniacal solution with chloroform,** contains residues of *cinchonin* and *papaverin,* as well as *narcein, cinchonidin, berberin* and small quantities of *morphin* and *muscarin.*

Portion VII, obtained by treating the **ammoniacal solution with amylic alcohol,** contains: *morphin, solanin, salicin,* as well as residues of *convallamarin, saponin, senegin,* and *narcein.*

Portion VIII, viz., the residue which had not been taken up by any of the solvents used, is evaporated to dryness after addition of pulverized glass, then powdered and extracted with warm chloroform, which will dissolve *curarin* and the main quantity of *muscarin.*

3. The Methods of Precipitation differ in that some of them serve the purpose of obtaining in the precipitate, the substances sought for, while others, conversely, exclude these from the ppt. In the latter case the ppt. consists of: coloring matter, tannins, albumins, mucous matter, &c., the main quantity of which is precipitated from a varying mixture of organic and inorganic material. The substances, which are included in this precipitate, do not interest us here, but they would in every case be the source of great disturbance in the matter of chemical isolation and purification of the poison. We mention the following methods as examples:

(*a*) **Precipitation according to Sonnenschein and Palm.** According to SONNENSCHEIN, the mass in question is extracted with *water* which had been *strongly acidulated with HCl*, the extract evaporated at + 30° C. to syrup consistency, and again diluted with water. Filter and add to the filtrate *phospho-molybdic-acid.* The precipitate formed includes all *alkaloids*, but neither all *glycosids* nor *picrotoxin* and *digitoxin.* Wash with water containing some phosphomolybdic acid and nitre, and finally introduce the moist ppt. into a flask. Now add barium hydrate until alcalescency obtains, and distil at the temperature of boiling water. The vapors which pass over contain *ammonia* and all *volatile bases.* Towards the end, when merely water passes over, cool the contents of the flask and precipitate barium carbonate by means of CO_2; do not filter but evaporate to dryness and extract the mass with strong alcohol. All the fixed poisons liberated from their phosphomolybdic-compounds by barium hydrate, will pass into the alcoholic solution, which is evaporated.

This method is suitably modified according to PALM, by using water, slightly acidulated with sulfuric acid, for the extraction of organs or the pulp of food, and precipitating the filtered extracts by means of *sugar of lead* solution. Of the known poisons only *quillaic acid* is thus precipitated. Now filter and precipitate with ammoniacal sugar of lead solution: all *glycosids*, *e. g.*, *sapotoxin*, *digitalein*, *convallamarin*, *helleborein*, *adonidin*, etc., as well as *picrotoxin.* This ppt. after decomposition with hydrogen sulfid is separately analyzed. To the filtrate, which is tolerably clear, and which contains neither sugar nor glue and coloring matter, an excess of diluted sulfuric acid is added, which precipitates all lead in the form of plumbic sulfate. Then the filtrate is treated according to SONNENSCHEIN by precipitating with phosphomolybdic acid, etc.

(*b*) **Precipitation according to Brieger,** invented in 1886, is particularly adapted to the detection of ptomaines in parts of the cadaver. The chopped parts are extracted with *water acidulated by*

hydrochloric acid, and also boiled for a few minutes. Filter, evaporate the filtrate to syrup consistency and add 96 per cent. alcohol in excess. This leaves a great amount undissolved (albumin, pepton, etc.) The alcoholic solution is filtered and, while still warm, *alcoholic sugar of lead solution is added.* The lead precipitate contains no bases. Filter again. Remove lead from the filtrate by means of a current of hydrogen sulfid and precipitate the resulting clear filtrate *with alcoholic corrosive sublimate solution.* This precipitate contains a part of the *ptomaines* and *alkaloids*, while its filtrate contains the remainder, which, after removal of mercury and alcohol, is precipitated by *phospho-molybdic acid.* For the purpose of further characterizing and isolating the ptomaines, precipitations are resorted to, by means of *platinic chlorid*, *auric chlorid*, etc.

4. Method of Detection by the Aid of Reagents. We use two kinds, viz., *reagents for precipitation* and *reagents causing changes of color.* We have mentioned some of the more prominent reagents for precipitation in discussing the methods of SONNENSCHEIN, PALM and BRIEGER. We review these in the table pages 49-51. The color reactions are necessarily performed with the questionable substances in a pure, dry state. The precipitates produced by the pertaining reagents will keep for days, but the color changes are very evanescent, and require for their study incessant observation, beginning from the moment when the reagent is added to the contents of the watchglass or small dish. Failing to do this we will overlook the most important change of shades. Some of these colors will show characteristic absorption bands on *spectroscopic* assay.

A few substances, as *cocain*, *physostigmin* and *atropin*, may be detected by peculiar *odor* reactions. Many alkaloids and glycosids are detectable by the polariscope and spectroscope.

TABLE OF THE MOST IMPORTANT REAGENTS FOR ALKALOIDS AND GLYCOSIDS.

No.	NAME AND COMPONENTS OF THE REAGENTS.	WHAT IT PRODUCES.
	PRECIPITATION REAGENTS.	
1	Ammonical sugar of lead	White precipitation of all glycosids, very voluminous
2	Reagent of de Vry-Sonnenschein: Phosphomolybdic acid	Precipitation of alkaloids and glycosids in acid solution, white

No.	NAME AND COMPONENTS OF THE REAGENTS.	WHAT IT PRODUCES.
3	Reagent of Scheibler: Phospho-tungstic acid	Precipitation of alkaloids and glycosids in hydrochloric acid solution, white
4	Reagent of von Planta, Ferdinand Mayer, Tanret, etc.: Mercuric-potassium-iodid	Precipitation of alkaloids, albumin, albumoses, etc., mostly in acid solution
	Reagent of Dragendorff: Potassium-bismuthic-iodid	Precipitation of alkaloids in acid solution, crimson
6	Reagent of Marmé: Potassium-cadmic-iodid	Precipitation of alkaloids in acid solution
7	Potassium-zinc-iodid, concentrated aqueous solution	Precipitation of alkaloids in acid solution, for narcein and codein, beautifully crystalline
8	Reagent of Bouchardat: Iodin-potassium-iodid	Precipitation of alkaloids, yellowish-brown, later on crystalline
9	Reagent of Dittmar: Solution of chlor-iodin	Precipitation of alkaloids containing the pyridin nucleus, yellowish-brown
10	Brom-potassium-bromid	Precipitation of many different alkaloids
11	Bromin-water, saturated solution	Precipitation of many different alkaloids, brownish
12	Platinic chlorid, concentrated aqueous or alcoholic solution	Precipitation of most alkaloids in crystalline form
13	Auric chlorid, aqueous solution	Easily decomposible, crystalline, precipitation of most alkaloids
14	Mercuric chlorid, concentrated aqueous or alcoholic solution	White precipitation of many alkaloids and ptomaines
15	Potassium di-chromate, concentrated aqueous solution	Crystalline precipitation of many alkaloids, yellow
16	Potassium permanganate, aqueous solution	Characteristic precipitation of many alkaloids
17	Picro-nitric acid, viz., nitric acid + picric acid	Characteristic precipitation of many alkaloids, yellow
18	Reagent of Essbach: Picro-citric acid	Precipitation of alkaloids and of albumin

No.	NAME AND COMPONENTS OF THE REAGENTS.	WHAT IT PRODUCES.
19	Tannic acid, newly prepared, concentrated solution	Precipitation of alkaloids and of many glycosids
20	Ammonium-diamin-chromium-rhodanid	Precipitation of of most alkaloids in form of constant double salts
21	Reagent of Baumann and Udransky: Mixture of sodium hydrate solution and benzoyl chlorid	Precipitation of diamins and polyatomic alcohols
	COLOR REAGENTS.	
22	Concentrated sulfuric acid containing some nitric oxid	Glycosids turn red, many alkaloids assume different colors
23	Erdmann's reagent: Concentrated sulfuric acid + nitre	Characteristic colorations of individual alkaloids
24	Froehde's reagent: Concentrated sulfuric acid + sodium molybdate	Morphin will at first turn violet, then green, bluish-green and yellow. Characteristic colors also with: veratrin, narcotin, codein, papaverin, thebain, narcein, nicotin, brucin, solanin, etc.
25	Buckingham's reagent: concentrated sulfuric acid + ammonium molybdate	
26	C. Brandt's reagent: Concentrated sulfuric acid + selenic- or telluric acid	Similar colorations as with Froehde's reagent
27	Mandelin's reagent: Sulfuric acid mono-hydrate + ammonium meta-vanadate	Very characteristic colorations with many alkaloids and some glycosids
28	Brociner's reagent: Concentrated sulfuric acid + ammonium niobate, or ammonium uranate, or ammonium tellurate	For some alkaloids, characteristic colorations
29	Wenzell's reagent: Concentrated sulfuric acid + potassium permanganate	For individual alkaloids, as, *e.g.*, atropin, characteristic colorations
30	Concentrated sulfuric acid + furfurol	Characteristic coloration with veratrin, quinin, papaverin, morphin, strychnin, sabadillin, digitalin
31	Bloxam's reagent: Hydrochloric acid + potassium chlorate	For certain alkaloids, peculiar coloration
32	Mercurous nitrate	Flesh colored with helleborein, chelidoxanthin, etc.

V.—REMARKS CONCERNING THE DETECTION OF METALLIC POISONS.

Frequently we cannot detect those metals which, as a rule, are easily recognized, even in most diluted solutions, by characteristic reactions. This is the case if the metals are contained in such organic mixtures as blood, macerated food, extract of the liver, etc. It would seem to be a temptation in such cases, to simply desiccate the organ or the pulp, to incinerate it, and to analyze the ash. This, however, would be a grave slip, since some of the metals, *e. g.*, *mercury* and *arsenic*, would volatilize under such circumstances. We are therefore compelled at times to apply more complicated systems of research, of which there exist several. We mention but two, the others being now only of historical value.

1. Method of Fresenius and von Babo. *Chlorin in statu nascendi* is used for the destruction of the organic substances. This energetic agent will act in a threefold manner, viz.: oxidizing, splitting and chlorinating. The process in detail is the following:

The properly cut, rubbed and ground masses are freed from possibly remaining alcohol, and conveyed into a genuine porcelain dish, or still better, into a spacious flask, with doubly perforated stopper (preferably of glass). The flask should not be more than half-filled. One opening incloses a very long condensing tube, one end of which terminates closely underneath the stopper. It serves as an outlet for the developing vapors, and should empty into the hood or chimney. A glass funnel, with glass stop-cock, leads through the second opening. The mash to be acted upon is rubbed well with a mixture of respectively, 2 gr. potassium chlorate and 100 gr. conc. hydrochloric acid, free of *arsenic*, and gently warmed on the water bath. This temperature is frequently insufficient for perfect destruction, and it is commendable, after a while, to heat the flask cautiously on an iron plate, over the gas flame. At the same time a continuous series of drops of a cold-saturated potassium chlorate solution, is received into the frothing mass. This must continue until the contents of the flask, after dispersion of the chlorin vapors, are and remain of bright yellow color. Now, once more, chlorate solution in slightly larger quantity is added, and heating is continued until the odor of chlorin has passed off. Eventually the ultimate quantities of chlorin are driven out by a current of CO_2. It is sometimes necessary, during the process of destruction, to add new quantities of hydrochloric acid, particularly in case the contents of the flask had become too pasty. It is supposed that the reaction between hydrochloric acid and potassium chlorate, in the hot

dish or flask, takes place according to the equation: $6\ HCl + KClO_3 = 3H_2O + 6\ Cl + KCl$. The potassium chlorid (KCl) formed, does not of course act in a destructive sense; if anything, its presence is a disturbing factor.

2. Method of Sonnenschein and Jeserich. For the reason just mentioned, in this method there is used free chloric acid instead of potassium chlorate, which causes a reaction expressed by the equation: $5\ HCl + HClO_3 = 3H_2O + 6Cl$. It is decidedly a rational method, and can be applied every time if arsenic-free chloric acid is at our disposal. Care has to be taken, however, that chloric acid is always present in excess; if this is neglected, *arsenic* may easily be partially volatilized in the form of *arsenic* chlorid. In this modification of the process, the manner of working is preferably conducted in such a way as to add the chloric acid in portions, warming cautiously every time, and only in case the mass becomes spongy, to run the hydrochloric acid in through the funnel, and then increasing the temperature. In both methods, if *antimony* or *tin* are suspected in the mass, the delivery tube, instead of leading into the chimney, must pass into a cooled receptacle in order to catch traces of *antimonic* chlorid or *stannic* chlorid, which might pass over. In both methods great difficulty arises in regard to the total destruction of *fat* and of *cellulose*.

After the best possible destruction of everything organic has been achieved, and the free chlorin has been driven off, the yellowish mixture is filtered while hot. We then retain upon the filter: *plumbic chlorid*, *plumbic sulfate*, *thallic chlorid*, *argentic chlorid*, *mercuric sulfid*, *barium sulfate*, *tungstic acid*, *silicic acid* and *alumina*. If this precipitate should still contain larger quantities of organic material, its destruction may be again attempted. If it only contains fat, it is thoroughly washed with hot water and then the fat is extracted by ether. Then the precipitate is analyzed according to the ordinary rules of inorganic chemistry. It sometimes occurs that the hot filtrate of the insoluble precipitate, on cooling, deposits a precipitate. This may consist of additional quantities of *plumbic chlorid*, *argentic chlorid*, *thallic chlorid*, as well as *antimony*- and *bismuth*- compounds. It must be filtered and separately analyzed. A sample of this second filtrate is treated with sulfuric acid, which precipitates residues of *lead* and of *barium*, as sulfates, and which can be distinguished by the solubility of plumbic sulfate in basic ammonium tartrate. The greater part of the clear filtrate is now diluted with water, the acidity is slightly reduced, and *arsenic*-free hydrogen sulfid, preferably prepared from barium sulfid, is most energetically and persistently passed through the liquid. *Arsenic*, *antimony*, *tin*, *copper*, *lead*, *silver*, *mercury*, &c., will precipitate from the acid solution as sulfids, while *zinc*, *chromium*, *iron* and *barium* remain in solution. The color of the precipitate is yellow, for *arsenic* (*tin* and *cadmium*); orange to brick-red, for *antimony*; black,

for *mercury*, *silver*, *lead* and *copper*. If no precipitate at all forms, viz., after an excess of hydrogen sulfid had been applied and after the liquid thus stood for days, then none of the metals mentioned is present. Now the liquid, or the filtrate as the case may be, is heated with *sodium acetate* so that all of the free hydrochloric acid is transformed into sodium chlorid. Again, hydrogen sulfid is passed through the solution, when *zinc* will precipitate as white zinc sulfid; *nickel* and *cobalt* as black sulfids. The filtrate of these precipitates is treated with ammonium hydrate, and ammonium sulfid, productive of the following precipitates eventually: *iron*, black; *manganese*, flesh-colored; *chromium*, greenish-blue; *aluminium*, if still present, white or almost colorless.

X.—Physiological Detection of Poisons, and Systematic Course of the Action of Pharmacologic Agencies.

For this research the rule is always potent that *the substance must be as pure as possible, before we are justified in using it for physiological experiments. The aqueous solution of such substances should be of as neutral reaction as attainable.*

The course adhered to, begins with the investigation of the influence upon the lowest possible organisms of vegetable and animal nature. It is then extended to higher cold-blooded animals and upon single organs of these, isolated to all possible extent, or upon parts of such organs. Finally, experiments upon warm-blooded animals, in ascending series, are performed. Upon the immediately following pages we review the course of experiments. Later on some important points will be treated more in detail. The great majority of experiments can only be indicated here.

I. REVIEW OF THE COURSE OF EXPERIMENTS.

1. Experiments on enzymes, such as pepsin, trypsin, ptyalin, diastase, emulsin, myrosin. By means of these experiments we have to establish the facts as to whether or not the activity of the mentioned enzymes is simply disturbed by the poison or permanently stopped (protoplasma-poisons), and if the poison is decomposed by the enzymes. Some poisons, such as hydrocyanic acid and cyan-iodid, do not permanently destroy the activity of the enzymes, but as long as these poisons are present, the enzymes are paralyzed. (Enzyme-poisons.)

2. Experiments on micro-organisms, keeping the same two leading points in view as in the foregoing. In case the poison is proved to be detrimental to microbes, we have to ascertain

what degree of dilution is sufficient to stop their multiplication, and what strength is necessary in order to kill them outright (Bacteria-poisons).

3. Experiments on lower plants (especially on algae, characeae and yeast) and on lower animals (infusoria, amoeba, crawfish, worms, insects, etc.), as also on leucocytes and red blood-corpuscles, ciliated epithelium-cells, spermatozoa, cells with protoplasma-movement of higher plants, etc. If the life-activity of all these objects of experimentation is ended by the substance, then we have to do with a *general protoplasma-poison.*

4. Experiments on entire native blood, on defibrinized blood, on serum, on mash of blood-corpuscles, on crystallized haemoglobin, and on stroma. Many blood-poisons are recognized in this manner.

5. Experiments on surviving organs of cold-blooded animals, *e. g.*, of frogs, toads, turtles, etc. The muscles and nerves of these animals can be kept alive for 24 hours, in physiological sodium chlorid solution. The extirpated heart is introduced into an artificial circulation by means of WILLIAMS' apparatus, which maintains its working capacity for from 6 to 12 hours. We need not even use blood as a nutrimental liquid. By such experiments we recognize muscular, neurotic and cardiac-poisons.

6. Experiments on surviving organs of warm-blooded animals, which are flushed by undiluted blood of body-temperature under uniform pressure, in an incubator. The organ most suited for such flushing is the kidney; we can, however, use the spleen, pieces of the intestine, liver, uterus, foot, etc., for this purpose. The vascular system can be longest kept alive; it will respond promptly to excitations by dilatation or contraction, even after 1 to 2 hours. These oscillations of calibre can be easily calculated from the quantity of blood flowing from a principal vein in a unit of time. The experiments of this paragraph serve for the recognition of poisons for *vessels* and *organs.*

7. Experiments on entire, or especially prepared cold-blooded animals. In these, the frog (*Rana temporaria* and *Rana esculenta*) plays the most prominent part. We must take

particular cognizance of general appearances (tetanics and poisons causing paralysis), to appearances of the organs of sense, the digestive tract, the urinary organs, the skin (poisons for the glands of the skin), the reflexes, circulation, heart, muscles and nerves. The most important experimental procedures are: fenestration, ranging out the brain and the spinal marrow, exposure and excitation of the vagus, GOLTZ' percussion-experiment, experiment of hypnosis, TUERCK-SETCHENOW'S experiment, curarization, CLAUDE BERNARD'S experiment. With the aid of these, the results of the preceding experiments are confirmed and supplemented, particularly as regards the differentiation between the poisons of the brain, spinal marrow, peripheral motor-nerves, and peripheral sensory-nerves.

8. Experiments on *embryons* of cold- and warm-blooded animals cannot be dispensed with, regarding tetanic- and cardiac-poisons particularly. The most accessible embryons are larvae of frog and partly-hatched chicken's eggs. Of course the experiments on the latter have to be performed in an incubator.

9. Experiments on entire normal warm-blooded animals, without vivisectory interference, and on pregnant and non-pregnant, as well as on old and young. We have to direct our attention to the points already mentioned (sub. No. 6), and, in addition, to the following: respiration, temperature and metabolism (urine, faeces, perspiration, gases of the breath, weight of body). The experiments involved are those with the respiration-apparatus and with the calorimeter. In this manner we recognize poisons involving: kidney, breath, metabolism, fever, inflammation, vomiting, laxation, saliva, perspiration. Concerning the sensory organs, the pupils in particular are to be scrutinized (Pupillæ-poisons). The administration of the poison is partly performed *per os*, partly hypodermically, partly by dropping into the eye. It is customary to use for these experiments the following animals: cat, dog, rabbit, sheep, cattle, horse, hedgehog, rat, mouse, chicken, pigeon, goose.

10. Experiments on *entire warm-blooded animals, previously either operated upon or prepared in some way, but being restored to health at the time of the experiment.* To this series belong

experiments upon animals in which fever had been artincially incited, that had been infected by bacteria, immunized, chronically poisoned, such as had received in advance an antidote, animals with removed kidney, or liver, or pancreas, or cortex of the cerebrum, others with artificial fistula of the stomach, liver, pancreas, small intestine, etc., and finally upon animals whose spinal marrow had been cut. Thus we obtain in this a material supplement to our knowledge and important data concerning the therapeutic applicability of the agency in question. We recognize in this manner poisons of the stomach, small intestine, large intestine, liver, pancreas, spinal marrow, cerebrum, etc.

11. Experiments on *warm-blooded animals, necessitating binding or restraining, and operative invasions during the time of the experiment.* Under these conditions everything already mentioned (sub. 6-10) will be observed, but, in addition, a number of other phenomena, such as excitability of the cerebrum by electric currents, sensibility of the skin, strength of the muscles, excretion of perspiration (poisons of perspiration), urine, saliva (saliva-poisons), blood-pressure (vasomotoric poisons), frequency of pulse, curve of pulse, volume of the exposed kidney, spleen and tongue, intensity and form of respiration (respiratory-poisons), movements of the stomach, the intestines, and the uterus (uterus-poisons), flow of lymph (lymph-poisons), pressure of the secretion of the kidneys, liver, pancreas, salivary glands, etc.; chemical, physical and microscopical changes of the blood, etc. It is understood that many of these experiments require quite complicated methods of preparation and adjustment. Those most frequently used are: tying the animal upon its back and introducing a manometer into a large artery, and a catheter into a vein, for injection, tracheotomy, preparation of the pneumogastric-, sympathetic-, splanchnic-, ischiatic nerves. In addition, narcotization, curarization, artificial ventilation, opening of the abdominal cavity in the incubator or in the salt-bath, preparation and opening of the salivary ducts, of the thoracic duct, cutting of the marrow of the neck, extirpation of the supra-renal body, application of Eck's fistula (communication of portal-vein with inferior Vena cava), for the purpose of ranging out the liver, etc.

12. Experiments on *man*, viz, in the first place, on the experimenter himself, and then on others of different age and sex, who are in a healthy condition, and, finally, on properly suited sick.

It is quite customary nowadays to hastily experiment with substances on the sick, without taking the trouble to perform the experiments mentioned (sub. 1-10), but this is to be most emphatically condemned. Also, we claim that each painful experiment on an animal, that might have been shortened or replaced by one causing less pain, is positively deserving of punishment.

II.—APPEARANCES OFFERED BY THE NEURO-MUSCULAR SYSTEM.

Concerning nerves and muscles, a poison may cause phenomena of excitation as well as of paralysis, pre-eminently so in frogs, but also in warm-blooded animals.

1. Motoric Paralysis. The animal lies there as if dead. This paralysis may recognize central or peripheral causes.

(a) **Paralysis due to central cause.** With frogs this condition is not caused by paralysis of the brain alone, in contrast to warm-blooded animals, where this is often the case, but by paralysis of the brain and spinal cord, or of the spinal cord exclusively. But the two forms cannot easily be differentiated. This can be done, however, with a dog, whose cerebrum has been extirpated months before. [GOLTZ.]

The lowest and rarest degree of paralysis (*true brain-paralysis*) in frogs, is of such a nature that only the arbitrary motions cease, while the normal reflexes remain, and a Faradic current applied to the skin overlying the spine will yet be very well conducted by the spinal cord. In such a case, if the electrodes are applied to the exposed brain of the frog, it is, as a rule, impossible to produce convulsions of the lower extremities, particularly of the triceps. These can always be produced when acting upon the brain of normal frogs.

A second degree of paralysis (*stopped cross-conduction*) is shown by the absence of movements of repulsion in one leg, if a Faradic current be applied to the central termination of the severed sciatic nerve. The same current, when applied to the spinal cord or to the peripheral end of the aforesaid nerve, will at once produce violent convulsions of the extremity concerned.

The third degree of paralysis (*stopped longitudinal conduction*) consists in this condition: when a current is applied to the skin cover-

ing the spine, or even to the exposed spinal cord, no convulsions are caused, but a current meeting the sciatic nerve will produce them at once.

(b) **Paralysis due to peripheral cause.** This may concern either the nerve or the muscle. Again, the nerve may be affected in its course, or at its peripheral termination. The nerves in their whole course, viz., the primary nerve-trunks, will become incapable of functioning if they are directly bathed by strongly concentrated intense poisons, *e. g.*, by injecting concentrated cocain solution into the primary nerve-trunk.

The motoric nerves will be paralyzed at the peripheral termination, viz., at the end-bulbs, by the substances belonging to the so-called curare-group. Then the muscle will not be convulsed upon electric excitation, but it will be upon direct excitation. As a secondary and controlling experiment, a frog, one of whose lower extremities is deprived of blood-circulation by ligating all pertaining vessels, is to be poisoned. The nerves of this extremity must continue to show a normal electric behavior at a time when the others have become entirely non-excitable. [Cl. BERNARD'S experiment.]

Paralyses of the muscle, or rather of the substance of the muscle, never occur suddenly, and are betrayed in such a manner that upon direct or indirect excitation, the muscle will react more tardily and weakly, and finally will cease being convulsed at all. Naturally, only coarse changes can be observed in this manner. For the detection of more delicate ones, we use the myographion, KRONECKER-TIEGEL'S apparatus and ROSENTHAL'S frog *carousel*. Changes detectable in such a manner are brought about by the substances of the *saponin-* and *digitalin*-groups by the vomitives, the *lead* and others. As soon as we believe it justifiable to state that there exists paralysis of the muscle-substance, the muscle must at once be inspected microscopically, as it will sometimes under these conditions be found to have undergone certain changes, (*e. g.*, *cyclamin*, *sapotoxin*, etc.)

2. **Motoric Excitation** on the part of the neuro-muscular system, expressed by spasms or convulsions. In the first place, we must test to see whether or not they increase in strength upon external excitations, or if they are only eventually caused by such. (Increase of reflectory excitability.) Here we have to differentiate between mechanical, chemical, thermal, or electrical excitants. The means offered by the strychnin-group, for example, excel particularly in producing an enormous increase of reflectory excitability upon mechanical stimulus. The next thing in order is to find out the cause of the spasms.

(a) **Central Excitation.** The cortex and the basal part of the cerebrum, the elongated marrow and the spinal cord of warm-blooded animals, contain nerve-centres, the stimulation of which will produce spasm. In the frog, we do not know with certainty the "spasm-centres" in the brain proper which are affected by poisons, but we do know of such in the medulla oblongata and in the spinal cord. Next we have to differentiate between the two latter. For this purpose the medulla is severed from its connection with the spinal cord, and it is now observed whether or not the spasms stop permanently; better yet, we try to produce them on a so-called "reflex-frog."

(*a*) If they now obtain, they start as a matter of course from the spinal cord; thus it is, *e. g.*, with *strychnin.*

(*b*) If they do not set in, they originate in the spasm-center situated in the medulla oblongata. Here belongs the *picrotoxin.*

Naturally, the experiment must always be tried with greatly varying doses, as very large doses frequently act in a reverse manner to small ones. Excessive doses of *strychnin*, *e. g.*, will produce a simulation of curare-poisoning. In addition, spasms are produced by some poisons at once after injection, by others after hours or days. The latter is particularly the case in the so-called late tetanus of frogs, in poisoning by *morphin* and *atropin.*

(b) **Peripheral Excitations,** as far as the frog is concerned, are easily distinguished from those centrally caused, since after cutting the sciatic nerve, the latter cease at once in the extremity concerned; the former do not. Nevertheless, the peripheral excitation may be of two kinds.

(*a*) It is true that the—generally fibrillar—convulsions persist in case of cutting the sciatic nerve, but they will stop at once if the animal be curarized. We then have to do with excitation of the terminations of the motor-nerves which, *e. g.*, is the rule in *guanidin*-poisoning. The result of the experiment is in such a case to be controlled by a second frog, in which, before poisoning, the vessels of one hind leg have been ligated, as in CLAUDE BERNARD'S experiment. If now *guanidin* be given, the spasms will only occur in the extremity not ligated.

(*b*) The spasms persist in spite of cutting the sciatic nerve and of curarization. This a case of excitation of the muscle-substance, caused, *e. g.*, by most of the salts used in insufficient dilution.

(*c*) In order to render this state of excitation perceptible, it will at times be necessary to mechanically or electrically excitate the muscular system. It will then be seen that though a total contraction takes place promptly, relaxation occurs with exceeding slowness. Autographic tetanus-curves of such a muscle show an extraordinarily long descending side. This obtains, *e. g.*, with *veratrin.*

3. **Sensory Changes** are not at once perceptible, but they may be rendered objective by means of the following experimental arrangements: Take a reflex frog, viz., an animal in which, a few days in advance of the experiment, the cerebrum had been cautiously severed by a cut, from the medulla oblongata. Attach the animal to a stand by means of a ribbon which runs around the body behind the forelegs, so that the hind legs hang down free. Upon dipping one or both of these cautiously into well-water of the temperature of the room, no movements should take place. If, however, we dip into very dilute hydrochloric or sulfuric acid, there ought to follow in each experiment, after about the same length of time, a motion by means of which the moistened part of the extremity is withdrawn from the acid. Generally a wiping-off movement will at once follow. Washing and drying the leg well, permits at intervals of five minutes a repetition of the experiment with the same result. Noting in how many seconds or metronomic beats the withdrawal in both extremities takes place, we may poison the animal. This arrangement is called Tuerck-Setchenow's experiment. It has of late been improved in such a manner as to exclude the circulation. Now the following cases may obtain:

(a) If no variation is observable in the length of time after which the reaction obtains, immaterial whether the poison had been injected into the lymphatic sac of the back, or hypodermically into the lower part of the leg, or brushed over a foot, the poison is without influence upon the reflexes here in question.

(b) In case the time of reaction is considerably shortened, after the lymphatic sac of the back had been injected, the poison has an influence increasing the irritability of the centres of the spinal cord that are instrumental in bringing about the reflex-action.

(c) If the substance injected into the lymphatic sac of the back does entirely away with the withdrawal of the extremities, this may have two causes:

(1) In case the motor-nerves are still electrically excitable, the reflex-centres of the spinal cord have been paralyzed.

(2) If, however, the motor-nerves are no longer electrically excitable, their terminations are paralyzed, as seen in the action of *curare*. In this case the true diagnosis is ascertained by the mentioned experiment of CLAUDE BERNARD.

(d) If injection into the lymphatic sac of the back brings about no distinct action, but if upon hypodermic injection into the lower part of one leg, this will not be withdrawn from the acid, after 30 to 60 minutes, while the other leg still reacts normally, we have to do with the so-called *sapotoxin*-action. This consists in mortification of the nerve-fibres within the territory of the injection—in the first place, of the sensory ones, and then in consecution, also the motors. External application by brushing the poison over a foot will then rather heighten the excitability than lower it.

(e) In case of no distinct action after introduction into the lymphatic sac of the back, but of such decreased excitability of the brushed foot, that the withdrawal from the acid occurs very late or not at all, the so-called *cocain*-action is dominant. This consists in paralysis of the terminations of the sensory nerves within reach of the part brushed. If now the foot is carefully washed with water, the excitability will again become tolerably normal after half an hour, while in case of *sapotoxin*-paralysis, after this length of time it will rather have increased.

(f) If after hypodermic injection into the lower part of one leg the whole foot is restless, and if after brushing one foot, the other one begins at once the motion of wiping and defense, we have to do with a local irritant, one which excites the terminations of the sensory nerves, as, *e. g.*, *ammonia, acids, corrosive salts.* And in case the dip-experiment is really a success, the time consumed by the reflex appears shortened.

III.—APPEARANCES REGARDING THE HEART.

The changes concerning the nerves and muscles have so far been discussed, with the tacit understanding, that the heart remains at the same time entirely normal, or that it still supports circulation tolerably well. But in many instances of intoxication the heart-symptoms stand preeminent. They can be studied to much better advantage by means of WILLIAMS' apparatus and with a frog whose heart is exposed than with a warm-blooded animal.

For the sake of better understanding certain appearances, we will *persist in accepting the existence of heart-ganglions*, although modern science tends towards the belief of their non-existence.

(1) The heart fails to show any systole proper; it beats steadily slower and weaker and finally stands still in diastole (or hemi-diastole), while nerves and muscles of the animal continue to react normally. This is *primary arrest of the heart in diastole.* It may be an arrest by *excitation* or by *paralysis.*

(a) An arrest in diastole by excitation takes place: (*a*) if the restraining impulses traversing the path of the vagus are increased, or: (*b*) if the inhibitory centres within the heart itself are excitated, as in case of *muscarin*. Since the restraining influence of the vagus' fibres affect these heart-centres before making an impression upon the heart, it is evident that paralysis of those centres annuls the action of the vagus upon the heart. If then, in a given case, we want to decide whether we have to do with stoppage due to actual impediment of the heart, or by excitation of the same, we allow a drop of an atropin solution, of not higher than one per cent. strength, to fall upon it, by means of which the inhibitory centres of the heart are almost instantaneously paralyzed. If now a normal, persisting sequence of beats recurs, it proves the case in question to be one of heart-failure due to excitation.

(b) If, reversedly, the heart remains at rest in diastole, or if it merely beats for a few times, the case is one of *arrest by paralysis in diastole*. This may concern: (*a*) the exciting motoric-ganglions, or (*b*) the muscle of the heart. To decide which of these conditions obtain, we must have recourse to electric or mechanic excitation of the muscle of the heart, after it has become perfectly quiet. If upon such excitation the heart responds every time by a few strong, proper contractions, and stands still anew, we have before us a case a primary paralysis of the exciting motoric-ganglions. On the other hand, if the aforesaid excitation produces almost no effect, then either the muscle of the heart alone, or simultaneously its exciting motoric-ganglions, had been paralyzed. As a rule, paralysis of the ganglions quickly follows upon the paralysis of the muscle, and *vice versa*. If only the muscle is paralyzed, we sometimes succeed in reproducing the beats by muscular excitants, as, *e. g.*, by *camphor*.

(2) The heart beats periodically, now quite normal in the matter of frequency, now slower and slower, without, however, stopping altogether. This curious behavior, called the periodic beat, and which particularly results in case of some opium-alkaloids, may continue for hours, and so far remains unexplained.

(3) Immediately after poisoning, the heart beats slower, and may even once stand still in diastole, but suddenly it resumes its beats, returns to normal frequency and continues to beat in a seemingly normal fashion. Here we find, then, no repeated variations from retardation to acceleration, but note only one such deviation. If we look closer into the heart after the beats have resumed their normal sequence, we ascertain as follows: Neither GOLTZ'S percussion-experiment, nor excitation of the vagus, by means of faradic currents, at its points of departure in the brain, nor of its trunk in the course towards the heart, will inhibit the heart's action as they did before the poisoning. The reason for this is that the vagus had been paralyzed at its peripheral termination, and the preceding retardation of pulse corres-

ponded to an excitation of short duration of this nerve, which, however, was soon reversed to paralysis. This paralysis of the terminations of the vagus of the heart, does not extend to the adjoining ganglions of retardation of the heart, as we can show by the fact that they allow of pharmacological as well as electrical excitation. (The latter are produced by adjusting the electrodes to the sinus of the *vena cava.*) To the poisons acting in this manner belong *pilocarpin* and *nicotin.* In order to prove that the original excitation of the vagus was not a central but a peripheral one, the experiment is repeated with a frog, in which the trunk of the vagus had been previously cut through. Then all appearances will obtain just as well as though it were intact.

(4) The heart beats considerably slower after poisoning, but on cutting through the trunk of the vagus, this retardation ceases. This proves a *central retardation* by excitation of the vagus within the brain, or the medulla oblongata respectively. Frequently the poisons which act thus, produce still other effects upon the heart, which makes the interpretation of the results a very difficult matter.

(5) The heart, after poisoning, beats slower and slower, and arrives at a state of *peristalsis*, when the contraction like a slow peristaltic wave runs along the heart to its apex. (*a*) Finally the heart stands still in most exquisite systole, the animal being otherwise perfectly normal, jumping about. Dropping of atropin, changes nothing in this systole. This arrest in systole by excitation is characteristic of all poisons of the *digitalin*-group, but it will exclusively and regularly be brought about with cold-blooded animals. (*b*) Very few poisons allow an arrest in diastole to follow the stage of most typical peristalsis of the heart. This behavior characterizes, *e. g.*, *aconitin.* Naturally, this is a case of arrest in diastole by paralysis, as mentioned above.

We can attain an idea of the quantity of the poison or of its degree of concentration within the blood at which the discussed appearances set in, if we apply the poison gradually to a well-beating frog-heart, attached to WILLIAMS' apparatus and allowed to remain in connection with its *nervis vagis* according to HARNACK-HAFEMANN'S modification. We are granted the additional advantage of washing the heart with normal blood in the very moment when it stands still by paralysis, which then may often be reverted. In case of those substances which decompose the corpuscles of the blood, we take serum instead, which ought to be prepared as freshly as possible by centrifuging, or we take a weak alkaline solution of gum-arabic, impregnated with oxygen. WILLIAMS' apparatus has the particular advantage that by it we can measure the force of the

heart by means of a manometer, as well as the quantity of blood voided in one systole, *i. e.*, the volume of the pulse. It is commendable to repeat the experiment, by means of the same apparatus, with the heart of Emys europaea.

Finally, a solution of the substance is injected into snails (*e. g.*, Helix pomatia) with hearts laid bare. Here the neurotic heart-poisons remain as a rule inert, while the muscular ones act in the same manner as in the frog. Now the experiment is repeated on larvae of Melolontha, in which the heart is visible from outside, and upon which both kinds of poison are, as a rule, inactive, while only the protoplasma-poisons change the action of the heart. Finally, experiments are begun with the heart of warm-blooded animals and with the partly-hatched chicken's egg.

IV.—APPEARANCES IN REFERENCE TO THE PULSE.

The pulse of warm-blooded animals is studied either through the heart or through a larger artery by means of the sphygmograph and other apparatus. Under the influence of poisons it may change in regard to form, velocity of propagation, frequency and intensity. In regard to frequency, three conditions may obtain.

1. The rate is uninfluenced by small doses, and for large ones remains so until at least shortly before death. Then the substance does not belong to any of the following: *muscarin-*, *pilocarpin-*, *atropin-*, *digitalin-group*; in other words, it is no pulse-poison. Experiments on the frog in this case ought to show the same inertness.

2. The frequency of the pulse is retarded, *i. e.*, a toxic bradycardia is produced.

(*a*) If we now proceed to cut through the vagus and then find that the pulse resumes its normal rate, or if a preceding cutting of the vagus prevents the appearance of retardation, excitation of the centre of the vagus within the brain is the cause.

(*b*) In case a medium dose has a retarding effect while a strong one produces a greater retardation of short duration, even a temporary standstill of the heart, and then leads to acceleration of the pulse, we are justified in thinking of a member of the *pilocarpin*-group. If the poison really belongs to this group, the experiment must lead to the

same result if performed after the vagii have been cut through. Acceleration having begun, excitation of the peripheral stumps of the vagii ought not to lead to retardation, which, however, will follow upon injection of *muscarin*. The frog-heart ought to show the identical results. This is a case of passing excitation of the penultimate terminations of the vagus, which reverts spontaneously into paralysis.

(*c*) If the poison applied in a small or large dose has merely a retarding action, immaterially whether the vagii are intact or cut through, atropin is to be injected at the stage of greatest retardation, when the pulse just begins to fail. If subsequently the animal recovers at once, and if now the pulse remains normal for hours (only a little accelerated), we have a case suggestive of *muscarin*, caused by excitation of the true terminations of the vagus, *i. e.*, of the heart-retarding ganglions. However, the existence of these latter is now disputed. The same result must have previously obtained with the frog. From the start, the unpoisoned mammal shows a tolerable excitation of the vagus (normal tone), the frog does not.

(*d*) If the poison simultaneously retards the pulse and weakens its intensity, without atropin changing this state, this is a case of commencing paralysis of the motor-apparatus, *i. e.*, of the excito-mortoric ganglions or of the muscle-substance.

(3) The pulse is increased in frequency, *i. e.*, a toxic tachycardia is produced.

(*a*) If this is removed by cutting through the accelerans nerve on both sides, we have to do with an excitation of the extracardial centres of pulse-acceleration which send their impulses through the accelerans nerve.

(*b*) If electric excitation of the peripheral termination of a severed vagus has a temporarily retarding action, it is a case of excitation of the excito-motoric apparatus within the heart.

(*c*) If it is utterly impossible to diminish the frequency of the pulse by severing or by electric excitation of both vagii of the neck, *i. e.*, their peripheral stumps, but if muscarin still acts, we have to do with paralysis of the penultimate terminations of the heart-vagus, suggestive of *pilocarpin*. In the corresponding experiment on the frog, muscarin as well as excitation of the sinus should act retardingly.

(*d*) If even muscarin does not influence the acceleration, and if electric excitation increases instead of stopping it, we have a case of paralysis of the ultimate terminations of the vagus of the heart suggestive of *atropin*. Excitation of the sinus of the frog ought to give no response or possibly have had an accelerating action.

(*e*) The poison having been devoid of action upon the frog's heart, but it causes in warm-blooded animals fluttering of the pulse, *i.e.*, relatively quick, but very small, irregular pulsations to which corre-

spond fluttering movements of the muscle of the heart, and which in presence of greatly diminished blood-pressure, rapidly lead to death. This is a case of paralysis of the extra-cardial centres of the heart's activity in the central nervous system, which do not exist in the frog.

V.—APPEARANCES REGARDING BLOOD-PRESSURE.

Take the curarized animal, cut through its vagii, and observe for some length of time the blood-pressure with the aid of the kymographion and the manometer. Inject minute, gradually increasing, doses of the poison into a peripheral vein. The following results may occur:

1. **The blood-pressure remains still normal** after a dose known to be fatal to an animal not curarized, and begins to diminish, only ending fatally after a still larger dose had been applied, or after hours of waiting. The substance has then in doses short of fatal ones, no prompt action upon the blood-pressure, but merely changes the same secondarily during agony.

2. **The blood-pressure rises distinctly** after each injection. This may have three causes:

(a) The rise depends upon excitation of the vasomotoric central apparatus, namely:

(α) Upon excitation of the hemispheres of the cerebrum, or oftener of the main vasomotoric centre within the medulla oblongata. If this be the case, the rise should not occur in an animal in whom the cervical portion of the cord had been severed. Thus, *e. g.*, acts *cytisin.*

(β) It depends, in addition to the foregoing, upon excitation of, *e.g.*, the vasomotoric centres of the spinal cord which furnish innervation to the splanchnic nerves. If this should be the case, we find, after cutting through the marrow of the neck, the shock having passed, that the poison still causes increase of blood-pressure. This will stop, however, after the spinal marrow has been drilled out. Thus, *e. g.*, acts *strychnin.* There are no poisons existing which excite only the vasomotoric centres of the spinal cord.

(b) The rise depends upon excitation of the peripheral vasomotoric apparatus, imbedded in the walls of the vessels, *i. e.*, upon a contraction of the vessels, independently of the centre. If this is the case, the rise must obtain after injection of the poison into the blood of an animal, even if the marrow of the neck has been cut through and

whose spinal marrow has been drilled out. But in a much more humane manner we may convince ourselves that a poison, as, *e. g.*, *helleborein*, *digitalin*, etc., contracts the vessels, independently of the centre, by means of flushing through surviving organs, when we shall find that the velocity of the outflow decreases under the action of the poison.

(c) The rise depends not upon excitation of the vasomotoric centres, nor upon excitation of the peripheral vasomotors, but it originates from the heart. This increase of potency on the part of the heart, possibly recognized before by means of WILLIAMS' apparatus, is admitted proven in the warm-blooded animal, if, after paralyzing the vasomotoric central organs and the peripheral vasomotors (by means of amyl nitrite or of chloral hydrate), an increase of the blood-pressure may still be caused by the poison. This is admittedly not final proof, since then the two mentioned substances only bring about a perfect paralysis of the vasomotoric apparatus, if applied in doses which will also injure the heart. Again, *helleborein*, *digitalin*, *digitalein*, etc., belong to this group of poisons which increase the blood pressure, starting from the heart. However, we must emphasize the fact that those substances at the same time act in a contracting manner upon the peripheral vasomotors, or, more exactly, upon the walls of the vessels, thus increasing the blood pressure. We are then only justified in concluding that the increase of blood pressure originated from the heart, if we can prove that the vasomotoric centres had not been influenced, and if the flushing experiment did not evince a contracting of the vessels.

3. **The blood-pressure diminishes distinctly** after each injection, without rendering the animal moribund. This may have four causes:

(a) It is based upon paralysis of the vasomotoric centres within the brain and spinal cord. After switching off their influence as described above, the poison should then no longer produce a lessening of the pressure. Unfortunately this manner of proof is defective in that the destruction alone of the spinal cord as a rule excessively decreases the blood pressure. It is preferable, therefore, to reason by exclusion upon the central action, or to push electrodes into the marrow of the neck, thus exciting it electrically while the nervous system is intact; in case it is not paralyzed, the blood-pressure should be increased; in case it is paralyzed there will be no change during excitation.

(b) It depends upon paralysis of the peripheral terminations of the major and minor splanchnic nerves, quite independently of the centre. These two nerves are the vasomotors of a very extensive system of vessels of the body, *i. e.*, of the organs of the abdomen. Their vasomotoric fibres issue from the spinal cord, from which they depart between

the fifth cervical and the seventh dorsal vertebrae. Severing the splanchnic nerves of a normal animal in their course through the thorax, greatly decreases the blood-pressure, while the vessels of the intestines dilate considerably; if now the peripheral stumps are excitated, the pressure is increased even above the normal. In case of poisons which paralyze the peripheral terminations of those nerves, as *arsenious oxid* and *snake poison*, the excitability of the vasomotoric centres persists, while excitation of the peripheral terminations thereof remains entirely without influence upon the blood-pressure. At the post-mortem the abdominal organs are filled with blood, the intestine in particular, to such a degree that we may consider the condition that of inflammation.

(c) The decrease of blood-pressure depends upon paralysis of the peripheral terminations of all vasomotors, which is quite independent of the centre. We then do not see such a pronounced inflammation of the intestine, although, of course, the splanchnic nerves are at the same time paralyzed. The difference consists is this: that also the other vasomotors are paralyzed, as, *e.g.*, the sympathetic of the neck, innervating the vessels of the ear. Thus, excitation of the mentioned nerves does not render the ear (of the rabbit) pale, and cutting of those nerves is not followed by an hyperaemia of the vessels of the ear. The same experiment may likewise be performed with the vasomotoric fibres of the rear extremities, which run through the trunk of the sciatic. All vasomotoric centres together, with exception of those of the brain, may be (centrally and peripherally) excited by carbon-dioxid intoxication. This is produced by suspension of artificial respiration on the curarized animal. If the vasomotors are still excitable, all vessels will contract, the blood-pressure increasing to, or even above, normal; if they are paralyzed, no effect will be produced.

(d) The reduction of the blood-pressure depends upon a decrease in the force of the heart-beat independent of the centre and of the peripheral vasomotors. This is recognized with the aid of the so-called compression of the abdomen. Under its influence the blood-pressure is considerably increased, in case the vasomotoric centres or the peripheral vasomotors are paralyzed, but, in case of weakness of the heart, this mechanical manner of limiting the course of the current, will not suffice to increase the low blood-pressure. Pinching of the abdominal aorta may be substituted for the compression of the abdomen.

All the mentioned experiments in regard to blood-pressure have been performed upon animals, the cervical portion of whose vagii had been severed. It now remains to repeat those experiments upon animals with uncut vagii. Other results may then obtain, in case the poison respectively paralyzes or excites the vagus.

1. In case of paralysis of the vagus, an increase of pressure, not formerly noticed, is now the result, and a consecutive cutting of the vagus does not change the blood-pressure. In order to recognize whether the paralysis of the vagus is a central or peripheral one, we now excite the peripheral stump by means of DU BOIS REYMOND'S sleigh.

(a) In case of a sudden marked decrease of the blood-pressure, the peripheral termination is excitable; therefore the poison has paralyzed the centre of the vagus.

(b) In case the poison has paralyzed the terminations of the vagus, we shall obtain no result by electrically excitating them.

2. In case the poison excites the vagus, we have to determine whether this is caused by central or by peripheral action.

(a) Supposing the excitation is of central origin, then the decreased pressure due thereto should be eliminated by cutting the vagus.

(b) In case of peripheral excitation, the cutting makes no change in the blood-pressure, but after injection of a few milligrammes of atropin, paralysis of the peripheral terminations of the vagus ensues, and, consequently, an increase in the blood-pressure.

VI.—APPEARANCES REGARDING RESPIRATION.

The following cases may obtain:

1. Respiration remains normal until death; it will even continue for a short time after the heart has ceased beating. We have then not to do with a respiratory poison, but with a heart-poison, which should have been recognized by the experiments of the last series.

2. Respiration becomes stertorous under the influence of the poison, the rhythm remaining tolerably even; very soon froth will appear at the mouth and nose. We have then to do with a poison causing oedema of the lungs and acting peripherally.

3. The character of the respiration is periodically changed. This may obtain in two ways:

(a) Marked dyspnoea occurs without any anatomical changes within the lungs. This obtains, *e. g.*, for the poisons of the *camphor*-group, and it depends upon a periodic excitation by the poison of the main centre of inspiration.

(b) Respiration becomes periodically shorter until it temporarily ceases altogether. This phenomenon is called CHEYNE-STOKES' breathing.

4. Respiration is rhythmically increased:

(a) If the bronchi are found to be contracted, we must ascertain whether or not this contraction was due to reflex-action through the vagus of the lungs.

(α) In case of dilatation of the bronchi, after cutting the vagus on both sides, the poison had caused either excitation of the peripheral terminations of the vagus of the lungs, or of the endocardial membrane, and of the large vessels.

(β) In case the cutting of the vagus on both sides remains without effect, and if injection of atropin into the blood does not bring about any changes, we have before us the condition of a direct excitation of the bronchial muscles by the poison, or of their motor-nerves. Curare will readily remove the excitation of the nerves. We can get rid of the spasms of the very substance of the muscle itself, only inconveniently and slowly, by substances exerting a paralyzing influence upon the muscle, as *apomorphin* or *double-salts of copper*.

(b) In case the bronchi are not contracted, the cause of the increased respiration can only be found in the nervous centre, and it should continue even if the vagii have been cut. Such an action we notice, *e. g.*, in the first stage of poisoning by means of *prussic acid*.

5. Respiration grows continuously weaker, while the heart remains unaffected.

(a) Provided that electrical excitation of the phrenic nerves does not induce contraction of the diaphragm, there is indicated an action upon the nerves of the muscles of respiration suggestive of *curare*. However, this may set in without participation of the nerves of other muscles of the body, as, *e. g.*, after the exhibition of some kinds of *snake-poison*.

(b) In case the phrenic nerves are not paralyzed, excitation of the cerebral inhibitory-centre of respiration may be involved. This condition should accordingly cease after extirpation of the same.

(c) In case the phrenic nerves have not been paralyzed and the inhibitory-centre has not been excited, we may have to do with:

(1) Apnoea, as after small doses of *hydrogen peroxid*. This really being the case, the condition should disappear entirely upon stopping the injections.

(2) We may have to do with paralysis of the centres of respiration, as, *e. g.*, in case of *cytisin*. Some poisons will paralyze those centres only indirectly by causing anaemia; in the case of such, pinching the abdominal aorta, which increases the blood-pressure, should abolish the paralysis.

6. Respiration, though not becoming weaker, grows much slower and correspondingly deeper, just as after cutting the vagus. Here we have a case of paralysis of the pulmonary terminals of the vagus, or obstruction of the respiratory passages. Such paralysis is caused by large doses of *atropin*. But always, in such a case, the heart allowed to observe ere this, paralysis of the ultimate terminations of the vagus.

VII.—APPEARANCES CONCERNING THE PUPILS.

Of all mammals the cat is best suited for experiments regarding the effects of poisons upon the pupil. Birds are known to possess an iris containing transversely striated muscular fibres, rendering it, in their case, to a certain degree, subject to arbitrary change, and consequently exhibiting a peculiar behavior. The pupil of the cat assumes a moderately dilated state upon medium illumination of the room; and from this condition poisons may cause it to widen (mydriasis), as well as to undergo a slit-like narrowing (myosis). The nervous path from the centre of pupil-expansion towards the pupil, is lodged within the sympathetic trunk of the neck.

1. Toxic Myosis. Three forms are to be considered:

(a) The pupils, after hypodermic or internal application of a fatal dose, undergo contraction enduring until death; but this will not take place upon exhibiting it locally, that, is dropping into the sac of the conjunctiva, and it will disappear at once in the extirpated eyeball. This is a case of paralysis of the dilating centre, *e. g.*, of central paralytic myosis. Such an one seems to obtain in man following *morphin*-poisoning, but not so in the cat.

(b) A high degree of myosis persisting in the extirpated eyeball, follows hypodermic injection, as also upon the immediate application into the sac of the conjunctiva, even after medium doses; but in all cases giving place to dilatation after dropping in small doses of *atropin*. This is a case of peripheral excitation of the terminations of the constrictor-nerve, *i. e.*, the oculomotoric nerve which supplies the sphincter iridis. Consequently, we have to do with a spastic peripheral myosis, following, *e.g.*, as a rule, upon *muscarin*-poisoning. With this poison the phenomenon is maintained until death, but in the case of poisons acting similarly to pilocarpin, it may finally give way to marked dilatation.

(c) After hypodermic injection of medium doses, the pupil remains normal, while after fatal doses a high degree of contraction

ensues. Dropping even very small doses into the conjunctival sac will produce a narrowing, which will remain uninfluenced by minimal amounts of atropin, large quantities of which will, however, change this into a condition of widening. In this case, which corresponds to the appearances offered by the action of *physostigmin*, the action, according to HARNACK, is based purely upon excitation of the sphincter iridis muscle, *i. e.*, upon a spastic muscular myosis.

2. Toxic Mydriasis. Under this head the following cases have to be considered:

(a) Fatal doses hypodermically or internally applied, are followed by marked dilatation, which, however, disappears before death, changing to a slight contraction. Dropping into the conjunctival sac is wholly without influence. We have here a case of transient excitation of the centre of dilatation of the pupil, which, later on, changes to a paralysis, *i. e.*, a central spastic mydriasis. This state of affairs is well presented in *aconite*-poisoning. The cat exhibits it even after *morphin* has been given. After cutting the cervical sympathetic nerve it at once disappears.

(b) After hypodermic as well as local application, a dilatation ensues, which shall disappear by drops of *physostigmin* in medium dose. This appearance is characteristic for *B-tetra-hydronaphthglamin*, and it seems that we have here to do with a spastic peripheral mydriasis, viz., with an excitation of that branch of the sympathicus which supplies the dilatator pupillæ.

(c) Hypodermic as well as (mainly) local application of minima doses are followed by a high degree of dilatation, persisting even after death. No effect follows the dropping of muscarin or pilocarpin, but the contrary is true if large doses of physostigmin be administered. Here we have a case of paralysis of the peripheral terminations of the constrictor-nerves, *i. e.*, of that branch of the oculomotorius which supplies the sphincter iridis. We will term this state, which is characteristic of *atropin*, paralytic peripheral mydriasis.

The mentioned mydriatics produce not only pupil changes, but produce at the same time paralysis of the apparatus of accommodation, and the myotics will excite this apparatus; these symptoms, however, are much better studied in man than in the animal. Some substances produce, simultaneously with mydriasis, a protrusion of the eyeballs; so does *cocain*, in particular. However, so far as animals are concerned, this condition of exophthalmos is not a symptom dependent upon any uniform processes, but is generated, *e. g.*, in an entirely different manner in the rabbit or cat. The protrusion of the eyeball may be accompanied by increased separation of the

lids, as, *e.g.*, in the case of the human eye after the exhibition of *cocain*.

VIII.—APPEARANCES CONCERNING THE INTESTINES.

The intestine of an animal, after three to eight days' starvation, is not only quiescent, but the muscle does not even move upon excitation of the vagus. If now the splanchnic-nerves are cut, the intestine reddens, and by peristalsis reacts upon excitation of the vagus. If, instead of cutting the splanchnics, the supra-renal body is extirpated, there will be no increase of hyperæmia, but sensitiveness to excitation of the vagus will exist. But we may consider the supra-renal body as the seat of an inhibitory centre for the peristaltic movements of the intestine. This harmonizes with the fact that, after excitation of the supra-renal body, movement ceases: first, the existing peristalsis, and, second, the peristalsis brought about in satiated rabbits by excitation of the vagus. In addition, we find retarding and motoric centres of peristalsis in the spinal cord and brain, as well as (motors at least) in the walls of the stomach and intestine.

Keeping in mind the above statement, the following short sketch of the action of poisons upon the intestine will be understood. We must not forget, however, that in some respects it remains still quite hypothetical.

1. Tolerable doses will produce symmetrical and regular peristalsis; large doses will act fatally, by central paralysis, after spasms of short duration and strong agitation of the stomach and intestine. In this case, produced, *e. g.*, by *cetrarin*, the cause may be found in an excitation of the locomotor-centres of the gastro-intestinal canal, having their seat in the central-nervous system.

2. Even small doses will produce irregular movements of the intestine, which may be removed by excitation of the supra-renal body, as also by atropin.

(a) The experiment succeeds without further preliminaries only in the satiated animal; we have then a case of excitation of the terminations of the vagus, as, *e. g.*, after *pilocarpin* and *nicotin*. The starved animal admits of a successful experiment only after extirpation of the supra-renal body.

(b) The experiment succeeds without further preparation also upon the starved animal. We then have a case of excitation of the motor-

ganglions situated in the intestinal wall,—the plexuses of AUERBACH and MEISSNER. We cannot always decide whether or not a central excitation takes place. Of this action are: *carbon-dioxid* and *muscarin.*

3. The substance causes spasms of the intestine, but, under certain conditions, not immediately, if applied in not too small a dose to a starved animal. It is then generally a case of excitation of the masculature of the intestine. Here belong, in a certain sense, *lead, veratrin* and *physostigmin.*

4. Doses of the substance, not excessively small, will stop the excitability of the vagus of the intestine, in both the starved and the satiated animal.

(a) The poison itself, under certain conditions, causes movements of the intestines independent of the vagus, and which are produced even on the surviving intestine. This is the action of *atropin.*

(b) The poison in a marked manner renders the intestine quiescent, even the surviving specimen. In case of an injection limited to a single flexure, this one only will be concerned in the first place. We have here exhibited the action of *morphin*, and particularly of *opium.*

5. The substance produces a high degree of inflammation of the intestine:

(a) Because it is locally corrosive. Then, upon introduction *per os*, as a matter of course the stomach had been corroded. Under this heading belong, *e. g.*, strong *acids*, strong *alkalies*, and the *corrosive salts of metals.*

(b) Because, directly or indirectly, by disintegration of the blood-corpuscles, it gives rise to coagulation within the vessels of the intestine. The following exhibit this action: *cyclamin, sapotoxin, ricin, abrin, solvin*, etc.

(c) Because it is diffused through the mucous coat of the intestine, exciting the latter simultaneously. To this place belong the hypodermically injected and not directly cauterizing compounds of, *e. g.*, *mercury* and *antimony*, as well as the *gambogiacic-acid.*

(d) Because it paralyses the splanchnic nerves. This produces, without excitation, causing an inflammation proper, yet such a high degree of dilatation of the capillaries of the mucous lining as to convey the impression of inflammation. In addition, the substance is frequently brought here by osmosis. Such is the action of, *e. g.*, *arsenic, snake-poison*, and *urechitoxin.*

Concerning the appearances of other organs, we refer to KOBERT'S "Lehrbuch der Intoxikationen." For a compendium, we consider the above data as sufficient.

SPECIAL TOXICOLOGY.

We may arrange all poisons into three groups, according to the disturbances which they create.

The first group (A) embraces those poisons that cause, upon man, macroscopical anatomical changes at the place of application or of secretion, or in any organ. Consequently, their diagnosis will be an easy matter, even for the physician of little experience.

The second group (B) comprises those poisons, that in the first place alter the blood. They need not necessarily produce coarse and readily visible anatomical changes at the place of application. In consequence of such alteration of the blood, and should the patients not expire soon, we find present secondary changes, mainly affecting the kidneys, the intestines, and the vessels. But these latter effects cannot be attributed to the poison directly; they are caused by the products of change and decomposition of the blood.

The third group (C) includes such toxic substances as may act fatally upon man, without having caused any striking pathological changes in the organs or the blood. Their primary action is exerted through the central-nervous system (centrally or peripherically), or upon the heart. It is to be supposed, of course, that these substances produce some kind of molecular disturbance of an anatomical nature; our present *technique*, however, has not developed far enough to enable us to every time prove pathologically the existence of these disturbances.

A.—Substances that Cause Grave Anatomical Changes of the Organs.

The group that here interests us is perhaps properly classed under two sub-divisions, viz., substances which irritate mainly the site of application, and those which produce changes in remote parts of the body, yet more strikingly; and which may, in case of dexterous execution of the experiment, even leave entirely intact the place of application. But since there exist substances having an intermediate action between these two groups, we shall refrain from this classification, but yet, bearing in mind that by far the majority of the substances to be enumerated are local irritants. For the sake of easy survey, we divide all pertaining poisons into six divisions, viz.: 1, *corrosive acids;* 2, *corrosive haloids;* 3, *corrosive alkalies;* 4, *corrosive salts of the metalloids, and of the metals;* 5, *inorganic substances*, whose corrosive action is secondary to other anatomical changes caused by them; and 6, *organic substances*, that, at some point or other, bring about marked anatomical changes. Many substances in this group possess in common the feature of producing upon higher organisms, after a certain length of time, suppuration at the seat of application. This suppuration may run its course without complications due to bacterial influence. It is, as a rule, to be considered as the result of a limited inflammation around a necrotic center, primarily caused by the chemical agent.

I.—CORROSIVE ACIDS.

We know that the living human body is of alkaline reaction, with the exception of the stomach and the urinary passages. This is a fact, in spite of the continuous metabolistic formation of *phosphoric-* and *sulfuric-acid.* We have seen (on page 32) that this alkalescency is very rapidly changed even in a normal corpse. But the change takes place to a much higher degree in case of diminution of the alkalescence of the blood. Thus we find, in man and animal, that very soon after death by poisoning with *sodium salts of oxalic-*, *arsenious-* and *hydrocyanic-acid*, as well as by means of *ether* or *chloroform*, the blood is noticeably acidulated. We are consequently not justified in concluding, from the acid reaction of the organs, a

poisoning by means of free acid; nor even that the case is one of poisoning at all. The normal acids of the cadaver are volatile fatty acids and lactic acid; the corpse of a diabetic very likely contains *oxybutyric acid.* The acidulation of the corpse will become more intensified in case of the presence of carbohydrates in greater abundance in the tissues of the dying person. As a rule, we find it most strongly marked in the liver. Later on lactic acid disappears, and instead *succinic acid* will be found. Only then does the alkaline putrefaction set in.

We notice a remarkable difference between herbivorae and carnivorae, if we poison them by slow injection of very diluted acids into the stomach: the rabbit will very soon be paralyzed, lying there as if dead; but the dog will not be so affected. This is explained by the fact that the dog disposes of ammonia ready to neutralize the acid which entered the blood, while in the rabbit this does not obtain. In this regard, man behaves similar to the dog, being able to neutralize gradually immense quantities of, *e.g.*, *oxybutyric acid*, binding them to ammonia and secreting the product with the urine.

On the other hand, all concentrated acids cause at the seat of application—as a rule, the mouth, the oesophagus, and the stomach—an almost instantaneous irritating effect, which we call corrosion or cauterization, if the tissues are thereby chemically changed. But a pure corrosion is only seen by the experimenter upon animals. The coroner almost invariably witnesses the result of a combination of three actions, viz., corrosion of the living tissue, corrosion by reactive inflammation, and the post-mortem changes brought about by the substance. The reactive inflammation does not exist if the corrosive had been introduced after death. As a rule, the living person is found to be most severely affected in the mouth and stomach, while in most cases the oesophagus remains at least partially untouched. Within the stomach, provided they were not taken in excess, the corrosives pass along the lesser curvature towards the pylorus, moistening its entire surface. Thus is explained the astonishing fact that, in case of poisoning by corrosives, we most frequently observe ulceration and stenosis of the pylorus. Secondarily, this may

cause a considerable dilatation of the stomach. The blood in the vessels adjoining the corroded parts is at once coagulated, its coloring matter changing into met-hæmoglobin and hæmatin. The gastric juice begins to act in a digestive manner upon the corroded parts, in which, the normal circulation of the blood has ceased, the alkalescence is impaired, and thus the total effect of poisoning is rendered one of highly complicated appearance. The acid taken up by the blood is partly voided unchanged with the urine, producing severe lesions of the kidney. The mucous coat of the stomach diffuses, besides the normal *hydrochloric acid, hydrobromic-, hydriodic-*, and *hydrofluoric-acid,* and, conditionally, *lactic acid.* The acids, circulating with the blood, reduce its alkalescence, and thus cause degeneration of the heart, liver, kidneys, etc. After internal application, as a rule right after the first gulp, the symptoms of poisoning begiu quite acutely. They consist of: burning taste and violent pain in the mouth and stomach. Regarding the oesophagus, rulably only the entrance, the cardiac opening, and the part which crosses the left bronchus are corroded, causing relatively little pain in this organ. Very soon great thirst sets in, then follows vomiting of brown masses, dysphagia, terrible griping, colic, diarrhœa, and inflation of the abdomen, decrease of temperature of the body, and general collapse. The urine is remarkably acid from the start; it is voided with burning pains, and contains albumin or even haematin. If death does not directly follow, it will be frequently brought about by consequent complications. The introduction of the stomach-tube must be accomplished with the utmost caution, as it will easily perforate the weakened tissues. With most of the acids, only the gastro-intestinal canal need be considered as a seat of application; the epidermis will be merely injured through spilling or by overflow from the mouth. The scars resulting from these injuries by the mineral acids, are subject to strong and painful contraction. In the case of *carbolic acid,* we frequently find external injuries, if the concentrated acid had been used for washing the hands or in surgical dressing. Chemically considered, this substance is no acid at all, and is not called an acid, but *phenol.* Hypodermic acid-injections, for medicinal

purposes, are about limited to the use of *osmic acid*, and, spontaneously, to *formic acid*, by the sting of nettles, etc. *Osmic acid* and its salts will cause degeneration of the nerve-fibres involved, while the skin will be turned black in consequence of reduction of the acid. In case of nettle-hair, we have the action of the acid, and of an unknown enzyme, as well as that of the mechanical irritation. The air-passages are attacked after inspiration of, *e.g.*, *nitric-*, *hydrochloric-*, *sulfurous-acid*. It is understood that even greatly diluted vapors of these are of toxic action, provided the inhalation continues for a long-enough period. They will then cause an inflammatory broncho-pneumonia. If inhaled in the concentrated form, they produce spasms of the glottis, and if the glottis is unable to prevent the further passage of the vapors, œdema of the lungs. The froth expectorated in case of such œdema is not of pure white color, but reddish-brown.

Abbreviations used in the following tables are:

Source.—Preparations or substitutes causing the intoxication.
Stat.—Statistics.
Ætio.—Ætiology.
Dos. let.—Fatal dose.
Act.—Action.
Sympt.—Symptoms.
Dvpt.—Development.
Exit.—Departure.
Diagn.—Diagnosis.
Ther.—Therapy.
P.-Mtm.—Autopsy.
Detect.—Detection by chemical and physiological tests.

Emptying the stomach being an obvious measure, it is not mentioned in any one of the tables.

	SULFURIC ACID.	HYDROCHLORIC ACID.	NITRIC ACID.
SOURCE.	Mostly crude sulfuric acid, or oil of vitriol (30-40 p.c.)	Crude hydrochloric acid, 30 to 40 p.c., generally containing arsenic. Aqua Regia.	Fuming nitric acid, aqua fortis (40 to 50 p.c.); aqua regia, a mixture of nitric- and hydrochloric - acids containing 75 p.c. HCl.
STAT.	In Casper's time, 90 p.c. of all cases of poisoning; now rarer; 75 p.c. mortality.	Only 30 cases so far, in spite of the manifold uses. Mortality 73 p.c.	100 cases recorded thus far; now rarer than formerly; mortality 50 p.c.
AETIO.	Suicide, mistake, very rarely murder.	Accidents, mainly in soda factories; mistake.	Accidents in consequence of wide technical application.
DOS.LET.	4.0 to 5.0 gms. of the conc. acid if stomach is empty; otherwise more, about 5.0 to 10.0 gms. of the 40 p.c. acid.	10.0 gms. of the crude commercial acid.	8.0 gms. of the conc. acid; in full stomach more is required.
ACT.	Rise of temperature, dehydration, transformation of albumin, mortification, coagulation of blood.	Formation of syntonin (an acid albumin formed in the stomach during digestion); mortification; extended brown coloration, and coagulation of blood. Softening of tissues.	Oxidation and nitrification: viz., formation of xanthoprotein, if the acid is at least 33 p.c. strong.
SYMPT.	Cauterization, scab formation, severing of shreds of mucous membrane from the mouth, coffee-brown vomit, terrible pains, possibly spasms.	No scarification of epidermis; pseudo-diphtheritic changes in throat; brownish-green vomit; exhalations of white fumes from the mouth.	Color of mouth and vomit yellowish; otherwise symptoms as with sulfuric acid; retention of urine and faeces; if inhaled, oedema of glottis and of lungs.
DVPT.	Persistent, uncontrollable vomiting; salivation, swelling of mouth, cold perspiration, perforation of stomach, inflation of abdomen, peritonitis.	Similar to sulfuric acid; nephritis, hæmaturia, dysuria, pneumonia, bronchitis, painful diarrhœa.	Similar to sulfuric acid. Pulse small and quick; skin and extremities cold; after short inhalations of the acid, disturbances of the respiration only; pneumonia.
EXIT.	Death from collapse; or stenosis of mouth, stomach, oesophagus; fixed chin; chronic catarrh of stomach; shrivelled kidneys; marasmus.	Death from collapse within 24 hours; mediastinitis with ulceration and pleuritis; perforation of stomach; stenosis of front passages.	Death with stupor after 15 to 24 hours; in case of survival, strictures similar to those of sulfuric acid; parenchymatous nephritis; disturbances of digestion; marasmus.
DIAGN.	Vomiting of acid, non-odorous, brown masses. Urine very acid, brown, containing albumin.	Specific odor, fumes of vomit; urine contains free acid.	Specific odor, yellowish discoloration, nitric acid in urine.
THER.	Water, soap-water, sodium carbonate, magnesia usta, chalk, extract of wood-ashes, albumin, mucilaginous drinks, sweet milk.	Quite the same as with previous acids. In the use of stomach-pump great caution is indicated.	Mild alkalies and alkaline earths, as in the case of sulfuric acid; albumin, gelatinous drinks, sweet milk.
P.-MTM.	Externally, brown scabs; mouth and oesophagus white or grayish-white, with shreds of mucous membrane; stomach brownish-black; coagulation of vessels and acid reaction of neighboring tissue; perforation of stomach; peritonitis; mucous coat of intestines whitish-gray; degeneration of liver; necrosis of kidneys.	Stomach generally not as dark as in the case of sulfuric acid, but of slate-grey color; cauterized, whitish color of mouth, throat, and oesophagus; fragile consistency of corroded parts; mediastinitis; pleuritis; perforation of stomach; fatty degeneration of liver, kidneys and spleen also degenerated.	If the acid was more diluted than that of 20 p.c. strength, no constant characterization; if stronger than 30 p.c., then we find yellow cauterization of lips, mouth and throat. Stomach similar to sulfuric acid; perforation rarer; mucous coat of intestines grayish-white to lilac; kidneys inflamed and swollen; degeneration of liver; pneumonia.
DETECT.	In the contents of stomach and intestines and in the urine, a free, non-volatile, strong acid, forming barium sulfate with barium chlorid; and with lead salts plumbic sulfate; both precipitates are heavy, white, insoluble powders.	Distillate contains an acid which causes a copious white, curdy precipitate, in a solution of silver nitrate, soluble in ammonia. Barium carbonate and calcium carbonate are dissolved by the acid with effervescence of carbon-dioxid. Normally, the stomach contains only a 0.2 p.c. solution of hydrogen chlorid.	Extract with alcohol; determine strength of acid; neutralize with potassium hydrate and evaporate; on addition of pieces of copper and sulfuric acid, red vapors are seen; when mixed with sulfuric acid and ferrous sulfate solution (in the cold), a dark-brown ring is formed; on addition of cinchonamin hydrochlorid, crystals of cinchonamin nitrate are formed.

FORMIC ACID.	ACETIC ACID.	OXALIC ACID.	CARBOLIC ACID.
Hair of certain plants and animals, and stings of certain animals.	Vinegar, aromatic vinegar, liquor Villati.	Crude oxalic acid and acid potassium oxalate (C_2HKO_4)	Liquified, crystallized carbolic acid, generally containing 90 p.c. pure acid, also 3 p.c. aqueous solution.
No case of death solely by the acid.	Seven cases, two of which ocurred by injections into wounds.	Nearly 100 cases, mainly servant girls. In former times only in England.	More than 200 cases of poisoning are known; mortality high.
Stings of bees, ants, hornets, etc.	Mistake; treatment of wounds; suicide.	Suicide: mistake mainly of the salt for Epsom salts.	Mistake; improper use, or in too concentrated form, in surgery (dressing).
Smaller than with acetic acid.	20.0 to 30.0 grms. of aromatic vinegar internally.	5.0 grms. However, 45 grms. have eventually been overcome.	Ten grams. are surely fatal; into a wound-cavity, much smaller quantity.
Similar to acetic acid; in addition, the power of chemical reduction.	Similar to the mineral acids, only milder. Coagulation of blood in the vessels of the stomach.	Locally cauterizing, as the foregoing acids. Then, even in the form of its neutral salt, poisonous for nervous system, kidneys, heart and blood.	Locally, coagulates albumin; after resorption, brain and spinal cord are excited; degenerations.
Externally, itching, burning, inflammation of skin. Internally, similar to acetic acid, even if only 7 p.c.	Vomiting, gripes, spasms, staggering, paralysis of lower extremities, urine brownish-red.	Mouth, throat and oesophagus similar to dil. sulfuric acid; brown vomit; abdominal pains; spasms; unconsciousness, cyanosis; cold perspiration.	Burning and necrosis, when applied to the skin; on mucous membranes, scabs; internally, vomiting, dizziness, fainting, profuse perspiration, and spasms.
Same as with acetic acid; high degree of changes of the blood.	Diminution of the alkalescency of the blood, and in consequence, weakness of the heart and dizziness.	Spasms and paralysis, hæmatic diarrhœa, anuria, formication, feeblenss, pressure around loins.	Weakness of heart sets in rapidly, with suspended respiration; urine becomes dark when exposed to air, and finally blackish-green; pneumonia.
After external application, generally recovery; internally, when tried on animals, death with paralysis and spasms.	Death with oedema of the lungs, unconsciousness, paralytic phenomena, and lowering of temperature.	Death in deepest coma on first day. In case of recovery, persistent anuria, voiding of urine rich in crystals, and of reducing action; gastritis and feebleness maintains for a long time.	Death with unconsciousness, and almost simultaneous cessation of the heart's action and of breaing; spasms rare with man, but present with animals, as a rule.
Externally, the specific action is seen. The odor of the acid is perceptible.	Specific odor of breath and of vomit.	Urine contains sugar; octahedral crystals of potassium oxalate in the urine and faeces.	Specific odor; rapid collapse; urine dark and free from sulfates, becoming still darker on contact with the air.
For external injury, cooling compresses and ice; internally, as with acetic acid.	Same as with mineral acids: chalk, soda, magnesia.	Calcium saccharate, chalk, lime-water, magnesia, albumin, mucilaginous substances, sweet milk.	Washing of stomach with calcium saccharate; internally and hypodermically sodium sulfate; analeptics.
After internal action the finding is similar to that by acetic acid; blood is said to be of a remarkably bright color; urine contains formic acid; all organs have characteristic odor.	Violet post-mortem spots; oesophagus, stomach, and contents of stomach brownish-black, often also the duodenum. Acid, grayish-brown masses in small intestines; peritoneum grayish-violet.	Oesophagus and duodenum frequently whitish and cauterized. Sometimes the intestinal tract almost normal. In the stomach, frequently lentil-shaped haemorrhages; crystals upon mucous coat of intestines, as also in kidneys and urine; small quantities of oxalates in urine are physiological.	Mouth, throat and œsophagus may show inflammation; stomach, relatively little inflammation; broncho-pneumonia, nephritis, degeneration of the liver; in urine, phenol, partly as such, partly coupled; albumin, and blood; also the liver contains free and coupled phenol.
The distillate contains the acid, noticeable by its odor. Ammoniacal-argentic nitrate solution is reduced to silver even in the cold. Fehling's solution is reduced on warming. In cases of fever and of liver troubles, formic acid (CH_2O_2) is formed spontaneously.	The distillate smells of vinegar and is colored a deep wine-red by ferric chlorid. Sodium acetate heated with alcohol and conc. sulfuric acid yields odor of acetic ester; when heated with arsenic, odor of kakodyl.	Boiling alcohol, acidulated with hydrogen chlorid, extracts the acid, which is obtained from residue of evaporation by extraction with water. This yields charactsristic ppts. with lime-water and with plumbic acetate solution. The crystals of calcium oxalate have the appearance and shape of an envelope.	The acid distillate contains free phenol, which is turned lilac with ferric chlorid; with bleaching-powder and ammonia, blue; with mercurous nitrate (containing nitrous acid), rose-red; with bromin-water, crystalline tri-brom-phenol-bromid ppts. The coupled phenol of the urine and the organs must be liberated by boiling with diluted sulfuric acid.

The salts of most of the acids, if of neutral reaction and if not applied in a too concentrated condition, are non-poisonous. Exceptions are *salts of oxalic acid* and of *chromic acid*, the salts, of which latter, and of the mentioned *osmic acid*, remain toxic to a high degree. The nitrites are also poisonous, but they will be treated of with the blood-poisons. As an appendix to the acids or their respective neutral derivatives, we must mention *salol*, which in itself behaves indifferently, but which decomposes into *phenol* and *salicylic acid* in contact with wounds, or under the pancreatic influence. *Salicylic acid* acts similar to *phenol*. The principal symptoms which it ordinarily produces are: violent perspiration, disturbances of memory, buzzing in ears, hæmmorrhages into the retina and into the labyrinth. Much less poisonous than *phenol*, though chemically and physiologically approaching it, are *cresol*, *quinol*, *catechol*, *resorcinol* and *guaiacol*.

II. — CORROSIVE HALOIDS.

The action of *chlorin* is closely allied to that of *hydrochloric acid*, since in presence of aqueous vapor it is transformed into HCl. All of the recorded cases of poisoning refer to inhalations, chlorin being a gas. Concerning *bromin*, which is a liquid, we have the record of inhalations as well as of internal and external applications. *Iodin* is a solid, and the cases of inhalations are greatly in the minority. Generally we find cases of injecting or of taking *per os* solutions of free iodin (in alcohol or in potassium iodid-solution), But we meet cases, where also preparations such as the *alkaline salts of iodin* had been applied, from which iodin may be liberated within the organism, be it by *nitrous acid* of the nasal mucus, or in particular, by yet unknown enzymes of fungii of suppuration. We find here a key to understanding how it is that a certain person may tolerate 30 grammes of *potassium iodid*, while another one will be severely poisoned by 1 gramme. Free *fluorin* is a substance of such rare occurrence that it hardly needs to be mentioned. On inhaling it, it is at once transformed into *hydrofluoric acid*, ozone being produced at the same time. Consequently it is strongly corrosive. Its action resembles that of *hydrochloric acid*. *Sodium fluorid*, internally or hypodermically applied gives rise to the formation of *hydrofluoric acid* within the stomach, which may cause nausea, salivation and gastritis. A large

amount of the sodium fluorid taken, will be deposited in the bones in the form of *calcium fluorid* or *fluorspar*. The following tables (pages 86-87) embody the salient points in regard to Cl, Br and I. Concerning the quantitative relations of absorption of the inhaled poisonous gases, we have the rule, which by no means holds only for chlorin and bromin, that the absorption of the gases, readily soluble in water, occurs to a remarkable degree, and even takes place to the greatest extent within the nose itself.

III.—CORROSIVE COMPOUNDS OF THE ALKALIES AND ALKALINE EARTHS.

The intoxications which pertain here, are similar to those brought about by the acids, by the fact that immediately after introduction, there begin violent pains, based upon cauterization in the mouth, throat, œsophagus, stomach and abdomen. As with the acids, we may also here find that peritonitis follows, that a part of the blood is changed to hæmatin (alkali-hæmatin), and that, in case of survival, strictures are formed mainly of the œsophagus. We cannot state the exact size of the fatal dose for alkalies any more than for acids.

A cardinal difference between intoxication by means of lyes, as contrasted to that by acids, consists in this: the corroded parts do not turn dry and brittle, as in the case of acids, but become soft and pulpy. This happens in consequence of a colloidal swelling of the formed alkali-albuminates, which in presence of much water, will even be partially disolved. In medical jurisprudence this process is called colliquation. Cartilage and horny tissue will also swell and eventually dissolve in caustic alkalies, thus, *e. g.*, hair and skin. The destructive action of the caustic alkalies always furrows deeply, and extends widely into the tissue around the cauterized spot. It is quite common for the action of caustic alkalies, as with acids, to leave contracted scars upon the skin. *Alkali-sulfids* and *calcium sulfid* act in two ways, viz: corrosively, and, in addition, in a specific manner similar to *hydrogen sulfid*. They will be mentioned again with the latter. The action of *caesium* and *rubidium* is similar to that of potassium; that of *lithium* holds a place midway between rubidium and sodium. Strontium acts similar to barium, but it is less poisonous than barium. All other particulars in regard to alkalies and alkaline earths are shown in the table (pages 88-89).

TABLE OF INTOXICATIONS CAUSED BY THE MOST IMPORTANT HALOIDS.

	CHLORIN.	BROMIN.	IODIN.
SOURCE.	Chlorin vapors in bleacheries; in chlorid of lime factories; manufacture of disinfectants, chemical laboratories, in eau de Javelle (Labarraque's solution).	Liquid bromin, bromin vapor and bromin-water are to be considered, the bromin sticks contain free bromin and Kieselguhr (infusorial earth) and are of secondary importance.	Solid iodin; iodin vapors; tincture of iodin; iodin-paraffin; iodin-glycerin; Lugol's solution; iodin-alkalies; and iodoform.
STAT.	Light intoxications frequent; only 15 cases of a more serious nature.	Two cases of internal fatal poisoning; sporadic cases of external corrosion and of inhalation.	Light intoxications occur often; fatal ones only rarely heard of.
ÆTIO.	Unavoidable inhalations of the vapors while working in the presence thereof; hypochlorites are even decomposed by the CO_2 of the air.	Unavoidable inhalation of vapors in laboratories and in photographic workshops.	Inhalations of the vapors in technical pursuits; injections into cavities of the body of solutions containing iodin; decomposition of potassium iodid with liberation of iodin by pus-bacteria or by their products of metabolism.
DOS. LET.	Internally, large doses are withstood; when inhaled, often .06 grams per mlle. fatal.	Internally the large amount of 30.0 grams; when inhaled .06 grams per mlle. have proved fatal.	It is said that internally 3 grams are fatal. With animals intra-venous injection of 4 mgr. per kilo-gram. of body-weight are fatal.
ACT.	Destruction of all substances containing albumin and all epithelia, with the formation of chlorinated products of decomposition. Upon the moist mucous membranes, the chlorin is changed partly into hydrochloric acid and then acts as does this (see page 82). Both chlorin and hydrochloric acid irritate and cause, *e. g.*, broncho-pneumonia, cauterization of the eyes, nose, etc., and decomposition of the blood.	Formation of bromin substitution products of the albuminous substances and epithelia. Upon moist mucous linings H Br is formed of action analagous to H Cl. Bromin, as well as H Br, irritate all the respiratory mucous linings and cause broncho-pneumonia, etc. If the stomach is filled with albuminous food, the bromin is less dangerous. Centrally, we find narcosis and stupor.	Formation of iodin-albuminates which rapidly further decompose. Iodin being less volatile than the foregoing two halogens, acute poisoning by inhalation rarely occurs, if at all. In contact with dissolved iodin, all mucous membranes are severely inflamed, also the epidermis. Iodin causes albuminuria, hæmoglobinuria, bronchitis, dyspnœa, asthma, a peculiar nasal catarrh, somnolency.

SYMPT.	Dyspnœa, bronchitis, nasal catarrh, conjunctivitis, keratitis (in case of internal application, catarrh of mouth and stomach). Acceleration of the pulse, diarrhœa, pneumonia, icterus. In large doses, spasms of the glottis.	Yellowish coloration of the skin and formation of blisters; dyspnœa; bronchitis; nasal catarrh; conjunctivitis; keratitis; spasms of glottis; dizziness; headache; numbness. Taken internally, swelling of the mouth, vomiting of yellowish-brown masses, and sopor. Bromin-acne, in consequence of being secreted through the skin.	Burning and inflammation of parts first attacked; a characteristic nasal catarrh; dyspnœa; asthma; headache; dizziness; œdema of glottis; albuminuria; hæmoglobinuria; urticaria; acne; pemphigus; brown vomit; diarrhœa. Iodoform causes psychosis with depression in man, and in animals attacks of rage and convulsions.
DIAGN.	Characteristic odor of chlorin readily noticed in air of room, and on dissection of the body.	Unmistakable characteristic odor of bromin and yellow colorations. Even in case of dissection the odor may be noticed.	Odor and color of iodin characteristic, also the iodin nasal catarrh. Iodin may be easily detected in the urine by acidulating gently and adding starch paste, which will be blued.
THER.	Prophylactic: Pitzner's protective mask. Inhalations of aqueous vapor; narcotics; internally, mild alkalies, sodium sulfite or sodium hypo-sulfite.	Inhalation of aqueous vapor; internally mild alkalies (soda), albumin, and mild diuretics.	Washing of the stomach with sodium hypo-sulfite; internally sodium carbonate, sulf-anilic acid, sodium sulfanilate, ice, opiates; tracheotomy.
P.-MTM.	In case of inhalations of the vapors, froth in the air passages, the lungs show ecchymotic and pneumonic conditions, brown coloration, ecchymoses also of mucous lining of stomach, catarrh of conjunctiva and nasal mucous lining.	After inhalations, as with chlorin. After application of large doses, mucous lining of stomach brown and solid like tan; erosions; gangrene; stomach perforated. Extended coagulation of blood. Externally, tan-like skin, and partly necrosed.	Stomatitis; swelling of mucous membranes of stomach and duodenum, as well as yellowish-brown coloration; hæmorrhages; pseudo-membranes in pharynx and larynx. Fatty degeneration of heart, liver, kidneys and muscles of skeleton; glomerulitis. In chronic cases, ulceration of skin and intestinal tract.
DETECT.	Free chlorin acts upon potassium-iodid-starch-solution; color changes first to blue, then it is bleached. Litmus and indigo-blue paper decolorized. Metallic silver changed into silver chlorid, which turns black when exposed to the light. The best test for free chlorin is a sound nose.	Distil off the free bromin. Chemically combined bromin is liberated by potassium di-chromate and dilute sulfuric acid, but before this is done, the organic material should be evaporated with potassium hydrate and well charred. The bromin which distils over is known by the color, odor, and by a crystalline ppt. formed with phenol: tetra-brom-phenol-bromid, which also has a strong peculiar odor.	Distil off free iodin; carefully dry and ash the organs with addition of KOH and sodium nitrate; extract the ash with alcohol; evaporate on water bath; dissolve in water; add potassium acid chromate and sulfuric acid; distil again; the distillate contains iodin and turns blue with starch paste.

TABLE OF INTOXICATIONS CAUSED BY CORROSIVE ALKALIES AND ALKALINE EARTHS.

	POTASSIUM- AND SODIUM-HYDRATE (LYES.)	AMMONIA.	POTASSIUM CARBONATE.	CALCIUM OXID (QUICK-LIME).	BARIUM OXID (BARYTA).
SOURCE.	The caustic potassium-hydrate and sodium-hydrate solutions of commerce. Very rarely the fused caustic solids.	Caustic ammonia solutions, strong and weak, varying from 10 to 32 p.c. ammonia.	Potash or pearlash of trade; aqueous extracts of wood-ashes.	Milk of lime, mortar and quick-lime are under consideration.	Caustic baryta; barium sulfid; barium carbonate; barium chlorid; barium nitrate.
STAT.	Frequent intoxications with about 60 p.c. mortality. (In Vienna, 17 cases in 10 years.	45 cases known, with 50 p.c. mortality.	10 cases on record, with 90 p.c. mortality.	A few cases, partly internal and partly external.	25 cases with 60 p.c. mortality.
ÆTIO.	Mistakes concerning the preparations so abundantly used in boiling soap and for washing; suicides.	Accidents and mistakes concerning the much used liquids in chemical laboratories and in the household. Also of the liquefied gas in refrigerating pursuits.	Mistakes for the preparations used in technical pursuits and in the household.	Accident; prolonged handling of lime produces chalicosis pulmonum.	Suicide; mistakes; carelessness; medicinal poisoning; continued handling of baryta salts, which spread a dust around them.
DOS. LET.	About 10.0 grams.	10.0 grams of the officinal 10 p c. solution.	In 2 cases, 1.50 grams.	(?)	3.0 to 15.0 grams.; sometimes very much more.
ACT.	Potassium hydrate debilitates the heart; sodium hydrate to a lesser degree. Both change mucous linings into gelatinous masses, and the blood into hæmatin.	Cauterization of the intestinal tract; inflammation of the respiratory tract; excitation of the central-nervous system.	Same as KOH solution, only less cauterization. The action of K distinctly expressed.	Severe corrosion, and, after resorption, excitation of the central-nervous system.	Even the neutral salts poisonous (same as with oxalic acid). Excitation of the motoric centres of the intestines, brain, and of the musculation of the heart and vessels; paralysis.
SYMPT.	Cauterization and whitish discoloration of the mucous lining of the mouth. The vomit consists of brown, ropy masses. Terrible pains.	Swelling and formation of blisters in the mouth; salivation; vomiting and purging; dyspnœa; glassy sputa; violent spasms; odor of ammonia; perspiration.	Not as violent by far as those of KOH, otherwise identical; pulse remarkably weak.	Inflammatory swelling at place of application; salivation; vomiting; spasms.	Vomiting and purging; convulsions; very slow and wiry pulse; increase of blood-pressure; paresis of the extremities.
DVPT.	Swelling of the mouth; peritonitis; continuous vomiting; urine alkaline and containing hæmatin.	Continued vomiting; gripes; paralysis of the lower extremities; urine contains much alkali, albumin and hæmatin.	Vomiting; gripes; salivation; alkaline urine.	A boy, who fell into a lime-pit, died almost immediately.	Either death within three days or very slow recovery.

EXIT.	Death with or without convulsions; in the first days by collapse; later by ulcers or strictures.	Rapid collapse and death, or ejecting of croup-like membranes by coughing; or pneumonia and œdema of the lungs.	Same as KOH, rapid collapse and death.	In case the eyes have been touched, leucoma; scars on the skin; internally, sores with contraction.	Death by ascending motoric paralysis, or by apoplexy. In case of recovery, long continued weakness.
DIAGN.	Vomit has no odor, is ropy, and strongly alkaline; urine also alkaline.	Same as with the lyes, and, in addition, the odor of ammonia in the vomit and breath.	Same as with alkali-solutions.	Vomiting of white masses of alkaline reaction and limy odor.	The combination of retardation of pulse; spasms; and vomiting of masses containing baryta.
THER.	Vinegar; tartaric acid; malic acid; lemon- or lime-juice.	Same as with the lyes. Gentle inhalations of of H Cl vapors.	Same as alkali-solutions.	Internally as with alkalies; externally, linseed oil.	Hypodermic injection of atropin; washing of stomach with Glauber's salts.
P.-MTM.	Mouth and œsophagus swollen and show whitish discoloration; stomach softened; remarkaably bright red hemorrhagic changes. Perforation of stomach very rarely. Cirrhosis. Periœsophagitis. Empyæma.	The intestinal tract same as with the lyes; pharynx and trachea same as in croup; pneumonic appearance of the lungs; blood in contact with the air turns bright red; ecchymosis and fatty degeneration of the liver; glomerulonephritis; peculiar odor of all organs.	Swelling of the mouth; slight discoloration; stomach same as with acute poisoning by alkalies, softened and red.	The sores upon the skin appear cinnabarred; after internal application, stomach and intestines inflamed; blood remarkably red; urine contains calcium carbaminate.	Generally the stomach is not cauterized; multiple hæmorrhages in the gastro-intestinal tract and in the heart. The urinary canals contain granular masses which form crystals with sulfuric acid. The duodenum and jejunum may be inflamed.
DETECT.	Sodium- and potassium-salts being always present in the body normally, it becomes important to volumetrically determine the quantity of free alkali; the united alkali is hardly to be used for the determination. Potassium-salts form precipitates with tartaric acid and platinic chlorid.	Moist, red litmus paper when held over the vomit or before the mouth turns blue. A glass-rod moistened with strong H Cl forms white vapors of sal-ammoniac. The distillate of the contents of the stomach turns Nessler's-reagent brown. The odor of ammonia can also be used as a test.	Alkalinity is to be determined volumetrically. Compare what was said for KOH. Flame of the Bunsen burner turns violet with potassium-salts. Spectrum.	Calcium-salts give with oxalic- or sulfuric-acid characteristic crystals; the body always contains calcium-salts but never quick-lime. The amounts of calcium must therefore be determined quantitatively according to Fresenius-Babo.	First Fresenius-Babo, then precipitate with sulfuric acid; dry and heat the well washed precipitate, with the addition of a few drops of nitric acid, to a red heat. The heated residue, washed with water, turns the hydrogen-flame green (spectroscope). On dissolving in HCl we obtain upon addition of H_2SO_4 a precipitate of barium sulfate.

IV.—CORROSIVE SALTS.

Salts of the metals in general, not only of the alkali-and alkaline earth-metals, salts of the haloids and of the metalloids, can all of them act as corrosives. From a theoretical standpoint, we have to consider the possibility of four different kinds of such action: corrosion, in consequence of (1) acid reaction; (2) of strongly alkaline reaction; (3) of salt action purely; (4) of salt-action as well as of that of acid or alkaline.

The diluted solutions of all acid salts cauterize in consequence of acid reaction, *e.g.*: *potassium acid sulfate*, which in imbibing plastered wines for some length of time, acts detrimentally; *cream of tartar* or *potassium acid tartrate*, *acid potassium oxalate*, used so largely for the removal of ink-stains. This latter substitute is frequently mistaken for Epsom-salts, or used carelessly, and even its solutions produce genuine corrosion. Many others might be mentioned. It is evident that the corrosion will be more intense and more similar to the action of acids, in proportion to the stronger acidity of the solution.

Corrosion in consequence of strong alkaline reaction will be produced by solutions of certain salts of alkaline reaction, viz: *potassium-*, *sodium-*, or *ammonium-carbonate*, etc. It is obvious that in these cases the corrosion will be proportional to the alkalescence and analogous in the main to corrosion by free bases.

Corrosion by means of salt-action purely, is peculiar to even the most indifferent of all salts, viz: the common table salt, *sodium chlorid*, as such, or in concentrated solution. There are on record fatal cases of grave intoxication, of corrosive character, of man and domestic animals, caused by *sodium chlorid*, *sodium sulfate*, (GLAUBER'S salt), *magnesium sulfate* (Epsom salts), *potassium chlorate*, *potassium sulfate* (Sal polychrest), *potassium-* and *sodium-nitrate* (*nitre*, *saltpetre* or Chili *saltpetre*): Solutions of a certain concentration of each of these neutral salts, behave quite indifferently when in contact with the mucous linings; and in such strength we call it, with regard to the cells of our body, non-poisonous or isotonic. For *sodium chlorid* this is a solution of 0.75 per cent. Weaker solutions are called hypisotonic, and more concentrated oues hyperisotonic. The hypisotonic solutions cause symptoms of intoxication similar to the effects of distilled water, viz., they produce a

colloidal state of the cells; the hyperisotonic ones cause shrivelling and decay of those cells which are directly concerned, and a reactive inflammation around them. It remains an arbitrary matter in how far we should call these processes corrosion. Concerning the isotonic solutions, naturally they have a physiological action in addition to a chemical one. Isotonic solutions of *potassium salts* retain always a specific exciting action upon the heart, and in larger doses they have a paralyzing effect thereon. On the other hand, corresponding solutions of *sodium salts* are devoid of any such action. With regard to toxic qualities, the salts of *rubidium, caesium* and *lithium* occupy a middle position. Isotonic solutions of *salts of barium* remain highly poisonous, those of *strontium* are less active, and those of *magnesium* still less toxic. Internally applied, *magnesium salts* will merely cause diarrhœa; *salts of strontium* will show a diuretic effect. The *haloid-salts* containing oxygen, differ materially from those free of oxygen. We have already mentioned that isolonic solutions of the *alkali-chlorids* are toleraby indifferent; the *alkali-chlorates* have frequently proved fatal. We shall again refer to the latter when treating of the blood-poisons. The same obtains for *alkali-iodates* and *bromates.* The poisonous part in these cases is not the haloid, but oxygen, which can be proved to separate from, or to allow of the separation of the haloid, so that the oxygen-free compounds reappear in the urine. Some of the *salts of metalloids,* as, *e. g. sodium arsenite,* are poisonous to a high degree, even in greatest dilution. Later on we shall again refer to this point.

The corrosion produced by the action of the salt, as well as by that of acid or alkali, is, of course, twofold: The salts of the heavy metals show a combination of salt-action and acid-action, even if they are not of acid reaction. This takes place, because the albuminous substances combine with metal-oxids, forming peculiar metal-albuminates, insoluble in water. If a simple salt of a metal is brought together with albumin in neutral solution, a precipitate forms consisting of albumin, metal-oxid and the acid in question. The latter, however, may be washed away from the precipitate, to which it does not closely adhere. The circulating blood, *e. g.* may do this and then the acid as such can act, causing the symptoms described. Consequently two factors enter into corrosion by *metal-salts,*

viz: the action of the *metal-oxid*, consisting in changing living albumin into dead metal-albuminate, and the action of the acid, which latter represents ordinary acid-corrosion. From the foregoing it follows that the intensity and the character of the action of metal-salts must be conditioned partly by the nature of the metal-albuminate produced, partly by the quantity as well as the properties of the liberated acid. The latter may be only slightly corrosive and it may be contained in relatively small quantity in a basic salt, whose metal-oxid forms an insoluble, tough mass with the nitrogenized tissues (epithel and albumin). This mass will adhere closely to the lower layers of tissue, so that the scab formed prevents further penetration of the poison. In such cases, as we find them with *zinc* and *silver*, the corrosion is restricted to the surface. But the metal-oxid does not always form an insoluble, solid scab with the tissue; frequently, a soft, semi-fluid pap is produced, which is of no effect to protect the underlying parts from further penetration of the poison. With the soluble *metal-chlorids*, the action of the liberated hydrochloric acid is the main feature, *e.g.*, they cause symptoms similar to those described for hydrochloric acid. In addition, the action of *free chlorin* obtains, thus increasing the destructive capacity. It is obvious that in all cases a reactive inflammation follows, provided the case was not immediately fatal.

Of the following two tables, the second one refers to the more important heavy metals, excepting lead, bismuth and tin; the first one contains all those members of the nitrogen-group, exclusive of nitrogen itself, which are poisons in an oxidized state. Phosphorus will be treated of later on; though belonging to the nitrogen-group, it is only poisonous in an oxygen-free state and hardly corrosive.

Of the heavy metals forming corrosive salts, which heretofore have been toxicologically of minor importance, *uranium* is the most poisonous one. Even if hypodermically injected in the form of non-corrosive double-salts, it produces parenchymatous nephritis, hæmorrhagic enteritis, hepatitis, etc. Even in a still smaller dose, it causes glycosuria. Metals now used to a large extent, as *aluminium* and *gold*, are, properly speaking, non-dangerous. *Iron*, *nickel*, *cobalt*, *silver*, *manganese*, and a great many other heavy metals pass exclusively from the blood into the intestine, being secreted by LIEBERKUEHN'S

glands. In case of the circulation of very large quantities within the blood, they are in the beginning partially secreted by the kidneys, causing parenchymatous nephritis. But the main quantity of the metals circulating with the blood is retained by the liver, from where it is slowly transported toward the intestine by lencocytes.

V.—INORGANIC SUBSTANCES WHOSE CORROSIVE ACTION IS OF MINOR IMPORTANCE COMPARED TO OTHER ANATOMICAL CHANGES CAUSED BY THEM.

In the table (pages 98-99) only four substances are discussed, viz.: *tin*, *lead*, *bismuth* and *phosphorus*. It is difficult to draw a line between three of them and those metals treated of in the last chapter. But if we consider the most common forms of intoxication which they produce, we find that the former may be differentiated by the fact that corrosion proper is with them a very insignificant feature, if not totally absent. Admittedly, many points of analogy exist between the action of *phosphorus* and *arsenic;* however, the total phenomenon of intoxication shows many differences between the two. *Phosphorus* is poisonous in itself; its different states of oxidation are not. *Arsenic* is non-poisonous in itself; its combinations with oxygen are. *Phosphorus*, as such, is hardly corrosive; while *arsenic* is very strongly so.

VI.—ORGANIC SUBSTANCES CAUSING GROSS ANATOMICAL CHANGES.

In a wider sense, the substances belonging to this group being very numerous, we shall here omit those which, at the same time, are strong heart- or blood-poisons. We shall then later see that they produce changes of other parts of the body in addition to those of the heart or blood. Some others, as *colchicin*, will produce gross anatomical changes in a lower animal, if the experiment is suitably arranged. But, heretofore, they have alwas proven fatal to man without offering any pathologico-anatomical finding. The principal and not necessarily the local action of the poisons will be here mentioned. Some of them, as, *e.g.*, the poison of *lathyrus*, while not at all producing changes at the place of application, does present very peculiar effects in other parts of the body. It is permissible in adhering to the nomenclature of therapeutics, to denote as "Acria" the substances which are extraordinary by their powerful local irritation.

	ARSENIC AND ITS COMPOUNDS.
SOURCE.	Metallic arsenic, also called cobaltum; arsenious oxid (As_4O_6), generally called arsenic; arsenic acid (H_3AsO_4); potassium arsenite (Fowler's solution); sodium arsenite (Pearson's solution); realgar (As_2S_2); orpiment (As_2S_3); Scheele's green; hydrogen-copper arsenite ($HCuAsO_3$); Schweinfurt green: double cupric met-arsenite and acetate.
STAT.	Most frequent intoxications for last two centuries, even now not rare; mortality low.
ÆTIO.	Murder, suicide; food containing arsenic; drinks; utensils for daily use; wall-papers; clothes; medicinal poisoning. The poison may be resorbed by the stomach, lungs, skin or even by the vagina. Grass, growing in the neighborhood of arsenic-furnaces, and affected by the vapors, may cause intoxications of domestic animals.
DOS.LET.	0.1 gram. arsenic; generally much more is used.
ACT.	Local cauterization, paralysis of the splanchnics, paralysis of the heart, degeneration of the albumin of the body, disturbance of activity of the central-nervous system and skin; voided by means of all glands; in case of small doses, the action of arsenic preparations is slow.
SYMPT.	*Acute poisoning:* Disagreeable sensations in throat, vomiting and purging, difficulty in swallowing, dizziness, headache, pain in limbs, weakness of pulse, cyanosis, cold extremities, fainting, formication, spasms, general paralysis, eczema, erythema, pseudo-erysipelas, catarrh of mucous linings, salivation. *Chronic poisoning:* Catarrh of stomach and intestines, nasal catarrh, bronchitis, laryngitis, conjunctivitis, anæmia, universal eczema, Herpes Zoster (melanosis), dermatitis squamosa, degeneration of mental faculties, paralysis of lower extremities, anesthesia, paresthesia, multiple neuritis, amauroses, epileptiform seizures, nephritis, hepatitis, atrophy of muscle.
DIAGN.	Proof of the presence of the poison in the vomit, urine, and fæces; in acute cases, the cholera-like, rice-water diarrhœas are important for diagnosis. The mummification, according to Zaaijer, is without value.
THER.	After repeated very thorough washings of the stomach, give internally, the antidote—magnesia usta and ferrous sulfate newly mixed; afterwards diuretics, hot baths, and electricity to stimulate the activity of the glands.
P.-MTM.	Contents of the stomach hæmatic; mucous lining partly velvet-like and swollen, reddened, ulcerated, after large doses necrotic; parenchymatous gastro-adenitis; multiple hæmorrhages in intestines; marrow like infiltration of the plaques; dysenteric, fatty degeneration of kidney, liver, heart and vessels, body well preserved, mummification may be absent; in chronic cases, degeneration of muscles and nerves.
DETECT.	The clear solution, prepared according to Fresenius-Babo, is treated energetically with hydrogen sulfid; the yellow precipitate is dissolved in hot ammonium sulfid; filtered, evaporated, and fused with a mixture of sodium carbonate and sodium nitrate. The fused mass is dissolved in water; the filtrate heated with sulfuric acid and conveyed into Marsh's apparatus, in which hydrogen is developed from c.p. zinc and dil. sulfuric acid; arsin (AsH_3) is formed, which, when passed through a calcium chlorid tube, and then through a strongly heated glass tube, gives an arsenic-mirror. In a solution of argentic nitrate, arsin forms a black precipitate of metallic silver. In case small pieces of arsenic have been found in the body, one of them is thrown on a piece of glowing charcoal, giving rise to vapors of a garlicky odor. If another small piece is reduced in a narrow glass tube with charcoal, and the black arsenic-mirror which is formed is heated with potassium acetate, it gives rise to the odor of kakodyl. Concentrated solution of stannous chlorid will deposit in aqueous solutions of arsenious acid, brown flakes of reduced arsenic. The arsenic-mirror is soluble in sodium hypochlorite.

ANTIMONY AND ITS COMPOUNDS.	BORON AND ITS COMPOUNDS.
Almost exclusively tartar emetic, and perhaps the faintly poisonous sulfur auratum, Sb_2S_5 (antimony penta-sulfid) are of importance.	Only boracic acid $B(OH)_3$ to be considered, and sodium bi-borate (borax, $Na_2B_4O_7$, $10H_2O$).
Formerly murders, medicinal poisoning, and mistakes were more common than now. The mortality is greater than with arsenic, since the intoxications are generally of a more profound nature.	Fatal terminations rare; intoxications not infrequent.
Technical and medicinal use. Tartar emetic is resorbed by all parts of the body. Anilin colors that had been fixed by means of antimony have caused intoxications.	Use of boracic acid in surgical dressing and in washing of bladder; internal application of both preparations, even hypodermically.
Scarcely smaller than with arsenic.	Very much greater than with arsenic.
Very similar to arsenic, only that the oxygen-compounds of the latter are more easily resorbed than those of antimony. Voided by means of all glands. Upon the epidermis, arsenic is slightly less irritating than antimony.	Irritation of first passages; disturbances of alimentation; feebleness of heart; decomposition of the blood.
Acute poisoning: Nausea, metallic taste, salivation, non-appeasable vomiting, inflammation of mucous lining of mouth; cholera-like diarrhœa, cold skin, quick, small pulse, dizziness, unconsciousness, spasms in calves of legs, convulsions, collapse. *Chronic poisoning:* Catarrh of stomach and intestines, numbness, vertigo, loss of voice, weakness of muscles, cold skin, weak pulse, albuminuria, profuse diarrhœa, prostration, collapse.	*Acute poisoning:* Vomiting, darkening of the field of vision, paresis of muscles, weakness of heart, purple exanthema, hæmorrhages of bladder. *Chronic poisoning:* Chronic catarrh of stomach and intestine, with loss of appetite; stools contain undigested masses; salivation, marasmus, anæmia.
The unceasing vomiting and cholera-like diarrhœa indicate either arsenic or antimony. The analysis decides.	Is insured by the proof of poison in urine.
After thorough washing of the stomach, internal administration of tannin, magnesia, albumin, milk, analeptics.	Diuretics; washing of the organism.
Inflammation of first passages; parenchymatous gastro-adenitis; intestines as with arsenic; fatty degeneration of kidneys, liver, heart and vessels; also pneumonia has been observed; inflammatory changes in mouth more frequent than with arsenic; swelling, pustules, aphthæ. The œsophagus may show the same appearances.	No satisfactory post-mortem has so far obtained with man. With animals, we find gastro-enteritis, but less than with arsenic.
Organic masses destroyed as with arsenic; precipitation and redissolving also same as for As. The residue when fused with sodium carbonate and sodium nitrate allows arsenic to go into aqueous solution, while antimony remains insoluble. In Marsh's apparatus, antimony also gives a mirror, which consists, not of brown, but black, velvet-like spots insoluble in sodium hypochlorite. If stibin is passed into argentic nitrate solution, we get a black precipitate, but this is not metallic silver, but Ag_3Sb. The filtrate contains no Sb, while in case of As, all the As is found in the filtrate. NH_3 produces consequently no yellow precipitate, as in the case of arsenic, with the antimony filtrate.	The organic masses having been destroyed, the boracic acid obtained is mixed with sulfuric acid to a mush; alcohol is added and ignited; the flame is green. In case copper is present, it is to be separated before this test can be applied, as it also turns the flame green. The same holds good for baryta and thallium. If a piece of turmeric paper is put into a solution of a borate containing hydrochloric acid, it will appear brown on drying.

	MERCURY.	SILVER.
SOURCE.	The most important compound is corrosive sublimate (mercuric chlorid), $HgCl_2$; also the red oxid, HgO, and all other mercuric compounds are strongly corrosive. The mercurous salts and metallic mercury are also poisonous but less corrosive.	Almost exclusively lunar-caustic, $AgNO_3$. Argentic nitrate.
STAT.	The most frequent intoxications of the day.	Now very rare; formerly more frequent.
ÆTIO.	Used as medicine internally, externally and hypodermically; technical applications, and accidents.	Accidents; medicinal poisoning.
DOS.LET.	0.18 grams. corrosive sublimate.	More than 30.0 grms. of argentic nitrate.
ACT.	Local corrosion; also corrosion at places where voided in large intestine, lower small intestine, and mouth; necrobiosis of the epithelia of the convoluted tubules of the kidneys; weakening of the central-nervous system with various phenomena of excitation. The intoxication sets in late after hypodermic injection of metallic mercury (gray oil).	Local corrosion and formation of superficial dry scabs; formation of a black organic compound in the connective tissue of inner organs, and at places where voided in small intestines, and skin.
SYMPT.	*Acute Poisoning:* After internal application of the corrosive salts of mercury, metallic taste, stomatitis, salivation, glossitis, bloody vomit, dysenteric diarrhœa, anuria, œdema of glottis, great weakness, inanition, collapse, black edges on the gums. After hypodermic injection, local swelling and painfulness at the place of application, and also of the surrounding parts. *Chronic Poisoning:* Tremor, erethism, cachexia, necrosis of the jaw, chronic nephritis, rarefying ostitis, paresis of muscles.	*Acute Poisoning:* White scabs in mouth, œsophagus and stomach (?); griping; vomiting of light-colored masses, which turn black when exposed to light. *Chronic Poisoning:* Ulceration of stomach; blackening of cuticle (argyria), of the hair-follicles, of the glands of perspiration, of lips, of the gums. Even if the skin is blackened strongly, euphoria may maintain for years.
DIAGN.	Appearance of the mouth, and electrolytical proof of presence of mercury in the fæces, vomit and urine.	Discoloration of the skin in chronic cases; of the vomit in acute cases. The discoloration remains for life.
THER.	Potassium chlorate for a gargle; albumin, mucilaginous drinks, milk. In chronic cases, warm baths (Turkish). Potassium iodid and sulfur-baths do not act specifically.	In acute cases, sodium chlorid (table-salt), albumin, milk. Nothing can be done in chronic cases.
P.-MTM.	Teeth loose; tongue and gums swollen, ulcerated and bleeding; stomach less inflamed than lower small intestine; main lesion in the large intestine, which is much inflamed and suggestive of the most pronounced bloody dysentery; appendicitis only with herbivora; kidneys have casts containing calcium carbonate; heart, liver, etc., degenerated; peritonitis; loosening of the epiphyses of the long bones; edulla of bones much reddened; phenomena of inflammation of the central-nervous system.	In acute cases, gastro-enteritis; in chronic cases, ulcus ventriculi, and, besides all the parts mentioned above, the blackening of the glomeruli of the kidneys, Glisson's capsule of the liver, the villi of the small intestines, the edulla of the bones, etc. Microscopic sections of the blackened organs will be decolorized by potassium cyanid.
DETECT.	By electrolysis, we may even prove the presence of mercury in the organic masses without destroying them, but it is better to treat them according to Fresenius-Babo, and ppt. the metal as black sulfid; or by alkalies, as yellow oxid; or by potassium iodid, as red iodid. The hydrogen sulfid ppt. is dissolved, for proofs by further reactions, in aqua-regia. All mercury-compounds, upon heating in ignition-tube with soda, yield metallic mercury. (Compare page 41.) Even micro-chemically, we can prove mercury in the epithelia of the intestines and in the kidneys by means of ammonium sulfid.	Upon treatment according to Fresenius-Babo, the silver results as a ppt. (AgCl), which is soluble in ammonia. If the organs are simply ashed and then treated with nitric acid, silver nitrate is formed. A solution of this gives with potassium cyanid a white ppt.; with potassium chromate, an indian-red ppt. Microscopic sections that have been decolorized by potassium cyanid will again turn black with hydrogen sulfid.

CHROMIUM.	COPPER.	ZINC.
Chromic acid (CrO_3 or H_2CrO_4); potassium chromate (KCr_2O_4); potassium acid-chromate ($K_2Cr_2O_7$); chromalum, $Cr(SO_4)_2K + 12H_2O$.	Blue-vitriol ($CuSO_45H_2O$); cupric acetate in genuine verdigris, and cupric arsenite and acetate in Paris-green; basic cupric carbonate, cupric arsenite, cupric phyllocyaninate.	Zinc chlorid, $ZnCl_2$; zinc sulfate (white vitriol, $ZnSO_4 7H_2O$); zinc oxid, ZnO; zinc carbonate. (Calamine, Smithsonite.)
Formerly, mainly in England, frequent; now everywhere rare.	Formerly frequent in France; now everywhere rare.	In England, even to-day frequent; elsewhere rare.
Manufacture of chromium-preparations; medicinal use; accidents.	Accidents by vitriol much used technically; addition of copper to vegetables very rarely poisonous.	Murder, suicide, medicinal and œconomic poisoning; mainly accidents.
1.0 grm. of potassium acid chromate; much more of the salts of the oxids.	10.0 grms. of the sulfate or acetate should be sufficient.	7.6 grms. zinc sulfate; 6.0 grms. zinc chlorid.
Local corrosion and inflammatory action at places where voided, *e.g.*, in kidneys and mucous lining of intestines; formation of met-hæmoglobin by means of the acid and acid-salts; grave impairment of the central-nervous system.	Strong corrosion of the first passages mainly by vitriol; formation of met-hæmoglobin; marked effect upon the central nervous system; paralysis of the musculature; discharge of copper by the glands of the intestines, saliva, bile, urine, and skin.	Same as copper, only zinc less poisonous and has more of a sedative action on the nervous system; voided mainly by the glands of the stomach; only sparingly of accumulative action.
Acute Poisoning: The swollen and ulcerated mucous lining of the mouth is at first colored yellowish-red, afterward bluish-green; bluish-green to yellow vomit; violent colic; thin, bloody stools; thready pulse; scanty urine containing albumin. *Chronic Poisoning:* In case of handling dusting chromates, ulcerations of the hands, arms, penis, septum of nose; bronchitis, conjunctivitis, and nephritis.	*Acute Poisoning:* Green ecabs in mouth, green vomit, abominable metallic taste, salivation, sensitiveness and inflation of abdomen, brownish-red diarrhœa, small pulse, skin cold, icterus, paresis of limbs, collapse. *Chronic Poisoning:* Pale-green appearance, copper-edge on teeth, copper-colic, catarrh of stomach and intestines, marasmus, copper-paralyses (?).	*Acute Poisoning:* Mucous lining of mouth bloody or white and wrinkled; strong metallic taste; salivation; vomit whitish, later of blood-color, mainly after $ZnCl_2$; diarrhœa of coffee-brown color; general debility, vertigo, cramps in calves of legs, cold perspiration. *Chronic Poisoning:* Catarrh of stomach and intestines, fever of brass-founders, chills, pains in back, weakness of muscles, pains in forehead, perspiration, gradual convalescence.
Characteristic discoloration of the mouth in acute cases; ulceration of septum of nose and skin in chronic ones.	Characteristic discoloration of mouth and vomit, and in chronic cases of hair and skin.	We must determine the presence of a colorless metallic poison which is neither mercury, silver, nor lead.
Washing of stomach with sodium bi-carbonate; internally, magnesium carbonate or plumbic acetate.	Washing of the stomach with potassium ferro-cyanid; internally, albumin, milk, magnesium oxid; later on, Turkish and Russian baths, and electricity.	Same as with copper; in addition, tannin and alkaline phosphates.
Mucous lining of mouth swollen, ulcerated and discolored. Mucous lining of stomach ecchymosed and ulcerated, as also those of small and mainly of large intestine; fatty degeneration of liver and heart; kidneys show exudates and necrosis of the epithelium of the convoluted tubules. In chronic cases, rhino-necrosis, ulceration of skin and bronchitis.	Gastro-enteritis from mouth to anus; perforation of stomach, small and large intestines; greenish mucous lining of intestines, which turns deep-blue when touched with ammonia; ecchymosis of serous membranes; fatty degeneration of liver and heart; parenchymatous nephritis; atrophy of muscles (?).	Mucous lining of mouth tanned; mucous lining of stomach corroded and ecchymosed—even detached in shreds; walls of stomach leather-like; small intestine hyperæmic; soft membranes of the brain; lungs and kidneys rich in blood, and in severe cases the latter evidence parenchymatous inflammation.
After Fresenius-Babo, a green liquid, from which ammonium sulfid will ppt. grayish-blue hydrate, which, when dissolved in alkalies and treated with plumbic peroxid, will give a yellow solution; decant or filter this, and add acetic acid, which will ppt. plumbic chromate of yellow color. Free chromic acid will be reduced by alcohol when heated, turning green. Hydrogen peroxid will give blue coloration soluble in ether.	After treatment, according to Fresenius-Babo, a liquid is obtained, colored green by cupric chlorid and turning deep-blue upon addition of ammonia. (With chromium the green coloration is due to chromic chlorid.) Hydrogen sulfid will ppt. black cupric sulfid, and potassium ferro-cyanid will give a brownish-red ppt. (KOH blue ppt., see page .) The copper is contained in the blood, not in the serum, but united with the hæmoglobin.	After Fresenius-Babo's treatment, we obtain a colorless solution, from which, after preceding neutralization, ammonium sulfid will ppt. white zinc sulfid. Potassium hydrate and ammonia ppt. white zinc hydrate, soluble in excess. Alkali-carbonates ppt. insoluble zinc carbonate. Potassium ferro-cyanate ppts. white zinc ferro-cyanate. In the blood, zinc is combined with the hæmoglobin.

	TIN.	LEAD
SOURCE.	The soluble salts formed from metallic tin in preserve-cans. In former times, occasionally stannic chlorid ($SnCl_4$) contained in spiritus fumans Libavii.	Only sugar of lead has actually to be considered as a corrosive compound; generally we have to deal with the non-caustic oxid, viz.: litharge PbO; minium Pb_3O_4; with white lead, plumbic chromate, or water or food containing lead, and with lead subacetate.
STAT.	Genuine, pure tin-poisoning rare. Mortality very rare.	To-day one of the most common intoxications; formerly, however, yet more frequent. Fatal cases now very rare. Mortality now very low.
ÆTIO.	Generally from partaking of canned vegetables, etc.	Partaking of water that has stood in lead-pipes for a long time; sometimes in wine and food; toys containing lead; handling leaden objects (plumbers, lead-burners, and painters). Toys.
DOS. LET.	Unknown, but very high; only one fatal case so far.	Lead sub-acetate $[Pb(C_2H_3O_2)_2+2PbO]$ solutions, more than 20.0 gms.; sugar of lead, plumbic acetate $[Pb(C_2H_3O_2)_2+3H_2O]$, more than 50 gms.
ACT.	Cauterization only through stannic chlorid; paralysis of the central-nervous system; pareses and paralyses; voided by the inflamed mucous lining of the intestines; development very slow; deposits of tin in the brain, marrow, liver, kidneys and muscles.	Metal-tan at place of application, but only after large doses. After resorption, as observed in animals, excitation; and with man, paralyses of the central-nervous system, alimentation damaged, tetanic contractions of intestines, ample deposit of uric acid in kidneys, atrophy of muscles. Development very chronic; deposit of lead in many organs; voided by the glands of the intestines, kidneys, skin, saliva, and milk.
SYMPT.	Gastro-enteritis, purging and vomiting, marasmus, chronic catarrh of stomach and intestines, pareses of muscles, ataxia, impaired muscular sense, urine contains albumin.	In case of severe *acute poisoning*, salivation, metallic taste, lead-border on teeth, whitish-gray coloration of the mouth, spasms of the stomach, abdominal colic, constipation, perspiration, formication, pulse hard and much retarded, anæsthesia, stupor, unconsciousness, paralysis of the extremities (wrist drop); with animals, spasms and raving mania. In *chronic cases*, disturbances in general, colic, arthalgia, anæsthesia, amblyopia, amauroses, delirium, eclampsia, paralyses of the extensors (Radialis), gouty kidneys.
DIAGN.	By anamnesia.	Colic with constipation, retardation of pulse, lead-edge on teeth, paralysis of the extensors (Radialis).
THER.	Hot baths; avoiding of anything containing tin and of tin-lined vessels containing acid food. Empty at once tin preserve-cans.	In *acute cases*, sodium- or magnesium sulfate, albumin, milk. In *chronic cases*, potassium iodid, hot baths, electricity; in case of colic, atropin hypodermically, as well as large doses of olive-oil and opium internally. Prophylactic treatment very important.
P.-MTM.	No autopsy on man. Chronic state of excitation of mucous lining of stomach and intestines; grayish-brown coloration in the neighborhood of the ileo-cæcal valve; swelling of the follicles.	Cauterization of the intestinal tract very rare; diminution of the elasticity of the walls of the arteries in consequence of alteration of the endothelium and of the musculature; atrophy of the extensors of the lower part of the arm, and, independently of this, of their nerves. Kidneys contracted and gritty from urates; formation of vacuoles and turbid swelling of the parenchyma of the liver and many other organs; accumulation of serous fluid under the membranes of the brain and spinal cord.
DETECT.	Tin is obtained together with antimony and arsenic in the ppt. with H_2S of the liquid obtained, according to Fresenius-Babo. The leather-yellow stannous sulfid is soluble in ammonium sulfid and in alkaline sulfids, but insoluble in ammonia. Potassium cyanid reduces tin compounds when heated, without volatilization. From dilute gold-solution, stannous chlorid will ppt. Cassius purple of gold.	Treatment according to Fresenius-Babo, but filter while boiling-hot. The filtrate will deposit, when cool, plumbic chlorid; H_2S will ppt. black plumbic sulfid; H_2SO_4 ppts. white plumbic sulfate; HCl ppts. plumbic chlorid, insoluble in ammonia and not changed by it in color. Before the blow-pipe with soda, in reduction-flame, metallic lead is formed. Yellow potassium ferro-cyanid ppts. plumbic ferro-cyanid which is white. The presence of the metal has been proved in the bones, kidneys, liver, spinal cord, brain, linings of the intestines, and muscles of the heart. Even during the life of the patient, lead will sometimes be proved to exist in the urine and fæces and upon the skin. Urine and fæces must be previously destroyed. The skin will turn black when treated with ammonium sulfid.

BISMUTH.	PHOSPHORUS.
Bismuth sub-nitrate; bismuth citrate with ammonium citrate.	Only the non-oxidized, free yellow phosphorus and phosphin (PH_3 or P_2H_4) are to be considered. The oxygen compounds of phosphorus are non-poisonous, therefore also phosphorous acid often found in the corpse, phosphoretted oil, phosphorus-matches, phosphorus-salves, are of strong action; red phosphorus acts but to a trifling extent.
In the last decade a little more frequent than formerly; generally rare; mortality very low.	Until lately a very frequent intoxication, which, *e.g.*, in France, formed 33 p.c. of all poisoning cases; mortality, 55 p.c.
Used in surgical dressings, internally for summer complaints of children and others.	Manufacture of phosphorus and phosphorus-matches; murder, suicide, abortion, accidents.
Unknown, but very large; only a few fatal cases on record.	Even .05 gms. may prove fatal after internal application. The amount of vapor necessary to cause necrosis of the jaw is still unknown.
Cauterization of the mouth scarcely present, but there is inflammation of the mouth and inflammation of the colon by voiding of the bismuth; nephritis, from the same cause. With animals, spasms analagous to lead-spasms; with man, paralyses. Voiding also by the milk.	Very slow, since resorption is slow; local excitation may be absent; grave disturbances of metabolism; degeneration of the body-albumin to fat, leucin, tyrosin, sarcolactic acid, and phosphorus-bases. Multiple hæmorrhages by degeneration of the vessels; very pronounced icterus; fatty infiltration and degeneration of the liver, later cirrhosis; fatty degeneration of the kidneys and heart; in case of the kidneys, ending with shrivelling. The inhaled vapor will produce ostitis and necrosis of the jaw, particularly when the teeth are ulcerated.
Stomatitis and black discoloration of the gums, glossitis, catarrh and ulceration mainly of the large intestine, albuminuria. If introduced into the pleura, pleuritis; into the cavity of the abdomen, peritonitis.	Vomiting of matter of an odor of garlic and luminous in the dark; then for two days, euphoria, then icterus; pain in the stomach; vomiting of masses containing bile and blood; sensitiveness of the abdomen; increase of liver-dullness; the breath is luminous in the dark; hæmorrhages of nose, uterus, etc., and into the skin ecchymoses; headache, somnolence, fever, small, rapid pulse; renewed vomiting of dark bloody masses; coma, death. In the urine, biliary matters, fat, blood, leucin, tyrosin, lactic acid, bases containing phosphorus, and casts.
Discoloration of the mouth and inflammation characteristic.	The odor and luminosity of the vomit, icterus, hæmorrhages into the skin and urinary tests insure diagnosis.
Iron saccharate for counteracting H_2S in intestine; aqueous diuretics; potassium chlorate for a gargle; use of baths of unknown effect.	Cupric sulfate as an emetic; washing of the stomach with potassium permanganate in solutions of 0.5 p.c. or with hydrogen peroxid; give several times a day one or two capsules containing 1.0 gm. of *old* oil of turpentine; alkaline drinks to increase the alkalescency of the blood; transfusion and washing of the organism with solutions containing common salt, sugar, and soda.
State of mouth similar to mercury; large intestine black and ulcerated; microscopically, brownish-black granules of bismuthic sulfid are found in the vessels of the mucous lining; stomach and small intestines but little inflamed.	The highest degree of icterus of the skin, the sclera, and all inner parts; multiple hæmorrhages into the skin and into the organs most varyingly, in particular underneath the pleura; in the stomach, gastro-adenitis, which frequently extends into the duodenum; catarrh of the bile-ducts; liver in the beginning greatly enlarged, soft as butter and of a saffron-color and very fatty, later on small; all cells are filled with drops of fat; the microscopic finding of the kidney is similar. Muscles of the heart and skeleton show fatty degeneration. The capillaries of the cortex of the brain show fatty degeneration. The jaw shows new-formation of bone-substance.
Liquid obtained according to Fresenius-Babo will allow the bismuth to be pptd. as brown sulfid, insoluble in ammonium sulfid, but soluble in conc. HCl or in HNO_3. If the liquid be cautiously evaporated and then poured into much water, there will be a white ppt. of BiOCl. The chlorid is soluble in alcohol and will not ppt. on addition of H_2SO_4; the aqueous solution will be pptd. by alkalies; this ppt. is insoluble in KOH. It is questionable whether or not there are other black compounds besides bismuthic sulfid present in the intestinal mucous-coat.	The organic masses are acidulated with H_2SO_4 and heated in a flask supplied with descending condenser (Mitcherlich's apparatus). Phosphorus passes over with the aqueous vapor, and is luminous in the dark, provided that alcohol, ether, oil of turpentine, cupric sulfate, and hydrogen peroxid, etc., are absent. Argentic nitrate-paper will be blackened by vapors of phosphorus, but not so plumbic acetate-paper. Hydrogen developed from zinc and HCl in the presence of phosphorus contains phosphin, and will burn with a green flame. In a solution of argentic nitrate, phosphin will cause a black ppt. of argentic phosphid. It is also practical to extract the parts of the cadaver with a mixture of carbon di-sulfid and ether-alcohol. Then add copper-turnings to the extract, upon which cupric phosphid will deposit. On taking out the copper, washing well and treating with Zn and H_2SO_4, phosphin will generate. Phosphin may be formed from phosphorous acid in the same manner as from phosphorus, by means of Zn and HCl. It has never yet been observed that from corpses of the non-poisoned, under the influence of cadaver-bacteria, phosphin had formed from phosphorus-compounds normally present in the body.

All substances belonging to the acria have in common the following action: They produce, in the first place, a dilatation of the vessels of the skin, of the mucous membranes (especially in the intestinal canal), and of the subcutaneous connective tissue. This then causes hyperæmia, manifested by reddening, swelling, and elevation of temperature on the part of the body concerned.

The process may stop there and retrocede without having been actually detrimental. But if the irritating action of the poison extends further, coagulable serum, viz., so-called plasma, will exude from the capillaries, the direct result of which is an excessive engorgement of the lymphatics by the normal alimentary liquid of the tissue. Thus œdema is produced. The plasma by coagulation, may separate fibrin. Soon after exudation of the plasma, as a rule, a stage of emigration of leucocytes follows. The artificial production of this passage of leucocytes as observed under the microscope, in the tongue or the mesentery of the frog, or in the mesentery of all warm-blooded animals alike, we call COHNHEIM'S experiment. This experiment is of great significance in pharmacology, because there exist not only numerous substances which, just like the acria, favor its perfection, but reversedly, others as, *e. g.*, *quinin*, which prevent the emigration of leucocytes.

It is of additional importance to know that in case of inflammation, besides the leucocytes, red blood-corpuscles, although in much smaller number, also pass through the walls of the vessels; this occuring, as a rule, at points where leucocytes had previously made their egress, through the orifices in the walls of the vessels between the endothelial cells, viz., the so-called stomata or stigmata of the cement-substance.

This diapedesis of the moveless red blood-corpuscles follows much more abundantly in case of venous engorgement as when inflammation is present. This proves that the mere filtration-pressure is indeed capable of forcing soft lumps of protoplasm through the endothelium. We may suppose, therefore, that in case of inflammation, also a favoring action for the passage of leucocytes is to be attributed to the blood-pressure.

However, the significance of this factor is materially les-

sened in the case of inflammation, as here the pressure in the vessels concerned is more frequently reduced than increased. Under these conditions another factor obtains which is suited to much more than compensate for the reduction of the blood-pressure, viz., a greater penetrability of the walls of the vessels. The explanation of this process is to be found in the fact that when inflammation is present, the serrated and tortuous lines of the cement-substance between the endothelial cells of the vessels are broadened, and the stomata contained in them enlarged and increased in number, thus allowing dissolved colloids to pass out. It seems that this change is explained in a purely physical manner by the dilatation of the tubes of the endothelial intima, following of necessity upon the increased caliber of the vessel as a whole. After the retardation of the blood-current had been perfected by the cause of the inflammatory process acting upon the walls of the vessel, and widening its lumen, then the wall whose repose depends upon adherence of the liquid, will be extended; into this the leucocytes are pushed, because of the small amount of *vis viva* which they possess. They are, so to say, pushed aside by the red blood-corpuscles, and accumulate more and more in the layer which is in relative repose. This is the stage of edge-layer formation.

The leucocytes, in contact with the wall of the vessel, now changed by the agency causing the inflammation, are induced by this same agency, acting in a chemo-tactile manner, to abandon the globular form, their form of rest, and to emigrate by amœboid movement towards the chemo-tactile centre. In consonance with or after the passage of the leucocytes, an exudation of plasma may occur, which soon coagulates to fibrin. This, however, is immaterial since the now following histolysis, viz., the envelopment by pus, emanating from the leucocytes of smaller or larger parts of tissue, redissolves the fibrin. We can perhaps best comprehend this liquefaction-process by assuming thst a digesting enzyme is furnished by the leucocytes to the surrounding bodies; this is then supposed to change them into propeptones (albumoses), liquefying them at the same time. In fact, pus will always give the peptone-reaction, which, however, is also peculiar to the

albumoses. It is not proven that genuine peptone is formed in this case. The histolytic process need not of necessity dissolve uniformly the whole piece of tissue affected by the excitating agent; it mainly concerns the circumference, while the central part, where the action was most intense, will become necrotic and isolated, and, if possible, eventually breaking through the skin, be thrown off. All of the occurrences thus far discussed are together called, according to old tradition, "inflammation." Preferably this aseptic inflammation changes into a bacterial one, since enzymes, which may happen to be in the blood or in the neighborhood of the centre of attack, will be attracted by this process of disease, and will greatly multiply in this disturbed seat. Thus it happens that the physician never witnesses an aseptic inflammation proper. The difference in appearance of an inflamed central seat without bacteria, and one with bacteria, is to be found in the number of leucocytes; as soon as the enzymes step in, the emigration of the leucocytes increases as well as the histolysis which they cause.

We have now to consider as a particular kind of inflammation, that which is achieved by coagulation of blood and lymph; this is the case, *e. g.*, in the wall of the stomach when there exists intoxication by acids. Some poisons, as *abrin* and *ricin*, bring about such a coagulation of vessels exclusively, without simultaneous cauterization.

In conclusion, we note that a part of the body may become hyperæmic to such a degree, that in the very beginning a genuine inflammation is simulated, but from this under certain conditions, the true inflammatory process later on really follows. This may occur in a purely nervous manner by paralysis of the vasomotor governing a given locality. This holds true mainly for the intestinal canal, with poisons which paralyze the splanchnic nerves, as, *e. g.*, *snake-poison*.

A. A.—ANIMAL POISONS. (TABLE, PAGES 106-107.)

Experiments with chemically pure, exactly analyzed substances have been perfected only with *cantharidin*, which is the anhydrid of *cantharidic acid* of analogous action. In toad-poison, we have to deal with a mixture of *carbylamin*-substan-

ces, to which reference will be made later, with *phrynin*. This latter most interests us here, but chemically it is still awaiting research. For the garlic-toad, still another substance of an alliaceous odor, is to be considered. The toad-poison is contained not only in the secretion of the poison-glands, but also in the blood. The *samandarin* or *salamandrin*, of the *fire-salamander* has an alkaloidal character; it is a violent local excitant and kills under tetanic convulsions similar to strychnin. The poison of the *water-salamander, triton cristatus*, is not identical with the foregoing, but it also causes very pronounced excitation. The poison of *scorpions* (*buthus, scorpio, euscorpius*) is of action similar to the preceding ones, but it is of albuminous nature. The *genuine spiders* possess, just as the animals mentioned above, genuine poison-glands, while these are missing in the *pseudo-spiders*, which, however, produce an excitating saliva. The genuine spiders may contain the poison, aside from the poison-gland, in the juices of all other parts of the body. This is particularly true of the *diadem-spider*, whose poison from its poison-gland acts less strongly than that of the body-juice. All spider-poisons are toxalbumins which are to a greater extent digested within the stomach of warm-blooded animals, while they possess a terrible action when starting from the blood or the subcutaneous connective tissue. However, it is said of dogs that they will partially void through the mucous lining of the stomach, *snake-poison*, beginning from within the blood-circulation. By the term *fish-poison*, used in a wider sense, we understand not only the poison produced by certain fish in a state of health, but also such toxic products as generate in diseased or rotten fish. We shall here discuss only the first-named, which, however is subdivided into two classes: (1) Some fish possess genuine poison-glands which are in connection with thorny fins, while (2) others carry the poison in a form not available against themselves, within the blood (*eel-poison*), or produce it within the genitals (*fugu-poison*). The action of the latter starts from the intestinal canal of man, even if the fish had been partaken of in a boiled state. Toxicologically, the most important animal poison is *snake-poison*, of which there exist many kinds. Some of them are not rendered wholly inocuous by boiling for a

short time, and most kinds produce both local and remote marked effects.

B. B.—VEGETABLE POISONS. (TABLE, PP. 108-110.)

The great majority of the numerous pertaining vegetable poisons are mainly of local action. In regard to up-to-date chemical research, this branch of our subject is as defective as that of the animal poisons; consequently we must not be surprised to learn that satisfactory experiments also are unfortunately missing. But the practicing country-physician is interested in these poisons, because it is not a rare thing for him to see light and more severe intoxications caused by them in children, and in domestic animals. Therapy is generally of one kind. It consists in ridding the stomach of the poison and in administering cooling and mucilaginous drinks. In case of external intoxication, ice-compresses are indicated. Many of the poisonous plants in question share in common the feature that on drying, they either partly or wholly lose their toxic quality, and consequently render experimentation with these poisons a difficult matter.

Did not space forbid it, we might greatly increase the number of substances contained in the following tables by enumerating all of the spices as well as the drastica. So it may at least suffice to mention these two classes of substances. Finally, the so-called *saponin*-substances belong in a certain sense in this place. It is true that they are not excitants of the epidermis, yet producing violent effects upon mucous membranes, causing conjunctivitis and keratitis with the eye, acute nasal coryza with accompanying sneezing, and watery secretion, and furthermore, gastroenteritis. But in so much as all of these saponin-substances possess in addition still another, quite peculiar, action upon the blood, it would seem more suitable, in order not to excessively increase the number of vegetable poisons, to briefly mention them here, enumerating and discussing them more in detail when we are considering the blood-poisons.

The last table mentions three kinds of intoxication, markedly varying from those of the preceding one, the characteristic feature consisting in anatomical changes not of the alimentary tract, but of other organs, above all in the nervous system. We thus conclude the series of vegetable poisons,

which cause gross anatomical changes, and stop to merely mention the excitating enzymes of the micro-organisms, *e. g.*, *streptococcus.*

C. C.—ARTIFICIALLY PREPARED POISONS.

The large number of existing artificially prepared substances which cause anatomical changes, have but small interest for the practicing physician. We enumerate: chrysoidin, malachite-green, Bismarck-brown, fast yellow, wasserblau, fast blue, spritblau, methylene-blue, Victoria-blue, neublau, methyl-violet, gentian-violet. These are said to cause local manifestations of excitation and other phenomena of intoxication in workmen handling them for some time, and with animals after much dosing. When considering the blood-poisons we shall mention a few other dye-stuffs.

B.—BLOOD-POISONS. (TABLES, PP. 113, 115, 118.)

It is impossible to understand the protean action of poisons of this group before having elucidated their behavior with reference to the blood. In *praxi* this is hardly ever the sole change which they cause, but it is the one which may be readily studied *extra corpus* and which strikes the keynote for the understanding of most of the other actions.

We here present an abstract of the gross anatomical changes of the blood, caused by corrosive acids, alkalies and caustic salts.

The poisons here discussed and considered in their action *extra corpus*, do not act upon the serum but solely upon the corpuscles. Every one of these actions upon the blood may therefore be studied on red blood-corpuscles suspended in physiological sodium chlorid-solution. However, the manner of action differs. A **first group** of blood poisons changes the physical condition of the red corpuscles in such a manner as to render them gummy, so that on mutual contact they will stick to each other and form lumps having the appearance of red sealing-wax. These clog up the vessels and thus cause the gravest disturbances of blood-circulation. We will only mention one of these poisons, *ricin*, contained in the seeds of *ricinus communis*, (*euphorbiaceae*). We find, on taking castor-beans internally, or the pressed cakes of castor-beans, since the castor-oil does not participate in this action, that an indeterminable amount of *ricin*, of albuminoid character, is deprived of its

	CANTHARIDIN.	TOAD-POISON.	ACULEATÆ-POISON.
SOURCE.	Lytta vesicatoria L., cantharides, Spanish fly, Meloe proscarabæus L., Meloe majalis L., Mylabris, Cantharis. Our Lytta does not contain above 0.5 p.c. cantharidin; the Argentinian, 2.6 p.c.; the different kinds of Mylabris of the trade, 1.0 p.c.; the different Meloes much less. Emplastrum cantharidum, tinct. cantharidum, potassium cantharidate.	Bufo cinereus L., common toad; Bufo calamita, crosstoad; Bufo viridis Laur., green Italian toad; Pipa. Pelobates fuscus, garlictoad; Bombinator igneus. Alytes obstetricans. Juice of poisonous glands.	Apis mellifica L., honey bee; Xylocopa violacea Fabr., wood-bee; Bombus hortorum, and B. lapidarius, bumble-bee; Vespa vulgaris L., wasp; V. crabro, hornet; Formica rufa, forest ant; Formica herculanea; Ponera; Myrmica; Attus ferox; Culex, gnat; Tabanus, gad-fly (horse fly).
STAT.	Tardieu quotes 23 cases in 12 years; now rarer. Mortality great.	Only slight external intoxications are known.	Known isolated fatal cases in man and the horse.
ÆTIO.	Murder, suicide, quack practice, medicinal poisoning, mistake.	On touching the animals, secretion may come in contact with the human skin, eye, nose or mouth.	Attack by a swarm; emptying of a bee-hive; irritating the animals.
DOS.LET.	1.5 freshly powdered cantharides; 30.0 tincture; 15.0 plaster.	Unknown, and not the same in all species of toads.	Unknown, and varying with the different species of animals.
ACT.	Strongest local excitation at the place of application and where voided (kidneys). Priapism. Excitating action upon brain and spinal cord.	Severe local excitation. After resorption, the heart and vessels are influenced in a manner simulating the action of digitalin.	Strong local irritation around the stung point, in which the sting frequently remains.
SYMPT.	Skin blisters; internally burning and blistering in mouth; difficulty of swallowing; salivation; vomiting and bloody stools; violent pains in kidneys and urethra; urine very scant, containing albumin and blood.	Burning and itching, mainly of the mucous membranes; conjunctivitis; keratitis; swelling of the nose; inflammation of mouth; retardation of pulse; increased blood-pressure; nausea, vomiting, diarrhœa.	Reddening, swelling, burning, itching, extravasation, collateral œdema; fainting, delirium, vomiting, aphonia, urticaria. The temperature of the body may rise or fall.
DIAGN.	Formation of blisters and presence of green particles of the elytra.	The handling of squirting toads.	The presence of the animals or of the remaining sting.
THER.	Repeated energetic washing of stomach. Internally, mucilaginous drinks with addition of opium. Cupping near the kidneys. Warm, protracted sitz-baths. Avoid oily internal medicines.	Local cooling by means of ice. Cocain. The injured eyes must be treated by a specialist.	Local cooling by means of moist earth, scraped or sliced raw potato, cold water, ice, touching with ammonia. No scratching permissible. Cocain.
P.-MTM.	Gastroenteritis hæmorrhagica, particularly also of duodenum; glomerulo-nephritis; congestion and ecchymosis of ureters, of bladder and of urethra. The contents of the blisters is serous.	Accounts referring to man are not recorded. Leucoma, in the eye. The skin of the lips is exfoliated. With frogs, the heart stands still in systole.	Inflammation of skin from slighthest to severest degree, with partial gangræne and suppuration. Nothing definite known about changes of inner organs.
DETECT.	In case powders or plasters had been used, the finding of green particles is diagnostic. To prove the presence of cantharidin, boil with KOH, forming potassium cantharidate, acidulate with H_2SO_4, and shake with chloroform. The residue of evaporation is dissolved in formic acid, and, on again evaporating, doubly-refractive crystals are formed, which, dissolved in oil, blister the skin.	Imperfectly investigated. Evidently we have to do with a mixture of substances, of which phrynin is a volatile base. The carbylamin substances may be distilled off. It is questionable if toxalbumins are present, but this seems to be very likely.	In regard to formic acid see page . In addition, there evidently exists an enzyme responsible for the main action, but which is chemically absolutely unknown. In addition, undecan is present, which is volatile. It is questionable whether formic acid is to be found in the urine.

SPIDER-POISON.	FISH-POISON.	SNAKE-POISON.
Lycosa tarantula, genuine tarantula; Trochosa singoriensis, Russian tarantula; Cteniza sarmentaria; Chiracanthium nutrix; Epeira diadema L., diadem-spider; Lathrodectes tredecimguttatus (Malmignatte); Lathrodectes lugubris; (Karakurte) or black spider; Galeodes araneoïdes (Solpuge or Phalange).	Trachinus draco L., Serranus Scriba, Trachinus vipera L., Synanceia brachio, Cottus scorpio, Thalassophryne reticulata, Murnæna Helena, Scorpæna, Stomia boa, Tetrodon, Fugu, Anguilla, Eel, Conger, Petromyzon, Lamprey.	Vipera berus; Pelias prester, black variety; Vipera ammodytes; Vipera Redii; Crotalus durissimus and horridus, rattle-snake; Naja tripudians; Naja haje; Pelamys.
The bite of Lathrodectes has killed a number of human beings and thousands of domestic animals.	Fatal cases have only occurred with any frequency after eating Tetrodon, mainly in Japan.	Very frequent in India. The data in regard to mortality in Germany varies between 2.8 and 25 p.c.
Bite received while sleeping, or when marching barefooted over the steppe; irritating the spiders.	In some cases the hands are injured by handling the thorny animals; otherwise internal intoxication after eating.	Always bites, mostly into naked hands or feet.
Of the poison of lathrodectos, enumeration by milligrams suffices if the capillaries are invaded.	The fresh blood of 0.6 kgr. eels acted almost fatally; for the other fish the dose is unknown.	According to experiments on animals, the fatal dose is very small, to be expressed only in milligramms.
No local action at all with lathrodectes, but paralyeis of central-nervous system and formidable pains. With all nther spiders, inflammation, beginning at the bitten point.	Partly analogous to the poisons of aculeatæ and spiders. The genitals of tetrodon contain a locally excitating poison similar to curare. Fresh blood of the eel and lamprey causes vomiting and purging.	Varies according to the species in question. Generally local hæmorrhagic inflammation ensues; dilatation of vessels and central paralysis.
The bites of different species produce local changes of varying intensity, generally analogous to those discussed with the aculeatæ. Lathrodectes causes screaming with the pain, this latter not being local. Paralyses enduring for months.	Corresponding to the just-mentioned differences, varying action, viz., inflammation of skin, gastro-enteritis, paralysis, spasms. Neither mydriasis nor general exanthema are symptoms observed in consequence of handling or partaking of the mentioned species.	The bitten part swells, becomes painful and assumes a bluish color. Then the whole extremity swells and is covered with hæmorrhages into the skin. Next the trunk is attacked; paralysis, suffocation and death, generally with inflation of abdomen.
Easy, in case the spider is found; otherwise very difficult.	Only possible if the fish in question is at hand.	The local phenomena and the impressions made by the teeth leave no doubt.
For bites of the ordinary spiders, just as with the iniuries received by aculeatæ; for those of Lathrodectes, hot baths and internally opium. A popular remedy against the bites of all spiders is: spiders crushed in oil.	Purely symptomatic. Thorny fish must not be handled; new strange fish should not be eaten.	Ligating of the extremity and immobilization. Suck out, cauterize or cut out; potassium permanganate; locally, chromic acid; internally, alcohol, ammonia; hypodermically, strychnin. Washing of stomach. Many fanciful and unique expedients peculiar to different localities—all of doubtful value.
For the ordinary spiders, inflammation of skin, same as with aculeatæ; for lathrodectes, sometimes extensive coagulation of vessels.	Inflammation of the injured parts of skin. Hæmorrhagic gastro-enteritis. The abdominal veins surcharged with blood.	Rapid putrefaction of the dark-blue-colored corpse. Around the bitten point exudation of serum and blood; red-colored, serous extravasations into the cavities of the body; multiple hæmorrhages; mucous coat of intestines impregnated with blood.
With lathrodectes, we have to deal with a toxalbumin, which loses its activity even when treated with alcohol; it has no special chemical reactions; it is found in every part of the body of the spider. With the others we have to do with the secretion of the poison-glands and salivary glands, which have never yet been chemically approached.	We possess some chemical knowledge only of one poison of Fugu and one of Eel (ichthyotoxin); the locally excitating substances and tests for these are entirely unknown. Here we have evidently to do with enzymes. In case of the so-called barbel-cholera produced by eating barbel, ptomaines are possibly the cause.	We generally have to do with a mixture of toxalbumins, from which basic complexes are easily split off. Specific reactions are missing. The poison is contained in the gland corresponding to the parotid, which only extends beyond the head with caudus rhombeatus and with callophis. Evidently the poison of different groups of snakes is not identical.

TABLE OF INTOXICATIONS PRODUCED BY A NUMBER OF PLANTS WHICH CAUSE GROSS ANATOMICAL CHANGES.

No.	NAME OF PLANT.	NAT. FAMILY.	NAME OF POISON.	POISONOUS PART.	MODE OF ACTION.
1	Daphne Mezereum; D. Laureola; D. Gnidium; D. Cneorum; D. Striata.	Thymeleæaceæ.	Mezerein; the drug on drying loses some of its activity.	Leaves, flowers, fruit, bast.	Inflammation when applied to the skin; internally, gastroenteritis; very marked swelling of lips; hæmatic stool; convulsions.
2	Rhus toxicodendron, sumach; Rhus cotinus, smoke-tree or Venetian sumach.	Anacardiaceæ.	Toxicodendronic acid.	Leaves and other parts.	Externally, inflammation of the skin, even formation of blisters; internally, gastro-enteritis.
3	Ranunculus sceleratus, cursed crowfoot; R. flammula, spearwort crowfoot; R. acris, tall buttercup; R. aquatilis, white water crowfoot; R. bulbosus, bulbous buttercup; R. ficaria.	Ranunculaceæ.	Anemonol, Ranunculol.	All parts of the plants, so long as they are fresh.	Externally, reddening and formation of blisters; internally, gastro-enteritis; convulsions; paralysis.
4	Anemone nemorosa, wood anemone; A. pratensis; A. sylvestris, etc.	Id.	Id.	Id.	Id.
5	Pulsatilla pratensis (Pasque flower of Europe).	Id.	Id.	Id.	Id.
6	Caltha palustris, marsh marigold.	Id.	?	Id.	Similar to pulsatilla.
7	Oenanthe crocata and Oe. fistulosa.	Umbelliferæ.	A resin, oenanthin.	Juice of root.	Stomatitis, colics, diarrhœa, general paralysis.
8	Arum maculatum (or arisæma); A. dracunculus or draconitum, dragon-arum, dragon root, etc.; A. italicum.	Aroideæ or Araceæ.	?	All parts of the fresh plants.	Same as Daphne.
9	Calla palustris, water arum.	Id.	Same substance as in arum.	Id.	Id.
10	Caladium Seguinum or Dieffenbachia.	Id.	Id.	Id.	Id.
11	Tamus communis, yam family.	Dioscoreaceæ.	?	Berries and root.	Id.
12	Plumbago, leadwort.	Plumbaginaceæ.	Plumbagin or ophioxylin.	All parts.	Blisters the skin, and causes inflammation of stomach and intestines.
13	Sedum acre, mossy S. or wall pepper.	Crassulaceæ.	?	All fresh parts.	Id.
14	Asarum europæum, wild ginger; A. canadense.	Aristolochiaceæ.	?	All parts.	Id.
15	Actaea spicata, baneberry.	Ranunculaceæ.	?	Fresh berries, seeds, root and herb.	Id.
16	Robinia pseudoacacia, common locust tree or false-acacia.	Papilionaceæ.	A phytalbumose: Robinin.	Mainly the bark.	Hæmorrhagic enteritis, etc.

17 18	Aspidium Filix mas, shield-fern. Pteris aquilina, common brake.	Filices.	Filixic acid and ethereal filix oil.	Fresh rhizom.	Same as 16; in addition, spasms, paralyses, amaurosis.
19	Tithymalus or Euphorbium, spurge.	Euphorbiaceæ.	Euphorbinic acid anhydrid.	The milky juice.	Inflammation of skin and vomiting and purging.
20	Bryonia alba and dioica.	Cucurbitaceæ.	A glycosid: Bryonidin.	Bulb.	Gastro-enteritis, convulsions.
21	Gratiola officinalis, hedge hyssop.	Scrophulariaceæ.	A glycosid: gratiosolin.	The whole plant.	Id.
22	Rhamnus cathartica, buckthorn.	Rhamnaceæ.	Rhamnocathartin.	Juice of berries.	Gastro-enteritis.
23	Chaerophyllum temulum.	Umbelliferæ.	Chaerophyllin.	The whole plant.	Gastro-enteritis and paralysis.
24	Sium latifolium, water parsnip.	Id.	?	Id.	Id.
25	Hydrocotyle vulgaris, water pennywort.	Id.	Vellarin.	Id.	Gastro-enteritis and hæmatic urine.
26	Paeonia, paeony.	Ranunculaceæ.	?	Flowers and seeds.	Gastro-enteritis.
27	Polygonum Persicaria, lady's thumb; P. hydropiperoides, water pepper.	Polygonaceæ.	?	The whole plant.	Id.
28	Capsicum annuum, common cayenne or red pepper.	Solanaceæ.	Capsaicin.	Fruit.	Upon the skin, reddening and pustules; internally, gastro-enteritis.
29	Piper nigrum, black pepper.	Piperaceæ.	Piperin and pepper oil.	Id.	Id.
30	Brassica nigra, black mustard.	Cruciferæ.	Glycosid: Sinigrin, allyl mustard oil.	Seeds.	Id.
31	Sinapis alba, white mustard.	Id.	Glycosid: Sinalbin, paraoxybenzyl mustard oil.	Id.	Id.
32	Juniperus sabina.	Cupressineæ.	Ethereal oil (Sadebaum)	Branches, berries.	Gastro-enteritis, abortion.
33	Ruta graveolens, common rue.	Rutaceæ.	Ethereal oil of rue.	Herb.	Id.
34	Taxus baccata, European yew.	Coniferæ.	Taxus resin and taxin.	Pines and berries.	Gastro-enteritis, abortion, narcosis, convulsions.
35	Ledum palustre, Labrador tea.	Ericaceæ.	Ethereal oil, Ledum camphor, Ericolin.	Herb.	Id.
36	Mentha Pulegium.	Labiatæ.	Ethereal Poley-oil.	Id.	Excitation of the alimentary tract; abortion; action upon liver, heart, kidneys, similar to that of phosphorus.
37	Lupinus, Lupine; almost all species.	Papilionaceæ.	Ictrogen or lupinotoxin.	The whole diseased plant.	Same as 36, but in addition highest degree of icterus (with sheep, cattle, horses).
38	Chelidonium majus, celandine.	Papaveraceæ.	Sanguinarin, chelidonin, chelerythrin.	The milky juice.	Externally, excitation of skin; internally, of alimentary tract.
39	Croton Tiglium.	Euphorbiaceæ.	Crotonolic acid (see page).	Seeds and the oil therein.	Causes inflammation internally and externally.
40	Citrullus colocynthis.	Cucurbitaceæ.	Colocynthin.	The fruit.	Hæmatic diarrhœa.

	ERGOTISM.	MAYDISM.	LATHYRISM.
PREP.	Secale cornutum, the mycelium (sclerotium) of the fungus Claviceps purpurea, called ergot. It is found in most differing gramineæ and half-gramineæ, but mainly on Secale cereale (rye), from whence it finds its way into flour and bread. Ergot contains many indifferent substances, such as coloring matter and fat, but besides these, two strong poisons: sphazelinic acid and cornutin. The first named causes ergotism gangrænosus, the second ergotism convulsivus. The joint appearance of the two kinds of symptoms is sometimes called raphania. (Kriebelkrankheit.)	Unknown toxic substances, partly of basic nature (pellagrocein, etc.). They develop in the ripe Indian-corn (Zea Mays, Gram.) under the influence of such micro-organisms as bacillus maydis cuboni, and bacillus mesentericus fuscus. This development is produced if the flour had not been sufficiently dried or had been kept in a moist place. The disease subsequently produced in man is called pellagra.	Unknown toxic substances which develop, without the agency of bacteria, in the normal ripe seeds of the most differing varieties of Lathyrus — mainly in Lathyrus cicera, Lathyrus clymenum, Lathyrus sativus. The poison seems to be volatile, or to decompose when persistently heated.
STAT.	While formerly the whole of Europe was decimated by ergotism, this occurs now only in a measure in Russia and Spain. In some epidemics the mortality is large, in others small. The last German epidemic raged ten years ago in the neighborhood of Marburg.	In northern Italy, France, Portugal, Spain, Roumania, Wallachia, but mainly and frequently in Bessarabia. In the last-named country it is even on the increase.	Has occurred since Hippocrates' time. Now frequent mainly in Spain, France, Italy and Russia; also with the Kabyles in Africa, and in India.
ÆTIO.	Ergot possesses a decided action only during a few months after the harvest. Consequently wholesale poisoning will, as a rule, occur shortly after the crops are in, caused then by fresh bread. In other months we merely meet with the medicinal poisoning, or with infanticide caused by abortion.	The exclusive partaking for years of bread and other food prepared from spoiled Indian-corn.	Eating of bread made from flour of some species of Lathyrus. This is hardly avoidable in years of scarcity.
DOS. LET.	A few grams of new ergot will produce the severest symptoms; the fatal amount is unknown.	Fatal cases proper do not occur; the patients, however, frequently end by suicide.	Fatal cases occur sporadically, but they are not the rule.
ACT.	Sphazelinic acid causes local gangræne at the place of application as well as, after resorption, at peripheral points of the body. This gangræne is brought about by degeneration of the walls of the vessels and surrounding parts, with additional spasms of the vessels. Cornutin produces excitation of all centres of the brain and spinal cord; later on some or all of them become paralyzed, and then follow degenerations within the central-nervous system, the peripheric nerves, and certain muscles.	Three different systems of organs become most markedly chronically diseased: the skin, the digestive tract, and the central-nervous system. After each attack, cessation may follow; new quantities of the poison entering, the action is renewed and the case becomes gradually incurable.	Without characteristic prodromes in the intestinal tract or other organs, man and horse suffer a peculiar disease of the spinal cord with paralytic phenomena.

SYMPT.	Gangrænous and convulsive ergotism may occur separately or both together, since in the blighted grain now one and now the other substance predominates. It seems that both forms begin with a sensation of crawling in the tips of fingers and toes. (Kriebelkrankheit.) In case of gangrænous ergotism the mentioned parts of the body will become numb, cold and of bluish color, and finally they partially slough off. In the intestine the action is manifested by typhous changes of the plaques. For convulsive ergotism we find the crawling followed by retardation of the pulse, increased blood-pressure, abortion, and epileptiform spasms which persist for hours. Upon this may follow tabetic disturbances of walking, and atrophy of the muscles of forearm and lower part of leg.	Debility; reddening and swelling of the skin; antipathy to food; profuse diarrhœa; idiocy; delirium; raving mania; tabetic disturbances, such as paresthæsia, ataxic walk, etc.; spasms, contractions, debility of muscles, paraplegia. Finally we have general marasmus. Suicide not infrequently ends the life of these unfortunate ones who have become a burden to their surrounding friends and themselves.	Domestic animals, horse, pig, and man, will alike experience debility of the rear or lower extremities, and finally perfect motor-paralysis will take place. With animals, the normal function of the larynx will sometimes be impaired, in consequence of paralysis of the nervus recurrens. Man may experience an increased reflex-action of the tendons of the paretic lower extremities.
THER.	Only prophylactic. The blighted grain should be gathered and destroyed and the farmers or peasants should be instructed in regard to its poisonous nature. The disease itself is treated symptomatically, but requires admission to a hospital.	Give bread made of wheat or rye; drying of Indian-corn in drying-furnaces under control of state or other authorities; timely transferring of patients to hospitals.	Withdrawal of nourishment prepared from Lathyrus, and substitution of healthy food therefor. Electric treatment of the paralyzed parts; passive movements, massage.
P.-MTM.	Typhoid changes in intestines, mutiple hæmorrhages, these latter also in mesentery and in other organs. Liver, etc., contains amyloid flakes. The necrotic parts of the body present a picture of dry gangræne, in spite of all vessels being filled, the contents being coagulated and markedly degenerated. The spinal cord of man shows tabetic stringy degeneration. Degeneration of muscles.	Skin changes by inflammation but not in a characteristic manner. The intestines show multiple small ulcers and extensive catarrh. Combined diseased condition of system of posterior column and motor-area of the lateral tract of the spinal cord.	Atrophied ganglion-cells in the anterior horns of spinal marrow, of the ganglion-cells of the nucleus of the vagus. Atrophy of nervus recurrens and of muscles of the larynx.
DETECT.	The test for ergot is based upon very characteristic morphologic elements, and upon the presence of some substances, which, though inactive, will give characteristic chemical reactions. We have in the first place, trimethylamin, to be made noticeable through its odor by means of warming with potassium hydrate-solution; secondly, the red coloring substance erythrosclerotin. This has to be extracted from bread and flour by means of alcohol containing sulfuric acid, and by subsequent agitation with ether. In the spectroscope it shows one absorption band in the green and one in the blue.	The above-named bacteria can be shown to be present in the flour. In addition, Dragendorff's method admits of isolation of peculiar substances of basic nature, said to be of toxic action upon animals (?). Naturally these substances are only to be found at a particular stage of decomposition of the corn.	The test can merely be of a morphological nature, since hardly anything chemical is known about the poison. Unfortunately a physiological test is impossible; at least we did not succeed with seeds which had acted in a notoriously poisonous manner.

toxic action by digestion. Another part will be resorbed, causing coagulations, hæmorrhages and ulcers in the vessels of the intestinal mucous coat.

The number of people who have thus fallen sick or died is not insignificant. The pretty colored castor-beans ripening in the garden are particularly attractive for children, who will put them into their mouths.

A **second group** of blood poisons either dissolves the red blood-corpuscles or extracts the coloring matter thereof. A portion of this dissolved coloring matter, non-poisonous in itself, will be retained within the liver, and partly transformed into bile coloring-matter, provided it is small in quantity. But if the amount of dissolved corpuscles is large, a part of the hæmoglobin will be transformed into met-hæmoglobin, the alkalescence of the blood will be considerably reduced, and within the kidneys a mixture of hæmoglobin and met-hæmoglobin will be secreted with the urine. Under these conditions cylinders of hæmoglobin and met-hæmoglobin are easily formed within the kidneys, which will clog up the convoluted tubes, HENLE'S loops being mainly affected. It may also happen that a part of the hæmoglobin is temporarily deposited in the spleen, the lymphatic glands and the spinal-cord. The stroma of the blood-corpuscles, which had been partly extracted, partly dissolved, not only causes embolism of the capillaries, but it increases the disposition of the blood to form coagula, and clogs the vessels of the intestinal follicles and of the parenchyma of the kidneys by thrombosis. It is of no importance whether or not these poisons are, in addition, of specific action upon the nerves. The following table, which contains only vegetable poisons, mainly a number of the so-called *saponin*-substances (Nos. 3 to 11), might easily be enlarged; the common horse-chestnut, for example, contains such a substance. *Phallin* (No. 1.), is a toxalbumin, which is partly resorbed in an active form by the intestinal canal, after partaking of sufficient quantities of fresh *toad-stools*; many fatal cases are on record. *Solanin* (No. 9.), is located directly underneath the skin of the potato and will be removed with this. On boiling potatoes it is partly dissolved, which expiains the fact that occasionally small animals, mainly poultry, fall sick from potato-water.

TABLE OF THE MOST IMPORTANT POISONS WHICH DISSOLVE BLOOD-CORPUSCLES.

No.	NAME OF POISON.	PLANT FURNISHING IT.	FAMILY.	ACTION.
1	Phallin.	Amanita phalloides Fr., and its numerous varieties, *e. g.*, A. mappa, Am. viridis, Am. citrina.	Fungi.	Some length of time after partaking vomiting and purging, prostatation, cold perspiration, delirium, cyanosis. Post-mortem appearance like phosphorus.
2	Helvellaic acid.	Helvella esculenta Pers., while Morchella esculenta seems to be non-poisonous. A distinct species by the name of Helvella suspecta does not exist.	Id.	After some time, nausea, vomiting, icterus, somnolence. The convoluted urinary tubes filled with hæmoglobin - cylinders; urine contains hæmoglobin, met - hæmoglobin, bile pigment.
3	Quillaic acid and sapotoxin.	Quillaja Saponaria. The bark contains both poisons, which are very similar to each other and of glycosidic nature.	Rosaceæ.	Hæmoglobinuria, inflammation of intestines, dislocation of kidneys, only after direct introduction into the blood of animals. Internally, excitation of the alimentary canal.
4	Senegin and polygalaic acid.	Polygala senega, and some American species closely related to it. The bark of the root is the poisonous part.	Rubiaceæ.	Same as Quillaja. With man, after internal application; expectoration, vomiting, diarrhœa.
5	Paridin.	Paris quadrifolia.	Liliaceæ.	Vomiting and purging with man and animals.
6	Saporubrin.	Saponaria officinalis, common soap-wort.	Silenaceæ.	Id.
7	Cyclamin.	Cyclamen Europæum, sow-bread.	Primulaceæ	Id.
8	Agrostemma - sapotoxin.	Agrostemma githago,	Silenaceæ.	Domestic animals have often been killed by it. Resorbed by the intestines.
9	Solanin.	Solanum nigrum, black or common night shade. Sol. Lycopersicum, tomato; Sol. tuberosum, potato.	Solanaceæ.	Excitation of alimentary tract in man and animals; giddiness, staggering, weakness of back.
10	Dulcamarin.	Solanum dulcamara, bittersweet.	Id.	Same as with solanin.
11	Melanthin.	Nigella sativa, nutmeg flower.	Ranunculaceæ.	Excitation of alimentary canal.

Some of the *saponin*-substances will not be resorbed at all by the intact mucous coat of the stomach and intestines, *e. g.*, *quillaic-acid* and *sapotoxin*, while this does not hold true of others. In case of non-resorption, the action upon the blood will of course not take place, however, when internally applied, there will be excitation of the mucous coat of the alimentary canal. In case the drugs are marketed in a powdered state, as, *e. g.*, *quillaja-bark*, the dust must be kept away from the eyes, as it inflames them violently.

A **third group** of blood poisons fixes the oxygen of oxy-hæmoglobin in an inseparable manner to hæmoglobin, with or without previous solution of blood-corpuscles. Thus, the tissues not being supplied with oxygen, dyspnœa and even suffocation will follow. We call this modification of the coloring matter of the blood, remarkable for its sepia-brown color, *methœmoglobin*. In the spectroscope it is characterized by an absorption-baned in the red part. It is of importance for the physician to know that within the corpse the *methœmoglobin* will disappear after some days, *hœmoglobin* being reformed, while the oxygen had been used up for bacterial life. This occurs mainly in summer. Consequently, in suspicious cases, the post-mortem should be held at once, else we cannot expect to find any methæmoglobin at all. The decomposition of the coloring matter of the blood by corrosive poisons, always give rise to the formation of *methœmoglobin* in addition to *hœmatin*. Between those poisons which merely dissolve the blood-cor. puscles and those forming *methœmoglobin*, stand the bile-acids and *arsin*, the action of which gives rise to dissolved *hœmoglobin* at first, and later on to abundant quantities of *methœmoglobin*. A useful antidote for all the poisons forming *methœmoglobin* is sodium carbonate, or the so-called sodium sesqui-carbonate. This transforms the brown *methœmoglobin* into a red modification called alkaline *methœmoglobin* which latter allows the organism to transform it with comparative ease into *oxy-hœmoglobin*. The danger arising from a possible solution of red blood-corpuscles is the same with the poisons belonging to this group, as with those of the preceding one. In regard to therapy, in case of intoxications caused by the group now under discussion, it must be remembered

that the formation of *methœmoglobin* will set in more readily, and develop to a greater excess in proportion to the diminished alkalescence of the blood and the tissue-juices. Always attempt, therefore, to increase the alkalescence, never prescribing acid beverages.

TABLE OF THE MOST IMPORTANT POISONS CAUSING PRIMARY FORMATION OF MET-HÆMOGLOBIN.

NO.	NAME.	ACTION, ETC.
1	Chlorates.	Potassium chlorate, in particular, has killed many people. A few grammes are sufficient, as in cases of fever, dyspnœa and of partaking of acid lemonade (*e.g.*, of HCl or H_3PO_4) Icterus, met-hæmoglobinuria, somnolency, uræmia, anæmia. The post-mortem will show the blood to be of chocolate-brown color; the kidneys filled with met-hæmoglobin-cylinders.
2	Pyrogallol or pyrogallic acid.	The mere application of salves of from 5 to 10 p.c. rubbed over the whole body, suffices to cause headache, chills, vomiting, purging, cyanosis, somnolency and met-hæmoglobinuria. Post-mortem same as above.
3	Chrysarobin.	After extreme applications, as in skin affections, the same phenomena as with pyrogallol; in addition, a brownish-red coloration of the skin; conjunctivitis and swelling of the lymphatic glands. The urine contains met-hæmoglobin and chrysophanic acid.
4	Hydrazin, phenylhydrazin and pyrodin (hydracetin).	Chills, vomiting and purging, formation of met-hæmoglobin, but after small doses the urine will not have a brown color.
5	Salts of hyroxylamin.	Same as with hydrazin.
6	Nitrobenzene or essence of mirbane.	More than 150 people have so far been recorded poisoned by it. The breath has an odor suggestive of bitter almonds; profound cyanosis; vomiting; dyspnœa; maxillary spasms; chills; delirium; mydriasis; convulsions; blood of brown color.
7	Nitroglycerol.	Inflammation of alimentary canal; dizziness, dyspnœa, cyanosis, delirium. The poison is readily absorbed by the skin.

NO.	NAME.	ACTION, ETC.
8	Amylnitrite, ethylnitrite, butylnitrite, isobutylnitrite.	Entering most rapidly into the blood in case of inhalations, or if taken internally. Very pronounced dilatation of blood-vessels, and consequent lowering of the blood-pressure and unconsciousness. Blood brownish and of an odor suggestive of apples.
9	Sodium nitrite.	Acts as does amylnitrite, but slower and of more persistent duration. Internally applied, inflammation of the gastro-intestinal tract.
10	Dinitronaphthol, Martius-yellow; potassium di-nitrocresol, saffron-surrogate.	Both dyes act upon the intestinal tract similar to sodium nitrite, at the same time giving to it a yellow color. The nervous system will at first be excited, then paralyzed. But little met-hæmoglobin formed.
11	Picric acid and its salts.	Excitation of the gastro-intestinal canal, which is colored yellow. Intense picrin—icterus; urine colored red by Picraminic acid. Picrin-urticaria; delirium, spasms. At the post-mortem all organs are found to be yellow. But little met-hæmoglobin formed.
12	Anilin, toluidin, antifebrin, exalgin.	When inhaled or taken internally, anilin-oil will produce deepest cyanosis, caused not only by met-hæmoglobin-formation, but also by the production of anilin-black within the blood. Lowered temperature, chills; dilated pupils: coma; convulsions. Urine of brownish-black color. Antidote is Glauber's salt (?). Washing of organism.
13	Carbondisulfid.	After inhalation or when taken internally, headache; disturbances of vision; dizziness; vomiting; coma. With animals, convulsions in addition. But little hæmoglobin in the blood. In chronic cases, psychoses with excitation and following depression.

A **fourth group** of blood poisons is represented by *prussic acid, carbon monoxid* and *hydrogen sulfid,* in so far as there exist combinations of these with the coloring matter of the blood. But only with carbon monoxid is the formation of such a compound called *carbon monoxid-hæmoglobin,* the main cause of death. With hydrogen sulfid, the formation of *sulf-met-hæmoglobin* begins only after the action of the poison upon the nerves has proved fatal. With hydrocyanic acid, there

are even three compounds existing, one with hæmoglobin, viz., *hydro-cyan-hæmoglobin*; one with met-hæmoglobin, viz., *hydro-cyan-met-hæmoglobin*, and one with hæmatin, viz., *cyan-hæmatin*, which latter exists only in alkaline solution. With this acid, the poisonous qualities are not discerned in its action upon the coloring matter of the blood, but they consist in inhibiting the interchange of oxygen with the protoplasm of the body-tissues and with the red blood-cells. In contrast to the brown color of met-hæmoglobin, its compound with hydrogen cyanid is of a beautiful reddish hue, and this fact is utilized as proof of the presence of both met-hæmoglobin and prussic acid.

For Table of Intoxications caused by Prussic Acid, Carbon Monoxid and Hydrogen Sulfid, see pages 118-119.

TABLE OF INTOXICATIONS CAUSED BY PRUSSIC ACID, CARBON MONOXID AND HYDROGEN SULFID.

	PRUSSIC ACID (HYDROCYANIC ACID).	CARBON MONOXID.	HYDROGEN SULFID.
SOURCE.	Hydrocyanic acid, prussic acid; that of the pharmacopœas is a 2–4 p.c. aqueous solution; potassium cyanid, argentic cyanid, auric cyanid. Potassium ferrocyanid taken internally develops HCN only in the presence of acids in the stomach. Amygdalin of bitter-almonds, and of cherry, peach, almond, prune, and apricot-kernels, is decomposed within the stomach, by emulsin contained in the kernels, into HCN. Aqua-amygdalarum-amarum contains 0.10 p.c. HCN. Kirschwasser, persico and maraschino contain benzaldehyd-hydrocyanic acid.	The pure CO is rarely to be considered. The gases from furnaces or stoves contain 79.7 p.c. nitrogen, 10–13 p.c. oxygen, 0.6 p.c. carbon di-oxid, 0.3–0.5 p.c. carbon monoxid, provided these gases have been formed by imperfect combustion of wood, coal, etc., in closed spaces, *e.g.*, stoves with closed damper. Illuminating gas contains, when prepared from coal, 6–10 p.c. CO; prepared from wood, up to 62 p.c. CO; and it contains marsh gas and ethylene. Water-gas is a mixture of CO and H; the quantity of CO may be more than 30 p.c. The gases of coal mines contain from 4 to 10 p.c. CO, 0.6–1.0 p.c. H_2S, and 53 p.c. CO_2. The gases of smokeless powder are still more dangerous. The gases from the explosion of gun-cotton contain 30 p.c. CO, 20 p.c. CO_2, 10 p.c. CH_4, 8 p.c. N, 23 p.c. steam, 9 p.c. NO (nitric oxid), which in contact with air is oxidized to nitrogen tri- and per-oxids.	Pure hydrogen sulfid to be considered only in chemical laboratories. The crude illuminating-(coal) gas contains a trace of H_2S. The gas from water-closets and sewers contains from 2.5 to 8 p.c. (besides NH_3 CO_2, etc.) The spring waters containing hydrogen sulfid have so far caused no intoxications; just as little the internal or external use of the alkaline sulfids and calcium sulfid, though new experiments with both upon animals have elicited an exceedingly dangerous action.
STAT.	Hundreds of intoxications recorded, with 95 p.c. mortality.	One of the most frequent intoxications. Mortality differs much according to the kind of mixture inhaled and the length of time of exposure.	Not exactly rare; high mortality.
ÆTIO.	Murder, suicids, carelessness, and medicinal poisoning.	Leaky gas-pipes, open coal-fires, premature closing of dampers of stoves, explosions in technical pursuits.	The most frequent cause is the cleaning of cesspools and the like.
DOS.LET.	0.06 gms. are fatal, but generally much more is taken.	0.8 gms., according to Dreser, 11.5 milligms. per kilogm. for rabbit.	It might stand between CO and HCN. Even 0.02 p.c. in the air may have a toxic action.
ACT.	The transmission of oxygen from the blood-corpuscles to the tissues ceases, and, in consequence, an internal asphyxiation occurs in the presence of an excess of oxygen. The spinal cord is excited, and paralysis of the whole nervous system follows.	The capacity of the red blood-corpuscles to take up oxygen decreases in proportion to their saturation with CO, and thus a true inner asphyxiation takes place. Along with this, there is direct excitation of the spasm-centers of the central-nervous system, producing paralysis. Finally, in case of slow intoxication, we have degeneration of the kidneys, liver, heart, etc.	Even before the formation of sulfmet-hæmoglobin, excitation and then paralysis of the nervous system sets in; but in case of much hydrogen sulfid, death ensues with lightning rapidity, as with HCN.
SYMPT.	Dizziness, headache, oppression, disturbances of vision, dyspnœa, unconsciousness, and stoppage of respiration while the heart still beats well.	Buzzing in the ears, dizziness, nausea, vomiting, stertorous breathing, redness of the face, anæsthesia, fainting spells; involuntary passage of stools, spasms, entire paralysis, coma. With animals, the pulse in the beginning is slow, while the blood-pressure increases. With man, the action of the heart is frequently violent even during the sopor.	Redness and pains of the eyes, nasal-catarrh and catarrh of the throat, dyspnœa, cough, rapid beating of the heart, dizziness, headache, numbness, cold perspiration.
DYPT.	On artificial respiration, natural respiration may return; in absence of artificial respiration, violent convulsions.	When removed from the poisonous atmosphere, slow return to consciousness, but headache, nausea, and weakness persist for a long time. In case of continued inhalation of the poison, there is great dilatation of the peripheral vessels, causing large red spots on the skin.	Persistent inhalations of small amounts of hydrogen sulfid produce convulsions, mydriasis, paralysis of brain and spinal cord, and œdema of lungs.

EXIT.	Speedy death, with spasms; or recovery, after which the headaches will remain for a long time.	Death from paralysis of the respiratory apparatus. In case of a not fatal termination, we may have apoplexy, followed by softening of the brain. Blisters, decubitus, and paralyses may obtain, also chorea, idiocy, and ascending paralysis.	Death from complete paralysis of the central-nervous system. In case fresh air had access in time, life is preserved, but bronchopneumonia and catarrh persist.
P.-MTM.	Remarkably bright red post-mortem spots, in case of potassium cyanid also highly reddened mucous lining of stomach. The blood of the large vessels only bright red after application of very large quantities of the poison, blood containing oxyhæmoglobin and being poor in coagulated matter. Specific odor of the cavities of the body and of the brain. The urine may contain blood, reducing substances, lactic acid, and hydrocyanic acid.	The post-mortem spots; the blood of all organs cherry-red. The blood shows the spectrum of CO. Degeneration of the heart, liver and kidneys; ruptures of the vessels of the brain; blisters of the skin and decubitus; pneumonia, serous pleuritis; diphtheritic membranes of the throat may be superadded. The urine contains sugar or glycuronic acid, also lactic acid. There is no specific odor of the cavities of the body or of the blood. In chronic cases there are signs of softening of the brain and secondary degeneration.	In case of immediate dissection after rapid death, no changes at any place, not even in the blood. In case of slow death, œdema of lunge or pneumonia, and the corpse smells like rotten eggs. In case of late post-mortem, brownish-green sulf-met-hæmoglobin is formed, and the post-mortem spots have the same color.
THER.	Wash the stomach with a 0.5 p.c. potassium permanganate solution, or with hydrogen peroxid; the hydrogen peroxid may also be given hypodermically in small doses and with great caution. Artificial respiration; alkalies; bleeding, and subsequent transfusion.	Removal from the poisonous atmosphere, artificial respiration, oxygen, hydrogen peroxid very cautiously administered internally or hypodermically; bleeding, transfusion. In case of abnormally low temperature, making warm in bed; faradization of the phrenici; black coffee. As a prophylactic, remove the damper from the stove and prohibit the use of odorless gas.	Protective masks should be worn when going down into pits, etc.; frequent addition of large quantities of ferrous sulfate-solution into cesspool, etc., as prophylactic measures. Fresh air; artificial respiration. Inhalations of chlorin gas of doubtful value.
DETECT.	The acid distillate of the intestines is treated with ferrous sulfate, saturated with KOH, boiled, filtered, the filtrate acidulated, and ferric chlorid added; Prussian-blue will be formed as a ppt. Or the distillate, to which ammonium sulfid had been added, is evaporated over a water bath almost to dryness, acidulated with HCl, and ferric chlorid added, a blood-red coloration is formed. Or we perform the reaction mentioned on page ; this, however, will apply to other substances, *e.g.*, nitrobenzene. Or the distillate is treated with potassium hydrate-solution and picric acid, when potassium isopurpurate is formed, of a purple-red color. The distillate will decolorize the blue produced by iodin upon starch.	The blood, if treated with reducing substances, retains its peculiar double-banded spectrum. When mixed with caustic soda-solution, the blood will not turn brownish-black, but a beautiful red. When 4-5 times its volume of basic plumbic acetate ($Pb(C_2H_3O_2)_2 + 2PbO$) is added, the blood will not turn brown, but red; with potassium ferrocyanid and acetic acid, not a brown, but a red ppt.; with tannin, a crimson ppt is formed. The blood, when kept in a sealed glass-tube, will retain its peculiar double-banded spectrum, which is similar to that of oxyhæmoglobin, but the two bands are located in closer proximity to each other, and both are slightly displaced towards the right side. If such a tube is opened and a reducing agent added, the spectrum of reduced hæmoglobin will *not* obtain.	A good nose will detect even minimal traces. Moist plumbic acetate-paper or plumbic acetate-solution is blackened by hydrogen sulfid; same with argentic nitrate-solution or paper. (Compare page 40.) Sulf-met-hæmoglobin has a spectrum similar to met-hæmoglobin, but marked by a strong absorption-band in the red; intra vitam it is not formed, but only in the corpse.

C. Substances which may Prove Fatal without Causing any gross Anatomical Changes.

It would be a mistake to suppose that the following poisons never cause anatomical changes at all; we only assert that the pertaining fatal results, so far as observed in man, were generally brought about without presenting such marked pathological disturbances as might themselves have been regarded as the direct cause of death. It is obvious that we may at will arrange the experiments on animals in such a manner that any one of those substances will cause anatomical changes.

I.—POISONS OF THE CEREBRO-SPINAL SYSTEM.

The number of poisons belonging to this class is extraordinarily large, restricting consideration thereof to the more important ones only. At the outset we are tempted to classify them, according to the respective parts of the cerebro-spinal system immediately affected, into poisons of the cerebral cortex, the basal parts of the cerebrum, the cerebellum, the medulla oblongata, the spinal cord and the peripheral nerves. But such an arrangement would be extremely artificial, because most poisons, according to the dosage, attack different parts of the nervous system. Of the following two tables, the first (pages 122-125) records the vegetable poisons, while the second (pages 126-127) briefly enumerates the artificially prepared toxic substances. Following these are presented in three more tables, special data, referring to some vegetable poison-groups which are of particular practical interest. We repeat, that, concerning all poisons applied *per os*, speedy evacuation of the stomach being most obviously necessary, we have, therefore, not in any of the tables, made particular mention of this measure under the heading of "therapy."

The Indian-arrow-poison, *curare*, is not mentioned, since it has never actually caused poisoning in Germany, and its most important action has been already sufficiently stated. (Page 58). We learn from table, page 122, and seq., that *cytisin* and *coniin* will, under certain conditions, produce paralysis, suggestive of *curare*, of the peripheral terminations of the motor-nerves. The *coniin hydrobromid* has even been recommended as a substitute for *curare* in the treatment of *tetanus*. Unfortunately, the preparations of this salt offered by the trade are so lacking in uniformity, that we cannot endorse this recommendation. The *curare* of commerce

continues to depreciate in quality and it has been suggested to use as a substitute the active base contained therein, viz., *curarin*. And yet the marketed preparation of the latter, BŒHM-MERCK'S *curarinum purissimum* leaves room for improvement. *Curare* consists essentially of the aqueous extract of certain kinds of *strychnos*. We would therefore expect to see *strychnin* produce in frogs an action suggestive of *curare*, at least when an overdose is administered.

The active substances of the *boraginaceæ*, as, *e. g.*, anchusa, heliotropium, echium, cynoglossum, symphytum, lithospermum and myosotis are alkaloids, which are said to produce at first excitation of the central-nervous system, followed by paralysis suggestive of *curare*. But they have never yet caused intoxication in man. We have already mentioned (on page 107) the *fugin* of the *tetrodon*-fishes, acting similar to *curare*. Also the *mytilite*, living in stagnating water, develops a poison similar in action to *curare*, it is called *mytilotoxin*. The *aconitum septentrionale* growing in high latitudes, contains several alkaloids, of which one, called *septentrionalin*, possesses in addition to other actions, one decidedly suggestive of *curare*.

The leaves of *celastrus edulis* or *catha edulis* (*celastraceæ*), indigenous to southern countries, contain a base possessed of an action similar to *cocain*, and which constitutes the active principle of the Arabic *Kath*.

The American *condurando bark*, which is furnished by gonolobus condurango (*asclepiadaceæ*) contains a series of glycosids which are embraced by the one name, condurangin. They produce violent spasmodic attacks of purely cerebral origin, and are to be enumerated with the substances on page 130 and 131. It is remarkable that these spasms seem to have never been observed as occurring in man.

All poisons causing violent spasms may, by means of these, secondarily produce a severe disturbance of metabolism, (exchange of material). Under normal conditions, and under the influence of muscle-activity, carbohydrates are burned up to form carbon dioxid and water; but in case of violent spasms, particularly if simultaneously there is insufficient supply of oxygen, this combustion ceases at the stage of lactic acid-formation; the latter acidifies the blood, thus causing further disturbances.

The temperature of the body is by no means always increased in case of spasms, as it was formerly believed; frequently it is even lowered.

No.	PLANT.	FAMILY.	POISON CONTAINED IN IT.
1	Papaver somniferum. The statement that Escholzia california, belonging to the same family, contains morphin, is not true.	Papilionac.	The dried juice of the unripe capsules furnishes Opium, containing: 10-15 p.c. morphin, 1.5-4.0 p.c. narcotin, 1.0 p.c. thebain, 1.0 p.c. papaverin, 0.3 p.c. codein, narcein, laudanin, protopin, xanthalin, meconic acid, etc.
2	Cannabis sativa, var. indica, Indian hemp.	Cannabac.	Several alkaloids, a glycosid cannabin, a resin cannabinon, an ethereal oil, etc. In my own laboratory only one active substance, cannabindon, was found.
3	Lactuca virosa.	Compos.	Traces of a nameless base, and traces of hyoscyamin.
4	Lolium temulentum.	Gram.	Temulin.
5	Erythroxylon coca, and other species.	Erythroxyl.	Cocain, cynnamyl-cocain, isatropyl-cocain, benzoyl-pseudo-tropein or tropa-cocain, etc. The leavee and cocain hydrochlorid are in use.
6	Coffea arabica.	Rubiac.	Coffein, coffeol or coffeon. The latter is formed only on roasting.
7	Sterculia acuminata.	Sterculiac.	Coffein, colanin.
8	Paullinia sorbilis.	Sapindac.	Coffein.
9	Thea chinensis.	Camelliac.	Coffein, theophyllin, tea-oil.
10	Ilex paraguayensis.	Ilicin.	Coffein.
11	Ilex cassine.	Ilicin.	Coffein.
12	Atropa belladonna, deadly-nightshade.	Solanac.	Atropin, hyoscyamin, belladonnin.
13	Datura stramonium, thorn-apple.	Solanac.	Atropin, hyoscyamin, daturin.
14	Hyoscyamus niger, black-henbane.	Solanac.	Scopolamin, hyoscyamin, atropamin.
15	Scopolia atropoides.	Solanac.	Scopolamin, hyoscyamin.
16	Nicotiana tabacum, N. rustica, N. macrophylla.	Solanac.	Nicotin.
17	Pilocarpus pennatifolius.	Rutac.	Pilocarpin, pilocarpidin, etc.
18	Lobelia nicotianæfolia, etc.	Lobeliac.	Lobelin.
19	Lupinus albus, L. luteus, L. angustifolius.	Papilionac.	Lupinin, lupinidin, lupanin, etc.
20	Conium maculatum, spotted-hemlock.	Umbellif.	Coniin, methyl-coniin, conhydrin, pseudo-conhydrin, etc.
21	Cicuta virosa, water-hemlock.	Umbellif.	Cicutoxin.
22	Menispermum cocculus L. or anamirta cocculus W. and A.	Menisperm.	Picrotoxin.

REMARKS.	DETECTION.
The green plant contains only morphin and narcotin, preformed. Meconic acid, as well as the strongly odorous substance in opium, are inactive.	Meconic acid turns blood-red with ferric chlorid. According to Dragendorff, benzene extracts meconin from an acid solution, while amylic alcohol dissolves meconic acid. From an alkaline solution benzene will extract codein, narcotin and thebain; and chloroform, as well as amylic alcohol, narcein and morphin. Morphin turns dark-blue with ferric chlorid in strictly neutral solution; with Froehde's reagent violet, green, blueish-green and yellow.
The tips of the branches of the female plant, as well as the resinous masses exudated by these, represent hasheesh.	We cannot speak of detection by chemical means, since this plant has scarcely been examined. But the leaves may easily be determined pharmacognostically. The cannabindon which I found acts in a reducing manner and causes hallucinations.
Acts as a narcotic and mydriatic.	The base passes from alkaline solution into amylic alcohol on agitation. The plant is easily recognized pharmacognostically; generally, however, the condensed juice (lactucarium) is used.
Acts as a narcotic and mydriatic.	Ether, as well as chloroform, will abstract it on agitation, from alkaline solution.
The leaves are taken as a food. The leaves of all species contain cocain, but only the mentioned one contains an abundant amount of it.	Petroleum-ether on agitation with an alkaline solution will take it up. Amylalcoholic KOH turns cocain, evaporated with HNO_3 violet, on warming. Iodin-water renders its solution in sulfuric acid at first reddish-violet, afterwards kermes-red.
The roasted beans, which furnish the coffee, contain 0.5 to 1 p.c. coffein.	Coffein allows abstraction from alkaline solutions by means of chlorofoform. Boil the residue of evaporation, consisting of characteristic crystal-needles, with concentrated nitric acid, and after cautious evaporation to dryness, add ammonia. We then obtain the identic purple reaction, which, for uric acid, we call the murexid-reaction. Traces of KOH solution change the purple to violet. Within the organism the cocain is not destroyed, but secreted with the urine.
The seeds are the active part.	
Guarana-paste is furnished by the crushed seeds.	
The leaves represent the Chinese-tea; they contain about 3.5 p.c. coffein.	
The leaves, containing 0.8 p.c. coffein, furnish the maté, or Paraguay-tea.	
The Apalache-tea is furnished by the leaves, containing 0.3 p.c. coffein.	
All parts of the plant are poisonous, in particular the berries and the root.	Atropin, hyoscyamin and scopolamin behave chemically very similarly. They may be obtained by agitation of their alkaline solutions with benzene, chloroform or ether. Iodin in KI solution precipitates them even from most strongly diluted liquids. They turn first violet, then cherry-red, when evaporated with nitric acid and afterwards touched with alcoholic-KOH. With an alcoholic sublimate-solution they turn at first yellow, then brick-red. They produce the odor of the sloe-blossoms when warmed with conc. H_2SO_4 and following addition of H_2O.
All parts are poisonous, *e.g.*, the seeds and leaves.	
All parts are poisonous, in particular the seeds.	
All parts are poisonous.	
The treated leaves represent tobacco, and contain 0.5 to 8.0 p.c. nicotin.	From ammoniacal solutions nicotin is abstracted on agitation with petroleum-ether. It may be distilled off from alkaline solutions. It furnishes ruby-red crystals when mixed with a solution of iodin in ether. Chlorin gas will turn it blood-red. The fragments of tobacco-leaves show peculiar glandular hair.
The leaves, folia-jaborandi, are officinal.	Both bases go from alkaline solution into benzene. They turn green with H_2SO_4 + potassium bichromate.
The herb is used medicinally.	Lobelin is obtained from alkaline solutions by agitation with petroleum-ether or ether. The extract of the herb is turned violet with Froehde's reagent.
The herb and the seeds are poisonous.	The bases are partly volatile and similar to coniin, partly solid and extractable by ether.
The herb and the almost ripe seeds are quite poisonous.	Coniin is to be abstracted by agitation of alkaline solutions with ether, chloroform, petroleum-ether, etc. It behaves towards reagents similarly to nicotin, but its aqueous solution becomes turbid on boiling.
The rhizoma in particular is very poisonous.	Cicutoxin passes upon agitation into ether. Besides, the rhizoma contains umbelliferon, of beautiful fluorescence.
The seeds, known as cocculus, or fisher's-berries, are the poisonous part. They were formerly used for the purpose of stifling fish.	The extracts from the organism, purified by means of neutral plumbic acetate and freed from lead, yield picrotoxin to amylic alcohol or chloroform. It will reduce alkaline cupric sulfate solution. Evaporated with conc. HNO_3, then touched with conc. H_2SO_4 and finally upon addition of NaOH solution, it turns brick-red. With H_2SO_4 alone, a saffron-yellow appears.

No.	PLANT.	FAMILY.	POISON CONTAINED IN IT.
23	Artemisia maritima, wormwood.	Compos.	Santonin.
24	Artemisia absinthium.	Compos.	The bitter principle absinthin, and the ethereal absinth-oil.
25	Laurus Camphora.	Laurac.	Camphor.
26	Mentha piperita, peppermint.	Labiat.	Menthol, menthen, etc.
27	Tanacetum vulgare, common tansy.	Compos.	Tanacet camphor, etc.
28	Cinchona family of many species; we mention: C. succirubra, C. ledgeriana, C. officinalis, C. calisaya, and C. lancifolia.	Rubiac.	Quinin, quinidin or conquinin, cinchonin, cinchonidin, etc. Quinatannic acid, quinovin or quinic acid.
29	Physostigma venenosum.	Papilionac.	Physostigmin or eserin.
30	Veratrum sabadilla.	Melanthac, sive Colchicac	Veratrin, Veratridin, cevadillin, sabadillin, etc.
31	Veratrum album (white hellebore or Indian poke), V. viride, V. nigrum.	Melanthac, sive Colchicac	Veratroïdin, jervin, protoveratrin, etc.
32	Colchicum autumnale.	Melanthac, sive Colchicac	Colchicin, colchicein.
33	Aconitum napellus, true monkshood or officinal aconite.	Ranunculac.	Aconitin, isaconitin, etc.
34	Delphinium staphisagria, larkspur.	Ranunculac.	Delphinin, delphisin, etc.
35	Strychnos nux vomica.	Loganiac.	Strychnin, brucin.
36	Aspidium filix mas, shield-fern.	Filic.	Filixic acid.
37	Cytisus laburnum, goldenchair or bean-trefoil-tree; and other species.	Papilionac.	Cytisin.
38	Ulex europæus.	Papilionac.	Ulexin, identical with cytisin.
39	Andromeda polifolia.	Siphonandr.	The bitter principle, andromedotoxin, possessing an action suggestive of aconitin.
40	Cassandra calyculata, leather leaf.	Siphonandr.	
41	Rhododendron ferrugineum and hirsutum, rose-bay.	Rhodorac.	The glycosid arbutin and the bitter principle ericolin.
42	Equisetum palustre and limosum.	Equisetac.	Still unknown alkaloids.
43	Cetraria vulpina, or evernia vulpina.	Lichenes.	The beautifully yellow vulpinic acid.

REMARKS.	DETECTION.
Semen cinae is a popular vermifuge.	From acid solutions upon agitation with benzene and chloroform, santonin is taken up. Within the organism it is transformed into santogenin, and another substance which turns a yellowish-red upon addition of an alkali.
On protracted partaking of spirite of absinth, appearances of paralysis and of excitation of the central-nervous system obtain.	Absinthol, contained in the ethereal oil, appears in the urine as absynthol-glycuronic acid of reducing action.
All parts of the tree contain camphor.	See page 42. for detection. The urine will contain diverse reducing campho-glycuronic acids.
The leaves furnish an ethereal oil.	For detection see page 42. The urine will contain menthol-glycuronic acids, which reduce copper solution.
The leaves supply an ethereal oil.	For detection see page 42. The urine contains possibly glycuronic acids.
The bark contaius the alkaloids. They appear in the urine, peculiarly transformed. The cinchonamin contained in some barks serves as a reagent for nitric acid.	The quinin bases pass from alkaline solutions into chloroform, quinin itself into benzene and petroleum-ether besides. Upon addition of chlorin-water and ammonia to the solution of a quinin salt, green flakes are precipitated, soluble with emerald-green color in an excess of ammonia. Upon addition of an acid the tint turns sky-blue, then violet and fiery-red. Quinin sulfate is of strong fluorescence; its alcoholic solution gives, with iodin-tincture, crystals of herapathite of greenish lustre.
The beans, Calabar beans, are the poisonous part.	From alkaline solution physostigmin passes over into: benzene, ether, amylic alcohol and chloroform. Evaporated together with NH_3, it will dissolve with blue color in alcohol, turning red with acids and exhibiting fluorescence.
The seeds are supposed to act medicinally; salves or vinegar prepared by means of them may cause poisoning even on external application.	Veratrin passes even from acid solution into benzene, chloroform and amylic alcohol; and from alkaline solution into petroleum-ether, benzene, chloroform, amylic alcohol. A beautiful red color is produced on warming with conc. HCl. It will give as well the reaction with HNO_3, as mentioned for atropin. With conc. H_2SO_4 and sugar, it turns yellow, dark-green, blue, violet. With Froehde's reagent it assumes first the yellow color of gamboge, afterwards it turns cherry-red.
The whole plant is poisonous.	It is isolated in the same way as veratrin; it behaves in the same manner with H_2SO_4; HCl on boiling turns it cherry-red.
Bulbs and ripe seeds are poisonous. Within the organism oxydi-colchicin is formed.	From acid solution colchicin as well as oxydi-colchicin are taken up by chloroform and amylic alcohol. Erdmann's reagent turns them blue, and upon addition of caustic alkali-solution a brick-red color obtains; fuming HNO_3 produces violet to indigo-blue.
The root mainly is poisonous.	Petroleum-ether, benzene and chloroform abstract aconitin from alkaline solutions. Color reactions are wanting.
The seeds are the poisonous part.	Delphinin and delphinoidin are soluble in ether as well as in chloroform.
The seeds, bachelor buttons, are the drug.	Strychnin passes from alkaline liquids into chloroform and benzene. The chromate with conc. H_2SO_4 turns blue, violet, and cherry-red. Mandelin's reagent turns it first violet-blue, then vermillion-red.
The rhizoma is the poisonous part.	The acid passes from acid solution into ether.
All parts are poisonous, *e.g.*, the seeds, the bark, etc.	The alkaloid passes from alkaline solution into chloroform.
The herb is poisonous in certain months.	
The entire plants are poisonous. The Esth's use it as a universal medicine.	The poison is soluble in alcohol, amylic alcohol, chloroform, ether, benzene. Diluted mineral acids turn it red on evaporation.
Almost all other species contain andromedotoxin.	Arbutin yields on decomposition quinol, to be abstracted from acid solution by acetic ester. With ferric chlorid it furnishes crystals of greenish lustre.
In cattle it will produce paralysis of the rear extremities and death with spasms.	This intoxication is diagnosed in man by the production of very severe hæmoglobinuria, the explanation of which remains hidden from me. Chemical detection is wanting.
The acid and its salts produce spasms and paralysis.	According to Neuberg, vulpinic acid does not reappear in the urine; pulvinic acid, however, related to it, reappears.

No.	NAME OF SUBSTANCE.	FORMULA.	ACTION.
1	Chloroform or trichlor-methane.	$CHCl_3$.	See page 42 and page 128. In contradistinction to ether, chloroform can not be used as an excitant.
2	Ether or sulfuric ether, stronger ether.	$C_2H_5OC_2H_5$.	It acts on inhalation as chloroform. On hypodermic injection, the formation of gas readily obtains, as well as coagulation in the blood vessels, and degeneration of nerves in direct contact.
3	Aether bromatus, ethyl-bromid.	C_2H_5Br.	Narcotic for narcoses of short duration. After larger doses, speedy cyanosis and collapse. With people addicted to drink, appearances of excitation.
4	Aether chloratus, ethyl-chlorid.	C_2H_5Cl.	Modern local anæsthetic, very similar in its remote actions to ethyl-bromid.
5	Bromoform, tribrom-methane.	$CHBr_3$.	Internally of narcotic action. In large doses it produces a state of inebriation, sleep, collapse, total anæsthesia. Only one case on record.
6	Amylene or pental.	C_5H_{10}.	Acts on inhalation, as does chloroform. State of jovial excitation, then profound narcosis. Bad after-effects observed: dizziness, trembling, paralysis of tongue, Raphania. (Krlebeln.)
7	Amylene hydrate, or tertiary amylic alcohol.	$(CH_3)_2.C_2H_5.C.OH$.	Acts like paraldehyde as a hypnotic. Even 27 gms. have been endured, but caused total insensibility, mydriasis and somnolency lasting for days. The pulse is not retarded.
8	Laughing gas or nitrous oxid.	N_2O.	It paralyses, on inhalation, at first the sensation of pain, then consciousness, spinal marrow, medulla obl., and finally the heart. Since it will not be decomposed by the organism, it causes cyanosis if oxygen had not been admixed.
9	Marsh gas or methane.	CH_4.	Of feeble narcotic action, the quantities contained in the intestine are without influence.
10	Ethylene.	C_2H_4.	Of weak narcotic action.
11	Nitrogen.	N.	Acts similar to laughing gas, but paralyses the sensation of pain later than this one. Death with spasms of suffocation.
12	Carbon dioxid(carbonic acid misnomer).	CO_2.	Of local excitating action upon mucous membranes, and of remote paralyzing action upon brain and spinal cord.
13	Alcohol or ethyl-alcohol.	C_2H_5OH.	See page 129. The cheap kinds are always impurified with fusel (propylic-, butylic- and amylic-alcohol) furfurol, aldehyde, etc., all of which are still more poisonous than C_2H_5OH.
14	Chloral hydrate or tri - chlor - aldehyde hydrate.	$CCl_3.COH.H_2O$.	See page 128. Chloral-form-amid, chloralose, paraldehyde, acetal, somnal, are of similar action. Antidotes applied are picrotoxin and strychnin.
15	Sulfonal, or diethyl - sulfon - dimethyl-methane.	$(CH_3)_2C(SO_2C_2H_5)_2$.	Strong narcotic; however, 100 gms. have been endured, after causing twitchings, collapse of temperature, and protracted sleep for five days. Hæmatoporphyrin may appear in the urine. Therapy: washing of the organism, picrotoxin, strychnin.
16	Trional.	$CH_3.C_2H_5.C.(SO_2C_2H_5)_2$.	In every respect similar to the preceding.
17	Benzene and petroleum-ether.	C_6H_6, benzene; petroleum-ether consists of: C_6H_{14} and C_7H_{16}.	Both may produce, on inhalation or when taken internally, narcosis, twitchings, cyanosis, mydriasis. Even chronic poisoning obtains.
18	Antifebrin or acetanilid.	$C_6H_5.NH.CH_3CO$.	As with anilin, a strong blue coloration of the body, dyspnœa, weakness of heart. 28 gms. have been endured. Exalgin acts in a similar manner. Washing of the organism.
19	Antipyrin or dimethyl - phenyl - pyrazolon.	$C_{11}H_{12}N_2O$.	Cyanosis, dyspnœa, chills, collapse, spasms, exanthema. Three fatal cases on record. Tolupyrin acts in a milder way.
20	Apomorphin.	$C_{17}H_{17}NO_2$.	Most powerful emetic, causing excitation on animals incapable of vomiting. For man, a high degree of feebleness maintains, as well as dyspnœa and dizziness.

CHEMICAL PROPERTIES; DETECTION.

On narcotizing in presence of the light of a flame, it burns to carbonyl chlorid or phosgen ($COCl_2$), which is endowed with an excitating action upon mucous linings of its own, besides having the poisonous properties of CO. Within the organism, the greater part of chloroform will not be burned, but exhaled as such.

Easily volatile and combustible. Danger of fire. It is exhaled to the greater amount. Urine does not act reducing as in case of chloroform. A mixture of 1 pt. ether and 3 pts. chloroform, Spiritus Aetherus or Hoffman's drops, is a popular remedy in Germany. The commercial ether often contains hydrogen peroxid, if air and light had access. Concerning detection, see page 42.

Colorless liquid, sensitive to light and strongly refractive. Partly burned within the organism to form bromids. Breath has an odor suggestive of garlic; the same obtains for the organs. Detection as on page 42.

Easily condensible gas; danger of fire; partly burned within the organism to form chlorids. Breath and organs of ethereal odor. Detection as with ether.

Colorless liquid, suggestive of chloroform, sensitive to light, of specific odor, which is participated to the breath and the organs. In the urine partly in form of bromid. Detection as with chloroform.

Colorless ethereal liquid of specific odor, conveyed to the breath and the organs. Easily inflammable, very volatile. Separation as on page 42. In the only fatal case on record the detection was not attempted.

Boile at + 108°C; therefore on distillation it passes over much later than common alcohol, boiling at + 78°4C. It is of disagreeable odor; forms valerianic acid when heated with potassic chromate and H_2SO_4. To be obtained from mixtures by agitation with petroleum-ether or ether. In case of man, it is not found in the urine.

Compressible gas, appearing in the trade in the liquefied state and without detrimental impurities. When personally prepared it contains easily admixtures of nitric oxid, which in contact with air is transformed into nitrogen tri- and per-oxids, which violently attack the mucous linings and decompose the blood, forming met-hæmoglobin.

Forms in swamps, mines, sewers, and in the putrefactive processes in the intestine. It is odorless, colorless and inflammable.

Contained to the amount of 6 p.c. in illuminating gas from coal. Inflammable and of a somewhat sweet odor.

Normally contained in the atmosphere to the amount of 79 p.c.; together with CO_2, in the expirated air; odorless.

Formed in the processes of: combustion, putrefaction, decay. Best mode of determining it, by means of barium hydrate, with which it forms barium carbonate, insoluble in water.

The rectified distillate smells of alcohol, burns, forms acetic acid with spongy platinum and yields crystals of iodoform when treated with KOH and iodin. Diluted alcohol is transformed into chloroform when treated with bleaching powder. [$Ca(OCl)Cl$.]

For separation, see page 43. Uro-chloralic-acid is laevo-rotary, while the free glycuronic acid obtained from it by splitting with mineral acids, is dextro-rotary. Both will reduce Fehling's solution. Chloral hydrate or chloroform, when treated with resorcinol and an excess of KOH, give rise to a red coloration; in case of an excess or resorcinol, a yellowish-green fluorescence obtains.

The organs are extracted with alcohol, the residue of evaporation treated with boiling water, concentrated and taken up with ether. Powdered sulfonal, when treated with charcoal powder, will yield the odor of mercaptan. The formation of hæmatoporphyrin is all the more easy the less excess of alkali is at the disposal of the system.

Detection as for sulfonal. Here, also, hæmato-porphyrinuria may follow.

For detection, see page 42. A part of the benzene reappears in the urine as sulfuric acid-phenol-ester; another part will be exhaled. Petroleum acts similarly to petroleum-ether; detection on page 42.

No met-hæmoglobin in the urine, but an ample amount of free and coupled antifebrin. To be obtained by agitation with chloroform and ether. When treated with dry zinc chlorid, a moss-green fluorescence obtains.

Obtained by agitation with chloroform from acid or alkaline solution. Ferric chlorid turns the aqueous solution reddish-brown; nitrous acid turns it green.

Easily decomposable base. Petroleum-ether will abstract from the ammoniacal solution a raspberry-red product of decomposition. Ferric chlorid turns apomorphin the color of amethyst, auric chlorid a purple-red.

	CHLOROFORM.	CHLORAL HYDRATE.
ÆTIO.	Generally inhalations for the production of narcosis; rarely internally.	Mostly medicinal cases, caused by overdoses.
STAT.	More than 400 fatal cases on record; however, there is only one case of death to from 3000 to 5000 administrations.	More than 100 fatal cases on record, but since the maximal dose has been reduced new cases rarely obtain.
DOS. LET.	Internally, 60, even 99, gms. have been endured; in case of inhalations, 50 to 100 gms. have been withstood, while peculiarly susceptible persons have been killed by 10 gms.	People with a weak heart, potatores, may be killed by 5 gms. In individual cases, 1 gm. proved toxic.
ACT.	A protoplasmic poison, killing the protoplasm of the ganglion cells, at first those of the brain, then dissolving blood corpuscles and degenerating all tissues.	Protoplasm poisoned. In the first place, paralysis of the walls of all the vessels, then action of the heart and brain weakened simultaneously; later on the kidneys, mainly, degenerate.
SYMPT.	Locally, excitation at point of application. After resorption, we have first excitation, then paralysis of the cortex of the brain, then of the other parts of the brain, the spinal cord, and the elongated marrow. The pupils are first contracted, then dilated. The muscles become perfectly limp; all reflexes close; in case of long lasting narcosis, numberless red blood-corpuscles perish and produce icterus. In case of chronic administration, a certain adaptation takes place, and there is emaciation, anæmia, icterus, and weakening of the heart's action.	Local excitation at place of application, vomiting. After resorption, irresistible desire to sleep; great reduction of blood-pressure and dilatation of the vessels, mainly of the skin; later on, respiration becomes very slow and stertorous, skin cyanotic and cold. In chronic chloralism, disturbances of digestion, swelling of mucous lining of mouth, weakness of muscles, emaciation, diarrhœa, delirium, marasm. In case of sudden deprivation, appearances of abstinency, but not to such a high degree as with morphiomanes.
EXIT.	In acute cases, death normally by paralysis of the muscles of respiration; in cases of invalids or of awkward administration, by untimely stoppage of the heart's action. In chronic cases, the cause of death is generally the cessation of the heart's action.	Death ensues in healthy animals, and, in case of acute action, by paralysis of the muscles of respiration; with man, generally by sudden ceasing of the heart's action. In chronic cases, the cause of death is, exclusively, stoppage of the heart, generally occurring without prodrome.
DIAGN.	The specific odor leaves no doubt. The blood may be odorless, since the red blood-corpuscles enclose the chloroform.	The pupils, which are not contracted, exclude morphin, and the absence of any odor of the breath excludes chloroform.
THER.	Stop administration at once, artificial respiration, electric excitation of nervi phrenici. Ether injections of little value. Prophylactically much can be done: Closely investigate patient, before anæsthesia for heart diseases, for diseases of the vessels and lungs; allow plenty of air to be inhaled with the vapor, and use the purest chloroform, free from chlorin, phosgen-gas, arsenic. Insist on having constant supervision of pulse, respiration and pupils. In case of chronic poisoning, prevent access to the poison.	Artificial respiration in a warm bed; washing of stomach. Hypodermically, picrotoxin or strychnin; washing of organism with sugar-salt solution is indicated. Prophylactically, people with degenerated heart should not take it, and people with sound heart only doses below 3.0 gms, and never to be continued for months. In most cases chloralose may be substituted for chloral hydrate, which former is less poisonous.
P.-MTM.	In case of narcosis of short duration, nothing is found necessarily; sometimes a few bubbles of air in the blood on account of the forced artificial respiration. In chronic cases, icterus, fatty degeneration of the heart, liver, kidneys, and muscles of skeleton; general anæmia. In acute poisoning, all cavities of the body have the odor of chloroform. The blood contains unchanged chloroform, and the urine, besides a little chloroform, a reducing substance, and a substance similar to cystin. Chloroform itself, however, will reduce Fehling's solution.	Local phenomena of excitation of mucous lining of mouth and throat. Mucous lining of stomach degenerated and hæmorrhagic. The urinary canals show fine grains of fatty degeneration. The blood contains no chloroform; the urine contains large quantities of tri-chlor-ethyl-glycuronic acid, which will reduce cupric sulfate like sugar, but which turns the plane of polarized light to the left. The free glycuronic acid, however, split off by warming with mineral acids, is dextro-rotary.

ALCOHOL.	MORPHIN AND OPIUM.
Suicide, abuse, practical jokes.	Suicide, murder, medicinal poisoning, morphomania; frequent with physicians and nurses.
By far the most frequent of all intoxications is the chronic alcoholism produced by a long series of acute poisonings.	Next to alcohol, the most frequent intoxication, at least in England. The habit of smoking opium is increasing.
60.0 to 180.0 gms. cause acute cases, according to constitutional susceptibility, adaptation and surrounding temperature. For chronic poisoning, the doses are very variable.	Children under five years will be strongly poisoned by from 0.01 to 0.03 gms.; adults will die after 0.4 gms., provided they are not accustomed to the drug; morphomaniacs stand several gms. daily.
Locally, alcohol has a dehydrating and coagulating action upon albumin, thus causing inflammation. Remotely, its first action upon the brain is that of excitation, and then of paralysis.	The cat, horse and cow show cerebral excitation; man is narcotized. The peristalsis of the intestine is stopped, and the respiration paralyzed. In chronic poisoning, the morphin acts as an indispensable stimulant.
In case of acute poisoning, common intoxication; loses control of the motor nerves of locomotion and cannot walk steadily or at all; want of sensation, reddening of the face, stertorous breathing, vomiting, involuntary stool; pulse hardly noticeable; sticky skin, cyanotic; Temperature and blood-pressure greatly reduced. (Katzenjammer.) In chronic poisoning, catarrhal inflammation of the mucous lining of the throat, mouth, œsophagus and stomach. The liver at first is infiltrated with fat and enlarged, then cirrhotic; the vessels show fatty and atheromatic degeneration, kidneys degenerated. Decrease of visual faculties and of intelligence; tremor; skin furunculous; the resistance of the body greatly reduced; in case of sudden discontinuation, delirium tremens and collapse. (Quartal-Saufen.)	In acute poisoning, desire to sleep, stupefaction, want of sensation, Cheyne-Stokes' respiration, strongest retardation of pulse. To the excitation of the brain corresponds the widening of the pupils, where with narcosis there is contraction of the pupils to the highest degree. Red exanthema of the skin; there may also occur increase of the secretion of mucus and saliva. In chronic cases, insomnia and want of appetite; at times constipation, at others bloody diarrhœa; pupils persistently contracted; the skin, leathery and hardened, shows numerous abcesses; frequent vomiting; diminution of potency and of energy; habit of lying; carelessness in regard to family and occupation; intermittent attacks; tremor; hallucinations.
In acute poisoning, either a gradual change to a healthy sleep and recovery, or coma, œdema of lungs, and paralysis of muscles of respiration. In chronic poisoning, progressive paralyses, the same as with insane people, or suicide in a delirious spell, or death by intercurrent diseases, especially pneumonia, or dropsy, or cancer of the stomach or œsophagus.	In acute poisoning, deepest coma and strongest retardation; œdema of lungs, spasmodic twitching of legs, and bloody diarrhœa may occur. In chronic cases, marasm and complete apathy. In case of deprivation of morphin, terrible excitement, followed by fatal collapse.
In acute cases, the odor is sufficient for diagnosis. In chronic cases, along with the odor, palpation of the liver and inspection of the throat and face.	In acute cases, the contracted pupils, retardation of pulse, and narcosis are sufficient. In chronic cases, morphin is to be chemically proven in the vomit and stool.
In acute cases, when there is congestion of the brain, bleeding, washing of the organism, cold compresses on the head, ammonia, black coffee. In case of collapse of temperature, warm-bottles, mustard-plasters, artificial respiration and passive movements; in chronic cases, treatment in asylums and surveillance for years. Do not discontinue its use too suddenly. The appetite is stimulated by bitter substances. Baths. Excision and cauterization of the red acne. In case of delirium, do not give chloral hydrate but scopolamin. Prophylactically, support the temperance movement, and give warning, even in the schools, against alcoholic beverages. (Schnapps.)	In acute cases, same as with alcohol; hypodermic injection of atropin in maximal doses has repeatedly given good results. The most important measure is washing of the stomach, even after hypodermic injection. Potassium permanganate internally and hypodermically in exceedingly dilute aqueous solution; one grain to a tumblerful of water. One grain potassium permanganate will annihilate one grain of morphin (Wm. Moor). Useful, provided the morphin has not entered the blood. In chronic cases, we can think only of improvement in an asylum. Gradual and cautious deprivation with most careful surveillance. In case of collapse, single doses of codein, cocain, spartein. Prophylactically: every young physician should vow never to take it himself; never allow a patient to use the syringe himself; prescribe only the minimum quantity in *one* prescription.
In case of acute poisoning, hyperæmia of the brain and even apoplexy; ecchymosis and inflammation of the mucous lining of the intestines; œdema of lungs; all cavities of the body smell of alcohol. In chronic cases there is first fatty degeneration of the liver, then cirrhosis of the liver; fatty degeneration of the intima of all arteries, arteriosclerosis, pharyngitis, laryngitis, œsophagitis, gastritis, chronic nephritis. Stomach shows ulcers and carcinomatous changes of the mucous membrane. The fat of all organs shows a remarkable amount of water.	In case of acute poisoning by opium, its specific odor may be noticed in the intestines and cavities of the body. In acute cases of morphin-poisoning there may be no finding at all; however, we find, as a rule, an increase of blood in the meninges and accumulation of cerebro-spinal liquid in the cavities of the brain; there is congestion of the lungs and extreme quantities of urine in the bladder, which contains oxydi-morphin and morphin. In chronic cases, it is said that we may find, besides marasm, degeneration of the posterior columns of the spinal cord. The mucous lining of the intestines is ulcerated

	CICUTOXIN.	PICROTOXIN.	DIGITALIRESIN	TOXIRESIN	SANTONIN.
ÆTIO.	Mistaking the water-hemlock, cicuta virosa, for edible umbels, *e.g.*, for celery.	Partaking of beer containing picrotoxin, of cocculus indicus, of fish poisoned by it.	May be formed by decomposition of digitalin and of digitalein, and acts like picrotoxin.	May be formed by decomposition of digitoxin, and acts like picrotoxin.	Excessive doses of santonin or of wormwood powder in cases of ascarides.
STAT.	About 40 cases, with 45 p.c. mortality.	Growing less frequent nowadays.			Poisoning of frequent occurrence; mortality low.
DOS. LET.	Unknown.	2.4 gms. of seeds.			Unknown.
ACT.	Excitation of Nothnagel's tetanic centre in the elongated marrow, as well as of the centres of the vasomotors, respiration, and of the vagus of the heart.	The action is between that of cicutoxin and of strychnin. The picrotoxinin contained in picrotoxin acts in yet stronger manner.			Action strongly suggestive of picrotoxin, but the cortex of the brain is not involved. The drug contains, besides the poison, a poisonous ethereal oil.
SYMPT.	Nausea, vomiting, colics, increased heart-action, staggering, unconsciousness, terrible epileptiform spasms, grinding of the teeth; pupils widened, pulse hard and slow; dyspnœa, salivation.	Nausea, vomiting, diarrhœa, numbness, trembling, convulsions—even tetanus; salivation, dyspnœa, perspiration, delirium; on man, mydriasis; fish will be stunned.			Purple vision, xanthopsia, aphasia, hallucinations; further, as picrotoxin. For animals, icterus and hæmaturia. Violent diarrhœa may occur.
EXIT.	Death by paralysis of the previously irritated centres.	Paralysis of limbs, retardation of pulse. Death caused by paralysis of respiratory apparatus.			Death by brain-paralysis. Paresis of lower extremities.
DIAGN.	The vomit contains particles of the plant.	The vomit is of very bitter taste.			Xanthopsia assures the diagnosis. For urine, see below.
THER.	Chloroforming, chloral hydrate; artificial respiration.	Same as for cicutoxin. Prohibit the use of the seeds.			Same as for picrotoxin. The medicine should not be sold by peddlers.
P.-MTM.	Finding may be entirely negative. Or, we find ecchymoses, *e.g.*, underneath the pleura, and irritation of the mucous coat of stomach. The abdomen has at times been found much inflated. No poison can be chemically detected in the urine.	Generally nothing characteristic. Hyperæmia of brain and lungs; salivary glands swollen; heart flaccid; the unchanged poison detectable in the urine. Addition of KOH does not change the color of the urine.			Finding may be negative. Hyperæmia of the brain, ecchymoses; icterus, polycholia; the urine contains albumin, a chromogen and a compound of mono- and di-oxy-santonin. On putrefaction or upon addition of KOH, the urine turns scarlet-red.
REM'KS.	Comp. p. 122, No. 21.	Comp. p. 122, No. 22.	C. p. 137.		Comp. p. 124, No. 23.

ACONITIN.	COLCHICIN.	VERATRIN.
Murder, medicinal poisoning, accident, mistaking the plant.	Medicinal poisoning, mistaking the plant, whose leaves and flowers develop at different periods.	Unskilled handling of the seeds as a destroyer of lice; medicinal poisoning; mistaking the plant.
Poisoning not frequent; mortality large.	Infrequent poisoning; mortality about 50 p.c.	Poisoning rare; mortality low.
A few milligramms.	About 0.06 grms.	Unknown; probably same as colchicin.
Local irritation; in addition: first irritation, then paralysis of the central-nervous system. Correspondingly, first irritation of the vagus of the heart, then paralysis of the same.	Local irritation; ascending paralysis (after short duration of excitation) of spinal cord and medulla oblongata. Muscular action as in case of veratrin.	Local irritation of the terminations of the sensory, motor, and secretory nerves, as well as of the brain and spinal cord. Peculiar muscular action on the frog—veratrin rigidity. See p.
Burning in mouth, salivation, vomiting and purging, dizziness, formication, mydriasis, retardation of pulse, dyspnœa, spasmodic paroxysms. A part of the poison is voided in an unchanged state by the saliva.	Burning in mouth, salivation, hæmatic vomiting and purging, dizziness, delirium, slight convulsions, irregular pulse, great debility. Goats will secrete the poison in an active form, partially with the milk.	Burning in mouth, salivation, vomiting and purging, raving mania, spasms, retardation of pulse, dyspnœa, great debility, lowering of temperature. The action upon the muscles, so pronounced in the frog, is absent in man.
Retardation of pulse changed into acceleration of the same; paralyzed respiration.	Death with gastro-enteritis and collapse. In case of recovery, chronic dysentry.	Death with collapse by paralyzed respiration, after the pulse had become accelerated.
Widening of pupils simultaneously with retardation of pulse and burning in mouth.	Detection of the plant in the vomit; anamnesis in regard to medicines.	Detection of the plant in the vomit; anamnesis as to medicines.
Artificial respiration, for the spasms induce narcosis.	Mucilaginous potions, opium; warm compresses on the abdomen.	Scopolamin hypodermically; narcotics to stop excitation; abundant warm tea; calcium chlorid.
Finding possibly negative. Swelling and pointed ecchymoses in the intestinal canal and under the serous membranes. In the urine the unchanged alkaloid; also in blood, liver, kidney. On attempting to regain it, it decomposes easily. The then produced splitting-products are hardly poisonous.	Finding in man, so far negative; for the animal, hæmorrhagic gastro-enteritis, parenchymatous nephritis, degeneration of liver, hyperæmia of the articular surfaces of the joints and of the marrow of the bones. Oxy-di-colchicin detectable in the organism. The colchicein accompanying the colchicin in the plant and in the cadaver, is inert.	Finding in man so far always negative; in the animal, gastro-enteritis may be found. We have further observed hyperæmia of the brain and its membranes, and of the lungs and kidneys. The substance is very soon traceable in the urine. The accompanying alkaloids, such as veratridin, cevadillin, sabadillin, pass over into the urine.
Comp. p. 124, No. 33.	Comp. p. 124, No. 32.	Comp. p. 124, No. 30.

	PHYSOSTIGMIN.	STRYCHNIN.
ÆTIO.	Partaking of Calabar-beans through ignorance; medicinal poisoning; mistaking external for internal medicine.	Murder; medicinal poisoning; partaking of fox- or rat-poison; beer containing strychnin; confusion.
STAT.	Now a rare intoxication; fatal cases scarce.	More than 60 cases known; mortality 35 p c.
DOS. LET.	Unknown. In medicine bottles the physostigmin is soon rendered less active.	4 mgr. for children; 30 to 100 mgr. for adults.
ACT.	Local action almost absent; central excitation of the brain and peripheral excitation of the muscles of the iris, vessels and salivary glands.	Local action entirely absent. Excitation of the spinal cord, medulla oblongata and brain. Accumuulative action on repetition.
SYMPT.	Salivation, vomiting and purging, colics, abortion, retardation of pulse, increase of blood-pressure, maniacal attacks, spasms, contraction of pupils, dyspnœa. Myosis will follow promptly upon dropping of mineral doses.	Increased excitability of reflexes, drawing in limbs, rigidity of neck, stiffness, trismus, tetanus, opisthotonos; periodic increase of blood-pressure, retardation of pulse; sudden frights, fear, protrusion of eyeballs.
EXIT.	Death with spasms by paralysis of respiratory organs. Rigidity of muscles does not obtain during the interval between spasms.	Death with spasms by stoppage of respiration and marked decrease of blood-pressure.
DIAGN.	Spasms and raving mania, with markedly contracted pupils.	Terrified from slightest cause; rigidity of neck and increased reflex sensibility.
THER.	Scopolamin hypodermically. Artificial respiration.	Chloral hydrate, chloroform; wash-of organism, artificial respiration, curarization.
P.-MTM.	Finding in man may be negative; or we find slight irritation of the gastro-enteritic mucous membrane. For animals, gastro-enteritis. In urine, saliva, bile, the poison is contained in form of an insoluble modification. Such an one also occurs preformed in the drug along with physostigmin.	Pronounced rigidity of corpse, frequently in position of tetanic contraction. Hyperæmia and extravasation into spinal cord and brain. Lungs surcharged with blood; heart but little filled; urine contains albumin, lactic acid, strychnin, and sugar; liver and central-nervous apparatus rich in strychnin.
REMARKS.	See page 124, No. 29.	See page 124, No. 35.

CYTISIN.	CONIIN.
Accidental partaking of seeds, flowers, leaves or bark of the different species, which in Europe are nursed in gardens and parks. Cytisus.	Murder; suicide; medicinal poisoning; confounding the plant with parsley, chervil, parsnip; use of conium plasters.
131 cases, with 2 p,c. mortality.	So far, about 20 fatal cases.
Unknown, but not large.	0.15 grms. of the pure alkaloid.
Local action absent. Excitation of brain, medulla oblongata, and spinal cord, with following paralysis. On application of large doses, paralysis of the terminations of motor-nerves, suggestive of curare.	Local corrosion. Excitation of the motor-centres in brain and spinal cord, with often very rapidly-following paralysis. In frogs, paralysis, similar to the action of curare, of the terminations of motor-nerves. This, however, may depend on accompanying bases.
Nausea, vomiting, salivation, excitation, convulsions, tetanic spasms. Blood-pressure strongly increased. Colics, tumultuous beating of heart, diarrhœa, cyanosis. Herbivorae tolerate much more than carnivorae. (Goats pro kg. 36 times more than cats.)	Burning in mouth, soreness in throat, salivation, dizziness, nausea, vomiting, mydriasis, feebleness of legs, spasms of muscles of calves, convulsions, delirium, dyspnœa. Animals will tolerate, internally, very large doses. Action upon the heart suggestive of nicotin.
As a rule, everything is voided by vomiting. On hypodermic injection, death by stoppage of respiration.	Death by paralysis of respiration, with or without spasms. The data of literature are contradictory.
The vomit contains parts of the plant; the urine very soon contains cytisin.	The specific odor in the vomit of the base and parts of the plant.
In the stage of spasms give narcotics; much liquid; artificial respiration in case of paralysis of organs of respiration.	Artificial respiration; washing of organism. In case of spasms, apply narcotics.
Finding negative, as a rule. Sporadically, injection of the gastro-intestinal canal and œdema of the brain have been found. Accumulation of poison in central-nervous system not proven. In goats the poison passes unchanged and partly into the milk, which thus is rendered poisonous for man.	Now and then irritation of the mucous coat of the intestines, even hæmorrhages. Hyperæmic meninges; sometimes œdema of the lungs. All cavities of the body are pervaded by the odor of coniin. Blood is said to coagulate with difficulty. Confusing of coniin with cadaverin (corpse-coniin) easily possible.
See page 124, No. 37.	See page 122, No. 20

	ATROPIN.	NICOTIN.
ÆTIO.	Medicinal poisoning: ignorance in regard to plants in question; mistake.	Abuse of tobacco; use of tobacco as injection per rectum; partaking by children.
STAT.	Rather frequent; mortality 11.6 p.c.	Chronic poisoning very frequent, slight acute ones also; severe ones rare.
DOS. LET.	130 mgrs. for adults; 95 mgrs. for children.	Not more than 0.06 grms. for nicotin; 0.8 grms. of snuff, internally.
ACT.	Paralysis of secretion of all glands proper, of visual accommodation, of inhibitory nerves of heart, of the motor-elements of the intestines, and of the muscles of the vessels. Irritation of the cortex of brain and increased reflex excitability of spinal cord. Hyoscyamin causes spasms of accommodation.	Local irritation at place of application. The same organs, paralyzed by atropin, are at first excited and then paralyzed; this also obtains for brain and spinal cord. Death is caused by paralysis of the respiratory centre. In regard to the pupils, see page 72; 1, b.
SYMPT.	Dryness in mouth and throat, hoarseness, difficulty of swallowing, reddening of face, acceleration of pulse, dry, hot skin, disturbances of vision, mydriasis; dizziness, delirium, raving mania; scarlet exanthema. Death with general paralysis. In warm-blooded animals, twitchings; in the frog, the so-called late tetanus. Concerning the pupil, see page 72.	Burning sensation in mouth, salivation, vomiting and purging; pulse first slow, then arrhythmic; perspiration, myosis, visual disturbance. Dizziness, dazed state, dyspnœa, abortion, convulsions. In chronic cases, catarrh of pharynx, larynx and middle ear; violent beating of heart, limitation of field of vision, scotomy, weakness of memory, dizziness, gastralgia; arteriosclerosis; psychoses.
THER.	Injections of pilocarpin; washing of organism; dropping physostigmin into the eye. Ice-cap. Morphin acts splendidly in a symptomatic sense.	Prohibition of smoking and of remaining in places filled with tobacco smoke, in chronic cases. In acute cases, washing of organism; atropin at the stage of excitation.
P.-MTM.	But little is positive. Mydriasis; meninges overcharged with blood. In case of partaking of berries of deadly nightshade, stomach is of dark-blue color; in case of datura, extravasation and inflammation of gastro-intestinal mucous coat. The urine contains unchanged atropin; it is found in the brain and liver of the cadaver.	Tobacco odor of all organs; inflammation of mucous linings of throat and stomach (in case of internal application); intestine contracted; contains some hæmatic mucous. Heart flabby. Meninges hyperæmic. Urine contains unchanged nicotin. Pupils not necessarily changed.
REMARKS.	See page 122, No. 12.	See page 122, No. 16.

PILOCARPIN.	COCAIN.
Almost exclusively medicinal poisoning and mistakes are to be considered.	Medicinal poisoning; abuse of the alkaloid, as well as of the leaves.
Slight poisoning frequent; even fatal ones on record.	Since 1884 eleven fatal cases and more than 200 grave intoxications.
Somewhat larger than for nicotin. For the Jaborandi-leaves incalculable.	1.0 grms., internally or hypodermically, acts fatally.
Local irritation absent. Those organs paralyzed by atropin will be at first excited and then paralyzed; but the excitation lasts longer than with nicotin. Death with œdema of lungs. Concerning the pupils, see page 72; 1, b. As a food, pilocarpin need not be considered. The ethereal oil contained in jaborandi-leaves acts as a diuretic.	Central and peripheral action. First excitation, then paralysis of the cerebrum. Subdued sensibility of terminations of sensitive nerves. Paralysis of vagus of heart. Mydriasis, depending upon irritation of the peripheral terminations of the nerves of dilating muscles of pupils. In case of chronic intoxication, psychical degeneration.
Salivation, perspiration, coryza, nausea, vomiting and purging; pulse at first slow, then arrythmic; myosis, lachrymation, spasms of accommodation; abortion; stinging in urethra, and tenesmus with vesical pain; attacks of yawning, headache, dizziness, rattling sound in trachea. In chronic cases, inflammation of perspiratory- and salivary-glands. Unknown whether persistent visual disturbances obtain as with nicotin.	Dryness of throat, with burning and furry feeling; trouble in swallowing; nausea, vomiting, pain in abdomen; accelerated pulse and beating of heart; mydriasis; protrusion of eyeballs; Cheyne-Stokes' breathing. Gayness, state of inebriation, then melancholy. Precordial terror, sensation of swooning. Cyanosis. collapse, amaurosis; paresthæsia; twitching, convulsions, tetanus; paralyses. In chronic cases the state is very suggestive of morphinism.
For decrepit individuals pilocarpin should be altogether avoided. Never use pilocarpin without having atropin at hand; this will at once check the intoxication.	Sinapisms on heart and stomach; ammonium carbonate; washing of organism. In case of spasms, narcotics. Artificial respiration. In case of chronic cocainism, remove patient to a lunatic asylum.
Dissections of human subjects are scarcely on record. Organs inodorous. In gastro-intestinal canal injections of mucous membranes and liquid contents. Decided œdema of lungs. Hyperæmic meninges. Changes of pupils do not of necessity obtain. The urine contains unchanged pilocarpin; after partaking of jaborandi-leaves, also pilocarpidin.	Hyperæmia of brain, liver, spleen and kidneys. For animals, vacuolary degeneration of the liver-cells has been found, with accompanying fatty degeneration and necrosis. In chronic cases a high degree of emaciation. Widening of the pupil of the cadaver, in case of acute poisoning, need not of necessity persist. Urine contains unchanged cocain.
See page 122, No. 17.	See page 122, No. 5.

II.—CARDIAC-POISONS.

Nothing would be more erroneous than to suppose that the poisons thus far mentioned exert no influence upon the heart. Such action is present even to a high degree, in a paralyzing sense, following the use of *hydrocyanic acid, phosphorus, arsenic, antimony, carbon monoxid, alcohol, chloral hydrate*; and it is quite a peculiar one in the case of *atropin, pilocarpin, nicotin* and *coniin*. This has already been stated explicitly on pp. 62-65 and 134-135. We here add that *atropin* and *hyoscyamin* primarily paralyze the inhibitory ganglionic centers of the heart and thus will cause acceleration of the pulse in warm-blooded animals; small doses of *scopolamin* (*hyoscin*) in man, do not paralyze the mentioned ganglions, but feebly excitate them. A review of all poisons influencing the pulse, would properly belong here, but for the sake of brevity the above reference may suffice.

In the following table we mention only those poisons whose action upon the heart is predominant and directly instrumental in bringing about the fatal issue. The prototypes of such are *digitalin* and *muscarin*, the action of which is described in detail in table, page 137. *Digitalin* is by no means the only substance upon which the poisonous action of the foxglove (*digitalis*) depends; in addition to this, the plant contains two other substances of identical action, viz., *digitoxin* and *digitalein*. The latter is readily soluble in water and alcohol, while the two former dissolve well only in boiling alcohol. By putrefaction, or other decomposition, all three substances furnish two active products of splitting, viz., *digitaliresin* and *toxiresin*, both of which are without action upon the heart. They are mentioned on pp. 130-131. The *digitaliresin* may be further split when non-poisonous *digitaligenin* is formed. It is not known if *toxiresin* allows of further splitting. Badly dried *digitalis*-leaves or infusions decomposed by bacterial life may cause the decomposition of the three mentioned heart-poisons, and in using such medicines, violent spasms may obtain. *Digitonin* and its products of decomposition, *digitogenin* and *paradigitogenin*, etc., belong to the *saponin*-group, (page 112) and scarcely act when *digitalis* is given internally. But in the decoction of the leaves they hold in suspension the active substances which are insoluble in water.

Amanita muscaria contains *muscdrin* and *amanitin* (or *sinkalin* or *cholin*), but the latter is of no import in case of poisoning by the mushroom; the *amanita-atropin*, which is frequently present, modifies the intensity of its manifestation.

TABLE OF THE TWO MOST IMPORTANT HEART-POISONS.

	DIGITALIN.	MUSCARIN.
ÆTIO.	Medicinal poisoning; partaking of the leaves, flowers or seeds of the red fox-glove (digitalis), or of the officinal preparations.	Partaking of amanita muscaria (false orange), green, dried, uncooked or cooked.
STAT.	Slight intoxications witnessed by almost every physician, grave ones rare.	In Russia and Siberia not rare in man (food) and cattle.
DOS. LET	Unknown, but very small.	Unknown, but very small.
ACT.	Local irritation of mucous membranes and of the subcutaneous connective tissue. After resorption, irritation of the vagus-centre and of the peripheral apparatus of the heart-vagus and the vessels. The excitation of the vagus and heart-muscles is later on changed into paralysis. Action upon the frog heart, see page 65. $BaCl_2$ is of similar action.	No local action. After resorption, the amanita of high latitudes causes a certain inebriation conjointly with excitation. The pure muscarin irritates all peripheral apparatus which are paralyzed by atropin. Compare page 134. The musculature of the heart and vessels is not at all influenced. Action upon the frog-heart, see page 63.
SYMPT.	Nausea, vomiting, pressure in the region of the stomach, pains by colic, diarrhœa; cardiopalmus; pulse wiry, diminished to 40; hammering sensation of carotids; pain in forehead, dizziness, buzzing in ears, darkening of field of vision; anuria; abscesses after subcutaneous injection; pupils generally dilated. Concerning the action of the products of decomposition of digitalin-substances, see pages 130 and 136.	Nausea, salivation, perspiration, rumbling in abdomen, vomiting and purging. After initial acceleration of pulse, retardation, even below 40; pulse soft, intermitting. Pain in forehead, dizziness, visual disturbances, contracted pupils, spasms of accommodation, lachrymation; desire to urinate, salivation; abortion. The accompanying aikaloid reduces the strength of all symptoms.
EXIT.	Pulse becomes arrhythmic, greatly accelerated, small; fainting spells; face remarkably pale. Heart stops, and death in coma with or without spasms.	Stools become hæmatic. Thickening of blood as in the case of cholera. Rattling in trachea, œdema of lungs. Pulse becomes steadily slower, finally is imperceptible, but is never wiry.
DIAGN.	The pulse, at first very strong and very slow, later on becomes directly the opposite.	Fragments of the mushroom in the vomit; pulse very slow until death, but soft; increase of all secretions.
THER.	There is no antidote existing. Treat symptomatically. Atropin will only remove the retardation of pulse.	Atropin, hypodermically, will at once remove the danger of life and almost all symptoms. In spite of this, washing of the stomach is necessary.
P.-MTM.	The finding may be negative. According to experiments on animals, sub-endo-cardial hæmorrhages into the left ventricle should not be rare. Heart ceasing to heat in systole only occurs (for animals) after excessive doses. The gastro-intestinal canal may show appearances of irritation.	Aside from the œdema of the lungs, which cannot be considered, but little characteristic; the finding may be a negative one. The heart is remarkably flabby; both sides filled. The intestine contains a reddish liquid; the mucous membrane of the intestine may be partly hæmorrhagic. The blood very much thickened. Degeneration of the organs (?).
DETECT.	So far it has been impossible to find even a trace of the active substances of digitalis either in the organs or in the urine. It remains very questionable whether digitalin may be obtained from the contents of the intestines according to Dragendorff's method. The proof has to be restricted, therefore, in most cases, to digitonin, which is contained in digitalis besides digitalin, and which belongs to the saponin-substances mentioned on page 112. For instance, we can obtain it by agitation with amylic alcohol, and it will turn a beautiful red with conc. HCl or H_2SO_4. The pharmacognostic proof of fragments of leaves of fox-glove insures the diagnosis. Also the physiological proof is decisive.	Up to date the proof has never been rendered. Furthermore, even if we should be able to detect it, such proof would not be conclusive, since there exists a cadaver-muscarin. Compare pages 38, 48 and 49. The physiological proof ensures the diagnosis, and should be conducted in such a manner as to free the isolated alkaloid from the amanita-atropin, which is soluble in ether. A special reaction for muscarin is wanting; it might reappear unchanged in the urine; this has long been known to be true of the inebriating substance of the northern amanita. The pharmacognostic proof of fragments of amanita will also insure the diagnosis. Among the products of decomposition of muscarin, we find tri-methyl-anin.

No.	NAME OF PLANTS.	FAMILY.	POISONS.
1	Digitalis purpurea, ferruginea, ambigua, parviflora, grandiflora, aurea, nervosa, gigantea, eriostachys, glandulosa and Fontanesii, fox-glove.	Scrophulariac.	Digitalin, digitalein, digitoxin. The first two are glycosids, the latter a bitter principle.
2	Helleborus niger, fœtidus and viridis.	Ranunculac.	Helleborein, glycosid.
3	Coronilla scorpioides, juncea, montana, pentaphylla and varia.	Papilionac.	Coronillin, alkaloid.
4	Adonis vernalis, æstivalis, autumnalis, flammea, cupiana, amurensis.	Ranunculac.	Adonidin, glycosid.
5	Convallaria, majalis, lily of the valley.	Liliac.	Convallamarin, glycosid.
6	Polygonatum multiflorum and officinale, Solomon's seal.	Liliac.	Possibly convallamarin.
7	Nerium Oleander.	Apocynac.	Oleandrin and neriin, glycosids.
8	Nerium odorum.	Apocynac.	Neriodorin and neriodorein, glycosids.
9	Apocynum canabinum, common Indian hemp.	Apocynac.	Apocynin and apocynein; only the latter is a glycosid.
10	Scilla maritima or Urginea scilla, squill.	Liliac.	Scillain and Scillitoxin, glycosids.
11	Strophanthus, many species.	Apocynac.	Strophantin or methyl-ouabin, and ouabain, glycosids.
12	Cactus grandiflora.	Cacteæ.	Cactin, glycosid (?).
13	Tulipa gesneriana.	Liliac.	Tulipin, alkaloid.
14	Agaricus muscarius or Amanita muscaria, false orange (mushroom).	Hymenomyc.	Muscarin, alkaloid.
15	Agaricus pantherinus or Amanita pantherina.	Hymenomyc.	Muscarin, alkaloid.
16	Boletus luridus.	Hymenomyc.	Muscarin, alkaloid.
17	Boletus satanas.	Hymenomyc.	Very likely muscarin.
18	Areca catechu, areca-palm.	Palmæ.	Arecolin and other alkaloids.

ACTION; CHEMICAL BEHAVIOR.
Considerable difference in the toxic quantity exists between the different species. Dig-ferruginea acts strongest. The active substance, in all species, is contained mostly in the seeds; next the leaves; even the roots are not inert.
Mainly the root, and the leaves near the root, contain the poison. The last-named species in particular contains, besides, a glycosid possessed of action upon the heart, viz., helleborin. Conc. H_2SO_4 turns this a beautiful bright red.
The seeds mainly contain the poison, which is very bitter. It forms crystalline salts, soluble in water.
All parts of the plant contain the poison, the herb mainly. It is easily soluble in ether-alcohol. It disappears in the organism.
Also convallarin in addition, contained in the entire plant; soluble in water and alcohol, insoluble in ether. Beautiful purple coloration with conc. H_2SO_4.
It is probable that neriin and oleandrin are identical. Oleandrin is soluble in chloroform. The leaves contain the poison.
According to Schmiedeberg, identical with oleandrin and neriin. Common as an ornamental plant in England. The poison is contained in the leaves.
Similar to neriin and oleandrin. The plant is indigenous to America, and popularly used.
Possesses all virtues of digitalin, but causes stronger irritation as a vomitive. Soluble, with red color, in conc. HCl. Mostly contained in the bulb.
Now officinal in form of Tinct. Stropanthi, prepared from the seeds. Acts like digitalin.
The beautiful flowers of this popular ornamental plant are the poisonous part.
It is not assured whether this poison belongs to the digitalin-group or not.
The false orange is occasionally gathered and eaten instead of the non-poisonous Amanita cæsarea, the orange (mushroom).
Frequent in Europe and Asia.
This mushroom is of frequent occurrence in German pine- and leaf-forests.
This mushroom grows particularly on shell-limestone, and it is very poisonous.
Arecolin is nearly related in its action to muscarin.

APPENDIX.

A Review of Toxicologically Interesting Products of Metabolism (Exchange of Material).

We have mentioned on pages 13 and 38, that certain products of metabolism are of decided toxicological interest, particularly those consequent to micro-organisms and appearing in certain diseases and in corpses. For the most meritorious labors concerning our knowledge of these substances, we are indebted to FRANCESCO SELMI, L. BRIEGER, and later on, GRIFFITH. The following data, even though imperfect, will serve to give an insight into these substances. The albumin is the **source** of most of the poisonous products of metabolism. Albumin itself may undergo poisonous change (toxalbumins, enzymes), or, by decomposition, it may admit of the generation of numberless poisonous products; such products of decomposition belong to the groups of *alkaloids*, *amido-acids*, *acids*, etc. They generate partly in the intestinal canal and partly in the other organs most varyingly. Albuminous esculents, even before they are taken into the body, may give rise to the formation of poisons.

I.—Concerning Esculents Undergoing Toxic Change.

I.—ANIMAL ESCULENTS.

Poisoning by sausage is also called *allantiasis* (from ἀλλᾶζ, ἀλλᾶντος = sausage), or *botulism* (from *botulus* = sausage). The first one to describe it more exactly was JUSTINUS KERNER. It is identical with a certain form of fish-poisoning. Poisoning by meat has not infrequently been compared and confounded with typhus and intestinal sepsis. It has occurred principally, and in wholesale fashion, at popular festivals (Volksfeste, Vogelschiessen, etc.), and we cannot say that it is on the decrease. Similar cases of cheese-poisoning appeared during the last decade, mainly in North-America. The poison involved in this case, *tyrotoxin*, derives its name from τυρός = cheese.

TABLE OF POISONING BY SAUSAGE-, MEAT-, AND CHEESE-POISON.

	SAUSAGE-POISONING.	MEAT-POISONING.	CHEESE-POISONING.
ÆTIO.	Concerns sausage difficult to smoke because of improper additions (raisins, milk, bread-rolls), or because of excessive volume (sausages stuffed into the stomach-membrane or into entire bladders). Depends upon products of metabolism caused by microbes partly yet unknown. The most important of these products is ptomatropin. Occasionally it is also formed in rotten fish (mainly in sturgeon), in corned beef, in old roasted meat, stale shrimp, etc.	Depends upon the use of diseased animals for slaughter. The most dangerous diseases are pyemia, septicemia and metritis. The bacteria present in these diseases give rise to a series of poisonous products of metabolism which are not wholly destroyed on boiling. As a rule, beef and veal are involved. It is by no means essential that the meat shows green discoloration.	Depends upon (1) a poison developed in cheese by bacteria, which is called tyrotoxicon, or, more correctly, tyrotoxin; (2) a still unknown alkaloid, recently discovered by LEPIERRE. Occasionally we meet tyrotoxin in milk, ice-cream, cream-cake, oysters (cardium edule) and mussels (mytilus edulis). It is unknown in how far it is changed by boiling.
STAT.	In Suabia (sausage) and in Russia (fish) frequent. Mortality 40 p.c. In Württemberg 400 cases up to 1853; now less frequent there.	More than 40 intoxications of whole masses of people are on record in Germany and Switzerland; the last decade alone embraces 1500 cases. Mortality varies greatly.	Intoxication observed as far back as 1820. In 1883–84, 300 cases, very few fatal, occurred in Michigan. In Germany, only sporadic cases.
SYMPT.	They frequently do not occur sooner than 12–24 hours after partaking, and are similar to those of atropin. (See page 134.) Difference; the heart-beat is not accelerated, but enfeebled; ptosis is added to the oculo-pupillary appearances, even paralysis of the nuclei of the abducens, trochlearis and oculomotorius. Hoarseness, barking cough, decreased saliva, paralysis of throat, paresis of glottis.	Vomiting and purging, somnolency, fever. Sometimes the appearances of the disease were exactly the same as in abdominal typhus or in cholera. The time of incubation varied between 6 to 48 hours. In isolated cases, raging delirium, twitchings and cramp in the calves were added. Complications with sausage-poisoning very frequent.	In 2 to 4 hours after partaking, vomiting and purging of choleraic character, gastric pain, dizziness, tongue at first white, then red and dry; pulse weak and irregular; face at first pale, then cyanotic; spasms; feeling of prostration. Complication with sausage-poisoning occurs. In cats the cheese-poison is harmless; even the pure cultures of cheese-bacteria have here had no effect.
EXIT.	Death after 4–8 days, very likely by paralysis of the heart; or very slow recovery. Marked emaciation.	Death after 1–11 days, with cyanosis and collapse suggestive of cholera; or slow recovery.	Death by exhaustion after 4 to 8 days; generally, however, protracted convalescence of the greatly debilitated patient.
P.-MTM.	Gastro-intestinal canal hyperæmic; capillary apoplexia of the brain; enlargement of the spleen. Every one of these findings may, however, be missing.	Gastro-intestinal irritation, swelling of the plaques, intestinal ulcers, erosions; infiltration of the mesenteric glands; enlarged spleen.	Finding similar to sausage-poisoning. Unfortunately, detailed descriptions of findings are missing. In certain isolated cases no anatomical changes at all occur.
THER.	Pilocarpin, sweating, washing of organism, are to be tried. The eyes must be treated by a specialist.	Calomel, salol. Treatment as in case of cholera and typhus. Prophylactically, introduction of public slaughter-houses.	Same as for sausage-poisoning. Prophylactically, official surveillance of cheese-factories and of the sale of cheese.
DETECT.	EHRENBERG was able to prove the presence of cholin, neuridin, methylamin and dimethyl-amin. ANREP claims to have obtained the one last mentioned by agitation with ether. Proceed according to Dragendorff. Special reactions for ptomatropin are missing.	It has never yet been investigated if bases are found at all, or which. Only GAERTNER found in 1888 the bacillus enteritidis, whose poison passed into boullion. Probably several poisons are present. One of these is neurin (JESERICH).	Impure tyrotoxin is said to be soluble in ether, not so when pure. It is said to exhibit the greatest similarity in reactions and behavior to di-azo-benzol. The alkaloid of LEPIERRE can be obtained by agitation with a solvent. (?)

II.—VEGETABLE ESCULENTS.

It is obvious that from improper preparation and preservation of any of the many different vegetable substances, organic poisons may develop. Flour, *e. g.*, which had been kept in a moist condition, has been found polluted by ptomaines. In Dorpat, in 1893, intensely bitter, but relatively non-poisonous substances, formed in many hundreds of pounds of flour destined for the use of soldiers. We have mentioned on page 110 that poisons may form in imperfectly dried Indian-corn. Edible mushrooms, which had rotted before drying, or which had been insufficiently dried, may form poisons of most dangerous action, the most important of which is *neurin*, behaving like muscarin. On the other hand, *e. g.*, *helvella esculenta*, mentioned on page 113 loses its poisonous properties on drying, but if it now rots, new poisonous properties of another nature are acquired. The vegetable fats become rancid on preservation, viz., they split by hydrolysis into glycerol and free fatty acids. The vegetable carbohydrates, in particular the different kinds of sugar are readily changed into acids. The vegetable albumins decompose less readily than those pertaining to animals, because they as a rule, contain less water. But in a moistened condition, they are easily decomposed by putrefaction, and by mold-formation.

II.—The Most Important Chemical Groups of Products of Metabolism.

For the purpose of giving the physician some idea of the multitude of substances belonging to this class, we affix the following table (pages 143-147), making particular mention that this enumeration is not exhaustive. Here are some of the derivations of the strange names appearing in this table:

Cadaverin, from cadaver = corpse; asparagin, from asparagus; vernin, from ver = spring; mercaptan, from mercurium captans = readily seizing the mercury; gadinin, from gadus = cod-fish; susotoxin, from sus = pig; saprin, from σαπρός = putrid; mydalein, mydatoxin and mydin, from μυδάω = to putrefy; scatol, from σκατός = excrements; leucin, from λευκός = white; creatin, from κρέας = flesh; xanthin, from ξανθός = yellow; lysatin, from λύειν = to dissolve; adenin, from ἀδήν = gland; pyridin and pyrotoxin, from πῦρ = fire, fever-heat; pyocyanin, from πύον = pus, and κύανος = blue; ichthyotoxin, from ἰχθύς = fish; spasmotoxin, from σπασμός = spasm; phlogosin, from φλόγωσις = conflagration, heat, inflammation.

TABLE OF THE MOST IMPORTANT PRODUCTS OF METABOLISM.

No.	NAME.	FORMULA.	ORIGIN, ACTION, &C.	GROUP.
1	Formic acid.	CH_2O_2	Described on page 83.	I. Group of the fatty acids.
2	Acetic acid.	$C_2H_4O_2$	Non-poisonous in form of their neutral salts.	
3	Propionic acid.	$C_3H_6O_2$	The following members are omitted; see page 79.	
4	Butyric acid.	$C_4H_8O_2$		
5	Lactic acid, or oxy-propionic acid.	$C_3H_6O_3$	The lactic acid of fermentation and the sarco-lactic acid are involved. They lessen the alkalescence of the blood.	II. Group of the oxy-acids
6	β-Oxy-butyric acid.	$C_4H_8O_3$	Occurs in the blood of diabetic persons and causes the diabetic coma.	
7	Oxalic acid.	$C_2H_2O_4$	Described on page 83. Its formation in the organism causes oxaluria.	III. Group of the oxalic acid series.
8	Succinic acid.	$C_4H_6O_4$	Forms in fermentation; see page 79.	
9	Amido-acetic acid, or glycocoll.	$C_2H_3.NH_2.O_2$	Forms, *e.g.*, in putrefaction of glue. It plays a part in the formation of hippuric acid. See p. 11. It is non-poisonous.	IVa. Group o the amido-acids.
10	Amido - propionic acid, or alanin.	$C_3H_5.NH_2.O_2$	Linked to aromatic complexes, it is contained in the albumin-molecule, and is non-poisonous	
11	β-Amido-iso-butyl-acetic acid, or leucin.	$C_6H_{11}.NH_2.O_2$	Forms in digestion and in putrefaction of albumin and glue; many kinds of leucin exist, partly dextro-rotary, partly lævo-rotary, having a tendency to crystallize readily; non-poisonous.	
12	Amido-carbonic acid, or carbaminic acid.	$CO.NH_2.OH$	Stepping-stone to urea; compare page 88 (Lime).	
13	Amido-succinic acid, or asparaginic acid.	$C_4H_5O_4.NH_2$	Forms in the intestinal canal on splitting of albumin.	
14	Carbaminic acid-amid, or carbamid, or urea.	$CO.(NH_2)_2$	Normal final product of nitrogen in albumin digestion.	IVb. Group of the amido-acid-amids.
15	Asparaginic acid-amid, or asparagin.	$C_4H_4O_3.(NH_2)_2$	One of the most important products of metabolism in plants, *e.g.*, peas, beans, asparagus.	
16	Methylamin.	CH_3NH_2	In herring brine and in (putrid) corpses.	V. Group of the primary amines or amido-bases.
17	Ethylamin.	$C_2H_5NH_2$	In putrid yeast and in putrid corpses.	
18	Propylamin.	$C_3H_7NH_2$	In putrid gelatine and in putrid corpses.	
19	Butylamin.	$C_4H_9NH_2$	In cod liver oil.	
20	Amylamin and Iso-amylamin.	$C_5H_{11}.NH_2$	In cod liver oil and in putrid yeast.	
21	Hexylamin.	$C_6H_{13}NH_2$	In cod liver oil and in putrid yeast.	
22	Glycosamin.	$C_6H_{11}.NH_2.O_5$	Product of splitting of chitin and of chondrosin.	

All of these are poisonous, corrosive, and cause spasms like ammonia.

No.	NAME.	FORMULA.	ORIGIN, ACTION, ETC.	GROUP.
23	Di-methylamin.	$(CH_3)_2.NH$	In putrid flesh and in putrid human corpses. They cause spasms and are corrosives.	VI. Group of the secondary amines or imido-bases.
24	Di-ethylamin.	$(C_2H_5)_2.NH$		
25	Tri-methylamin.	$(CH_3)_3.N$	In herring-brine, in cultures of the coma bacillus, in putrid cheese, etc., causes spasms.	VII. Group of the tertiary amines or imin-bases.
26	Tri-ethylamin.	$(C_2H_5)_3.N$	In putrid corpses alongside of tri-methylamin. Causes spasms.	
27	Ethylidene-diamin.	$CH_3.CH.(NH_2)_2$	Product of putrefaction of corpses.	VIII. Group of the di-amines.
28	Tetra-methylene-diamin or Putrescin.	$NH_2.(CH_2)_4.NH_2$	In putrid corpses, and conditionally in the excrements and urine of living persons.	
29	Penta-methylene-diamin or Cadaverin.	$NH_2.(CH_2)_5.NH_2$	In putrid corpses, putrid fish, cultures of proteus- and cholera-bacilli. The free base is corrosive, the salts are non-poisonous.	
30	Hexa-methylene-diamin.	$NH_2.(CH_2)_6NH_2$	With the two preceding ones, in the urine in cystinuria; non-poisonous in form of its salts.	
31	Cholin.	$C_5H_{15}NO_2$	In bile, chicken's egg, sprouts of germinating plants, barks and seeds etc. of many plants. Forms in the putrefying corpse from protagon and lecithin. Non-poisonous.	IX. Cholin group. On further decomposition, trimethylamin is formed.
32	Betain or Oxy-neurin or Lycin.	$C_5H_{13}NO_3$	In matrimony-vine (lycium) and many other plants in poisonous mussles (mytilus edulis) etc. Non-poisonous.	
33	Mydatoxin.	$C_6H_{13}NO_2$	Obtained from very putrid corpses; acts similarly to muscarin and kills with spasms.	
34	Neurin or Vinyl-cholin.	$C_5H_{13}NO$	It is found in corpses on the fifth or sixth day, as well as in putrid helvella and acts like muscarin.	
35	Neuridin.	$C_5H_{14}N_2$	One of the most frequent products of putrefaction. Non-poisonous.	
36	Cadaver-muscarin.	$C_5H_{15}NO_3$	Similar to, or perhaps identical with the amanita-muscarin. Compare page 137.	
37	Guanidin.	CH_5N_3	One of the splitting-products of albumen, in germs of vetch or tare (vicia). Causes epilepsy by irritating the cortex of the brain. See page 60.	X. Guanidin group.
38	Methyl-guanidin.	$CH_4.N_3.CH_3$	In putrid flesh; acts as the preceding one.	
39	Methyl-guanidin acetic acid or Creatin or Methyl-glycocyamin.	$C_3H_6N_3O_2.CH_3$	Contained in muscles, nerves, blood, testicles, etc., of living persons and acts stimulatingly. Creatin is creatinin minus one molecule of water.	
40	Propyl glycocyamin.	$C_3H_6N_3O_2.C_3H_7$	In the urine in case of quinsy, very poisonous.	

No.	NAME.	FORMULA.	ORIGIN, ACTION, ETC.	GROUP.
41	Glycocyamidin.	$C_3H_6N_3O$	In the urine in case of measles as well as in pure cultures of whooping-cough bacilli.	X. Guanidin group.
42	Lysatin.	$C_6H_{13}N_3O_2$	Isomeric or identical with propyl-glycol-cyamin; prepared artificially from casein. Lysatinin ie lysatin minus one molecule of water.	
43	Arginin.	$C_6H_{14}N_4O_2$	Normal constituent of sprouting plants. Action unknown.	
44	Adenyl-imin or Adenin.	$C_5H_4N_4.NH.$	Normal constituent of thymus, lymphatic glands, pancreas, etc., but also of tea-leaves. Action unknown.	XI. Adenin group.
45	Adenyloxid or Hypoxanthin.	$C_5H_4N_4O$	In the sprouts of maple, plane-tree (platanus), lupine, etc., but also in spleen, pancreas, liver, muscle, marrow, blood and urine of normal animals. Amount increased in leucæmia and acute yellow atrophy of liver.	
46	Adenyldioxid or Xanthin.	$C_5H_4N_4O_2$	In pancreas, brain, liver, spleen, thymus and urine. It sometimes forms calculi.	
47	Methyl-adenyl-dioxid or Heteroxanthin.	$C_5H_3CH_3N_4O_2$	Found in urine, etc., besides xanthin.	
48	Dimethyl-adenyl-dioxid.	$C_5H_2.(CH_3)_2N_4O_2$	Three isomere exist: paraxanthin of urine, theophyllin of tea-leaves, and theobromin of cocoa-beans.	
	Trimethyl-adenyl-dioxid or Coffein.	$C_5H.(CH_3)_3N_4O_2$	In coffee it acts in an irritating manner, like the other xanthin-substances. See page 122.	
49	Adenyl-trioxid or Uric acid.	$C_5H_4N_4O_3$	Is the cause of gout, arthritis urica and nephritis urica. The latter also occurs in case of saturnism. See page 98.	
50	Adenyl-imin-oxid or Guanin.	$C_6H_4N_4NH.O$	In liver, pancreas, guano, excrements of spiders, and in the skin of various reptiles and fish. In hogs it causes the so-called guanin-gout.	
51	Vernin.	$C_{16}H_{20}N_8O_8$	In pumpkin-seeds, vetches, red clover, in clavicepe purpurea (ergot). On boiling with HCl, guanin forms.	
52	Episarcin.	$(C_4H_6N_3O)x$	According to BALKE, in normal urine.	
53	Pyridin.	C_5H_5N	Nucleus of many alkaloids. Forme on destructive distillation of organic, nitrogenized substances. Acts, like the whole series, in a manner suggestive of nicotin. One gulp killed a workman.	XII. Pyridin group.
54	Methyl-pyridin or Picolin.	C_6H_7N $C_5H_4.CH_3.N$	In tobacco-smoke, causing part of its toxic actions.	
55	Dimethyl-pyridin or Lutidin.	C_7H_9N= $C_5H_3.(CH_3)_2N$	So far it is of no pharmacological interest. Found in bone-tar and in Dippel's oil. Poisonous.	

No.	NAME.	FORMULA.	ORIGIN, ACTION, ETC.	GROUP.
56	Collidin.	$C_8H_{11}N$	Forms in putrefaction of gelatine (base of NENCKI) and in putrefaction of sea-polypus. Poisonous.	XII. Pyridin group.
57	Parvolin.	$C_9H_{13}N$	Found in putrefaction of flesh (base of GAUTIER and ETARD). Poisonous.	
58	Coridin.	$C_{10}H_{15}N$	In the putrefaction of various animal substances (base of GUARESCHI and MOSSO). Poisonous.	
59	Hydro-coridin.	$C_{10}H_{17}N$	Product of metabolism of bacterium Allii.	
60	Quinolin.	C_9H_7N	Nucleus of many alkaloids, in coal-tar. Protoplasmic poison; kills after a short time of irritation, by paralysis of respiratory-centre.	XIII. Quinolin. group.
61	Methyl - chinolin or Lepidin.	$C_9H_6.CH_6.N$	Found *e. g.*, in bone-oil and in coal-tar. Poisonous.	
62	Dimethyl-chinolin or Cryptidin.	$C_9H_5.(CH_3)_2N$	Found besides Lepidin, *e. g.*, in bone-oil, poisonous. Cairin and thallin are derivatives of this group.	
63	Hydroxyl-benzene or Phenol.	$C_6H_5.OH$	Discussed on page 83, under the name of carbolic acid, forms as a product of putrefaction in the normal intestinal bacterium - digestion and reappears in the urine as sulfuric acid - phenol - ester. Compare page 10.	XIV. Group of the aromatic series.
64	Methyl - phenol or Cresol.	$C_6H_4.CH_3.OH$	Mainly one of the three possible isomeric cresols, para-cresol, is invariably found among the products of the process of intestinal putrefaction. It appears in the urine as sulfuric acid para-cresol ester. The cresols are less poisonous than phenol.	
65	Di-hydroxyl-benzene	$C_6H_4(OH)_2$	Of the three isomeric di-hydroxyl benzenes, mainly the ortho-compound, catechol, is always formed in the intestine, and reappears in human urine as mono- and di-sulfuric acid ester. The meta-compound, resorcinol, is freely used as a medicine. The para - compound, quinol, forms in the organism in consequence of partaking of arbutin or of bearberry-leaves (Fol. Uvæ Urei).	
66	Phenyl-acetic acid.	$C_6H_5.CH_2.COOH$	Forms in putrefaction of albumin, poisonous.	
67	Para-oxyphenyl-acetic-acid.	$C_6H_4.OH.CH_2.COOH$	Forms in putrefaction of albumin and passes in an unchanged state into the urine. Poisonous.	
68	Phenyl - propionic - acid or Hydro - cinnamic acid.	$C_6H_5.C_2H_4.COOH$	Forms on putrefaction and reappears in the urine as hippuric acid. Poisonous.	
69	Phenyl - amido - propionic acid or Phenyl-alanin.	$C_6H_5.C_2H_3.NH_2.COOH$	Splitting-product of albumin, but it is further decomposed in the organism. Poisonous.	

No.	NAME.	FORMULA.	ORIGIN, ACTION, ETC.	GROUP.
70	Oxy-phenyl-alanin or Tyrosin.	$C_6H_4.OH.C_2H_3.NH_2.COOH$	One of the most ordinary products of putrefaction of albumin. In the normal organism it is further oxidized (burned), but not so in the case of phosphorus-poisoning and alcaptonuria. But slightly poisonous. It is also found in the tuber of stachys tubifera.	XIV. Group of the aromatic series.
71	Methyl-tyrosin or Andirin.	$C_9H_{10}.CH_3.NO_3$	It is found in the genuine geoffroya-bark (as surinamin or geoffroyin), in ratanhy-extract (as ratanhin), and in the resin of pereira spectabilis (as angelin). But slightly poisonous.	
72	Indol.	C_8H_7N	Forms in the intestine in putrefaction of albumin, and reappears in the urine as a salt of indican or sulfuric acid indoxyl ester. Many pathogenous bacteria will produce it even outside of the intestine. It is contained in the wood of celtis (hackberry or nettle-tree). Poisonous.	
73	Methyl-indol or Scatol.	$C_8H_6.CH_3N$	Forms in the intestine in putrefaction of albumin and reappears in the urine as a salt of sulfuric acid-scatoxyl-ester. Scatol is also formed on distillation of strychnin with lime. Poisonous.	
74	Hydrogen Sulfid.	H_2S	Found in every putrefaction going on with exclusion of air, consequently in the intestine. Poisonous. See page 118.	XV. Group of the albumin derivatives containing sulfur.
75	Methyl-mercaptane.	$CH_3.SH$	Similar to the preceding. Poisonous.	
76	Amido-ethyl-sulfonic-acid or Taurin.	$C_2H_4.NH_2.SO_2OH$	Forms on putrefaction of taurocholic acid in the intestine. Taken internally, it reappears in urine as a substituted urea. Non-poisonous.	
77	Potassium sulfo cyanate or Pot. thyocyanate or Pot. rhodanate.	$CNSK$	Forms on complete decomposition of adenin, and is found in saliva, urine and milk of man and various animals. Even ordinary mold, aspergillus niger will produce it. It accentuates the reflexes and causes spasms.	
78	Dithio-diamido-lactic acid or Cystin.	$C_3H_{12}N_2S_2O_4$	Forms in pancreatic digestion; its increased formation, due to specific bacteria, causes cystinuria. It is non-poisonous.	
79	Iso-cyan-methyl.	$CH_3.NC$	Sparingly contained in toad-poison. Poisonous.	XVI. Group of the iso-cyan-compounds.
80	Iso-cyan-ethyl.	$C_2H_5.NC$	Splitting product of the so-called pseudo-lecithin, which is contained in the triton-poison. Poisonous.	
81	Iso-cyan-amyl.	$C_5H_{11}.NC$	In the poison of salamandra maculata. Poisonous.	
82	Iso-cyan-acetic acid.	$CH_2.NC.COOH$	Abundantly contained in toad-poison. Poisonous.	
83	Iso-cyan-propionic acid.	$C_2H_4.NC.COOH$	In the poison of tritons. Poisonous.	

No.	NAME.	FORMULA.	ORIGIN, ACTION, ETC.	GROUP.
84	Tetanotoxin.	$C_5H_{11}N$	Isolated from cultures of the tetanus bacillus, with other substances. Both are poisonous.	XVII. Group of bases known by formulæ, but not by structure.
85	Tetanin.	$C_{14}H_{30}N_2O_4$		
86	Saprin.	$C_5H_{14}N_2$	Found in human corpses; non-poisonous.	
87	Mydin.	$C_8H_{11}NO$	Forms under the influence of typhus bacilli, from the albumin of blood; non-poisonous.	
88	Gadinin.	$C_7H_{17}NO_2$	Isolated from putrid glue and putrid codfish. Causes ascending paralysis.	
89	Typhotoxin.	$C_7H_{17}NO_2$	In typhus-cultures; poisonous.	
90	Nameless base.	$C_6H_{13}NO_2$	In tetanus-cultures; non-poisonous.	
91	Nameless base.	$C_7H_{17}NO_2$	In putrid flesh; poisonous.	
92	Nameless base.	$C_3H_8O_2$	Forms in cadavers; poisonous.	
93	Eczemin.	$C_7H_{16}NO$	In the urine of eczema patients; causes inflammation and fever.	
94	Malleus base (perhaps identic with No. 127)	$C_{15}H_{10}N_2O_6$	Isolated from cultures of bacillus mallei and from the urine of glandere patients; poisonous.	
95	Pneumonia base.	$C_{20}H_{26}N_2O_9$	In the urine of pneumonic patients; poisonous.	
96	Influenza base.	$C_9H_9NO_4$	In the urine of influenza patients; kills with high fever.	
97	Erysipelin.	$C_{11}H_{13}NO_3$	In the urine of erysipelas patients; causes violent fever.	
98	Convulsivin.	$C_6H_{19}NO_2$	In the urine of whooping-cough patients, as well as in cultures of whooping-cough bacilli.	
99	Pyæmia base.	$C_{22}H_{19}NO_2$	From the urine of a woman suffering from puerperal fever.	
100	Epilepsy base.	$C_{12}H_{16}N_5O_7$	Found in the urine in epilepsy.	
101	Anthraxin.	$C_3H_6N_2$	Produced from anthrax cultures.	
102	Morbilli- or Rubeola-base.	$C_3H_6N_3O$	In the urine of patients suffering from measles.	
103	Pyocyanin.	$C_{14}H_{14}NO_2$	Coloring matter of blue pus; non-poisonous.	
104	Sardinin.	$C_{11}H_{11}NO_2$	Forms in putrid sardines; causes death with vomiting and purging.	
105	Scombrin.	$C_{17}H_{38}N_4$	In putrid mackerel.	
106	Lepierre's base.	$C_{16}H_{24}N_2O_4$	In over-ripe cheese.	
107	Asellin.	$C_{25}H_{92}N_4$	In the dark cod-liver oil; the latter base not inert, but diaphoretic and diuretic.	
108	Morrhuin.	$C_{19}H_{27}N_3$		
109	Nameless bases.	$C_{14}H_{20}N_2O_4$	Found in putrid fibrin.	
110		$C_5H_{11}NO_2$	Found in putrid fibrin and putrid flesh.	
111	Pluviatilin.	$C_9H_{21}N_2O_5$	Produced on peptone gelatine by bacillus fluviatilis.	
112	Tetragenin.	$C_5H_6NO_2$	Produced by Micrococcus tetragenes, frequent in phthisical sputa; poisonous.	

No.	NAME.	ORIGIN, ACTION, ETC.	GROUP.
113	Pellagrocein of LOMBROSO.	Is said to form in putrefaction of Indian-corn and to cause pellagra.	XVIII. Group of substances quite insufficiently studied, of which neither the formula nor the structure are known.
114	Pyrotoxin of CENTANNI.	Is said to cause infection-fever.	
115	Sepsin or base of SCHMIEDEBERG and BERGMANN.	Obained from putrid yeast. Is said to produce the symptoms of septicæmia.	
116	Ptomatropin or KERNER'S base of sausage-poison.	Acting similarly to atropin; base formed eventually in putrid fish and putrid sausage.	
117	Ptomacurarin or base of HARKAWY.	Base acting similarly to curare, repeatedly found in putrid corpses.	
118	Peptotoxin of BRIEGER.	Forms when albumin is peptonized.	
119	Ichthyotoxin of MOSSO.	Poisonous substance of the blood of eel.	
120	Tyrotoxin of VAUGHAN.	Sometimes found in cheese; very poisonous.	
121	Mydalein of BRIEGER.	Obtained from human corpses, most likely a diamin.	
122	Spasmotoxin of BRIEGER.	From tetanus-cultures; poisonous.	
123	Phlogosin of LEBER.	Obtained from cultures of staphylococcus aureus; does not contain nitrogen; causes inflammation.	
124	Sucholotoxin, sucholoalbumin, susotoxin.	They form in hog-cholera or hog-pest.	
125	Tuberculin of KOCH.	Mixture of substances from cultures of bacilli of tuberculosis; poisonous.	
126	Tuberculoidin of KLEBS.	Purified tuberculin.	
127	Morvin of BABES.	Enzyme formed in bouillon of horseflesh from bacilli of glanders; causes fever, nephritis and marasm.	
128	Choleratoxopepton of SCHOLL.	Formed, by cholera-spirilli, from living protoplasm with exclusion of oxygen; causes paralyses and hyperæmia of the kidneys and the small intestine.	
129	Toxopepton of PETRI.	Formed by the same spirilli on dead albumin.	
130	Diphtheria-toxalbumin.	Produced by the baccilus of diphtheria; very poisonous.	
131	Gonococcus-toxalbumin.	Generated from Neisser's coccus; produces orchitis.	
132	Tetanus-antitoxin.	Acts as a cure of tetanus-infection.	
133	Diphtheria-antitoxin.	Acts as a remedy in case of diphtheria.	

Concerning toxalbumins derived from plants, we have mentioned *ricin* on page 105 and *phallin* on page 112; and of those from animals, *snake-poison* and *spider-poison* on page 107. Lack of space prevents the enumeration of all enzymes of plants and animals; according to the latest researches, they are by no means all of a poisonous character.

III.—The Autintoxications (Auto-intoxications).

Viewed from the present scientific standpoint, the appearance in the living organism of any one of the substances on the preceding table, must be called an autintoxication, viz., an intoxication produced within the organism itself. However, the clinical conception of autintoxication existed even at a time when we had no clear idea of ptomaines. This may serve as an argument for maintaining the designation, for the time being, in case of the appearance of the following products of metabolism. The six most important of those disturbances of metabolism are represented in table, pp. 152-153.

Now for a short consideration of the other autintoxications. *Mucinœmia* we call a grave, generally diseased condition accompanying an increase of the amount of *mucin* in the body. It offers the clinical aspect of tetanus, while at the same time the thyroid gland is not infrequently diseased or absent. Injections of thyroid gland extract may be useful. A similar form of disease is the *gastrotetany*, in which, however, abnormal products of digestion in the stomach produce the tetanic symptoms. *Lactacetœmia* we call the appearance of *lactic acid* in the blood and urine. This will occur occasionally in all cases in which the processes of oxidation in the organism are disturbed; consequently we shall observe it in case of intoxications by *hydrocyanic acid, carbon-monoxid, sodium oxalate phosphorus, arsenic*, etc. As diseases belonging under this heading, we mention trichinosis, acute yellow atrophy of liver, cirrhosis of liver, rachitis, leucocythæmia, osteomalacia, as well as agonia. In these cases we partly have to do with *lactic acid of fermentation* and with *sarco-lactic acid* (*para-lactic acid*). NENCKI found a bacillus which produces the latter acid in abundant quantity. The appearance of lactic acid with the exclusion of micro-organisms must be considered as a product of the action of a tissue-enzyme, contained in all organs, upon the carbohydrates of the body. As long as sufficient oxygen is present, this splitting at once goes further, if not in the entire

body, at least in the muscles, viz., carbon-dioxid and water will be formed. The bacterial formation of lactic acid may take place in the presence or in absence of oxygen. Lactic acid of fermentation is optically inactive; sarco-lactic acid is dextro-rotary; typhus-bacilli and different other microbes, however, form a *lævo-lactic acid.* The zinc salt of the latter is dextro-rotary. Administration of alkalies and of oxygen lessens all forms of autintoxication due to lactic acid. The appearance of *cystin* in the urine, we call *cystinuria,* which may run its course accompanied by formation of calculi. It is claimed that its presence is due to the existence of specific bacteria of the small intestine, which directly form cystin. Graver symptoms may be absent. At the same time *cadaverinuria* and *putrescinuria* may exist. We frequently find complications of *glycosuria* with *acetonæmia, acetonuria, diaceturia* (viz., the appearance of *acet-acetic acid* inthe urine). We call *lipacidæmia* and *lipaciduria* the appearance of lower fatty acids (*formic-, acetic-, propionic-,* and *butyric acid*) in the blood and urine, a condition which may obtain in diabetic patients. The appearance of *formic acid* is known by the special name of *formacidæmia* or *formaciduria.* The formation of considerable quantities of *oxalic acid,* in case the nutriments are free from it, we designate *oxaluria.* According to English opinions, this disease coexists eventually with a melancholic state of mind. For oxaluria produced by administration of oxalic acid and its salts, see p. 83. A state in which *albumoses* appear in the urine, and which should be called *albumosuria,* is erroneously styled *peptonuria. Toxalbuminuria* is the existence of poisonous albumin-substances in the urine.

It can hardly be doubtful that in the very near future all of the following diseases will be treated of under the heading of autintoxication:—1. All gland-diseases which depend upon decay of the parenchyma of the glands, viz., *thyroid-gland, pancreas-gland, kidney, liver,* etc.; 2, all diseases caused by microbes, as *malaria, measles, scarlet fever, variola, typhus, influenza, cholera, tuberculosis;* and 3, some other diseases, which, according to the present state of our knowledge, are not yet positively known to be produced by microbes, as *leucæmia, syphilis, carcinosis.* The therapeutic agents involved in these cases will belong in great part to the extended subject of serum-therapy.

	AMMONIÆMIA.	URÆMIA.	HYDROTHIONURIA.
ÆTIO.	Depends upon transformation of urea into ammonium carbonate, and is superadded to diseases which increase the excretion of urea through the kidneys, or which favor immgration of bacteria into the bladder, ureters, and pelvis of kidneys. In animals whose liver has been artificially cut out of the circulation an autintoxication by ammonium carbaminate takes place.	Depends upon insufficient secretion of the normal constituents of urine through the kidney, and may be superadded to each case of nephritis. However, it will also occur, without albuminuria, as so-called small uræmia. It is as yet unknown which constituent of the urine causes the disease. Erroneously, it seems, the potassium salts have frequently been held responsible for it.	H_2S may be formed: (1) In the urinary passages by microbes from the so-called neutral sulfur of the urine; or (2) in the intestine from food containing sulfur; or (3) at divers parts of the body by certain bacteria (proteus, pus-fungii). The H_2S formed is partly secreted by the lungs, partly it will be oxidized in the organs to form sulfate, which appears as such in the urine.
SYMPT.	If the normal secretion of the urine from the blood is impeded, it may take place through the mucous membrane of the intestine, where the microbes present will at once cause the formation of ammonium carbonate. This will give rise to violent inflammation of the intestine and, after resorption of the salt, all symptoms of ammonia poisoning as described on page 88. In spite of contrary assertions by MUSCULUS, there is no enzyme existing, at least not one allowing of isolation, which would form ammonia in sterile urea-solutions.	Buzzing in ears, deafness, dizziness, formication, itching of skin, sensitiveness of legs to cold, nocturnal cramps in calves, asthma, vesical tenesmus in presence of normal quantity and apparently normal composition of the urine, epistaxis matutina, sudden starts on falling asleep, temporal arteries hard and tortuous, galloprhythm of the heart, œdema. In grave cases, amaurosis, delirium, unconsciousness, mydriasis, general convulsions; but with dogs merely narcosis.	Striking fetid odor from mouth in spite of most scrupulous hygiene thereof. The urine stinks on voiding. (Difference from H_2S formation in unclean chamber-pots.) Flatus of great stench may issue in case the source of H_2S formation lies in the intestine. Nausea, dyspnœa only in grave cases. On administration of sulfur as such, an ample formation of H_2S in the intestine is caused without being detrimental. Merely diarrhœa will set in, with an odor strongly suggestive of rotten eggs.
THER.	In case of NH_3 formation in the urinary passages, arbutin, salol, mineral acids internally and antiseptic lavations of bladder. In case of retention of urea in the blood, diaphoretics and diuretics. Antiseptics into the intestine are useless. Acid lemonades, *e.g.*, from HCl.	Pilocarpin, Diaphoretic baths, washing of organism; hypodermic cataclysm; leeches, hæmaxis, oxygen inhalations, evacuation of intestine; alkalies. In case of convulsions, chloroform inhalations.	Remove the microbes or paralyze their action by suitable antiseptics. Purgatives. Salol, calomel. Lavations of bladder, arbutin. Injection of salol iodoform into possible cavities of abscesses.
P.-MTM.	The finding may be similar to that described for NH_3 on page 89. Not rarely formation of pseudo-membrane in the ileum and colon. Analogous changes in the urinary passages. All organs and cavities of the body are pervaded by the odor of ammonia. The blood may be slow to coagulate.	With man the alkalescence of the blood is reduced; in the dog it is not. Anatomical findings may be missing. Divers affections of the skin: erythema, papules, pustules, even pemphigus. In case inflammation of the intestine and gastritis obtain, complication with ammoniæmia is present. Then the alkalescence of the blood will be increased.	The finding may be entirely negative; eventually some sections of the body will emell of rotten eggs. Only in grave cases, and never while life lasts, will sulf-met-hæmoglobin be formed. But after death it will take place, surrounding the centre of disturbance, and this, or even the whole corpse, will turn greenish.
DETECT.	Compare page 16. For the composition of Nessler's reagent, see pages 89. and 169. Also an excellent reagent for ammonia is the nose and moist red litmus paper.	Up to date we do not know what to look for. I should direct my main attention to ptomaines.	Compare page 119. H_2S may be easily separated from urine and the organs by acid distillation. However, this does not prove its intravital formation. There are not only bacteria, but even enzymes existing, capable of producing it at ordinary temperature.

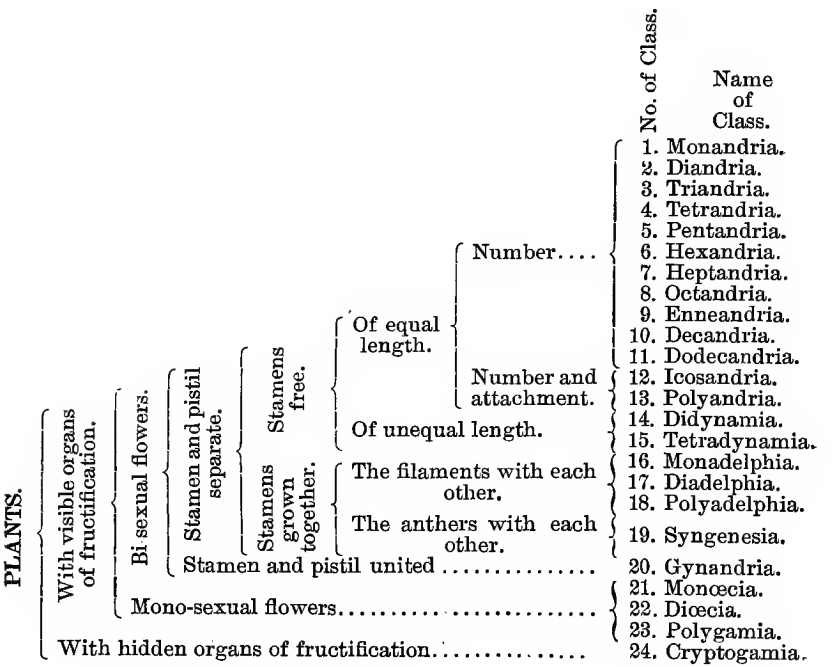
GLYCOSURIA.	URATÆMIA.	ALCAPTONURIA.
We have the following kinds of glycosuria: physiological, alimentary, constitutional, infectious, traumatic and toxic. Its most important form is the genuine diabetes mellitus, which depends upon hereditary tendency. It must of necessity depend upon the splitting of glucose from albumin—so-called grave form,—since frequently it does not disappear on administration of food free from carbohydrates. Generally we find nervous diseases common in the family.	Increased occurrence of uric acid in the body (gout) may depend upon hereditary tendency. But it occurs also (1) in case of overabundance of food and simultaneous partaking of strong alcoholic beverages; (2) in case of chronic lead-poisoning. Gout is of frequent occurrence with poultry; very rare with other animals. The pig has guanin-gout instead. Concerning the relations of uric acid to guanin, see page 145.	Specific bacteria of the small intestine incessantly produce uroleucinic acid, viz., tri-oxyphenyl-propionic acid, or homogentisinic acid, which is dioxiphenyl-acetic acid. Neither acid is detrimental to the healthy man and animal. They oxidize (burn) them with destruction of the aromatic nucleus.
Besides glucose, the blood and the urine may contain a lævorotary carbohydrate (Leo), β-oxy-butyric acid, aceton, acet-acetic acid, etc. Great thirst, frequent urgency to micturate, increased quantity of urine, periods of unconsciousness (diabetic coma). Peculiar odor from mouth, rapid decay of teeth, the secretion of the mouth rapidly turns acid; decay of sexual power; great craving for sweets; furunculosis. In regard to the most frequent complications, see page 151. Even oxaluria and uratæmia may occur. Frequently tendency to develop adipose tissue.	Urine sometimes rich in urates, sometimes poor. Painful attacks due to deposition of uric acid (tophi) in the joints (big toe), cartilage of ear, etc. Dyspeptic and neuralgic troubles. Urine remarkably acid, concentrated, of high specific gravity. Potassium sulfo cyanate of urine and saliva diminished. Complications with diabetes not uncommon. Tendency to neurasthenia, sciatica, hemicrania, lumbago, cramps in calves, skin feels cold, early atheroma of vessels.	The urine has a strong reducing action; upon addition of alkalies it absorbs oxygen and turns brown, hence the name alcapton (from alkali and καπτειν, to swallow with avidity). Diabetes, tuberculosis or perfect health may co-exist. Pure meat-diet increases the quantity of alcapton. Alcaptonuria is tolerated for years; it molests the patient so little, that in a certain case it was merely accidentally discovered.
Carlsbad-waters; deprivation of carbohydrates from food; in case of comatose attacks, alkaline injections into veins. Piperazin, tetra-ethyl-ammonium hydrate. In light cases, increased exercise. Most scrupulous hygiene of skin and mouth.	Piperazin carbonate, tetra-ethyl-ammonium hydrate, alkaline waters, sodium- and lithium-carbonate. Food should be poor in albumin. Prohibition of alcohol. Lithotomy. Partaking of lemons, sour apples and acid lemonades must be prohibited.	So far all means have been without success. It would seem that intestinal disinfectants and alkalies would be useful. But before knowing the specific microbes of this disease we can scarcely understand the therapy.
The finding may be entirely negative; or we find decay of the pancreas, glycogenous degeneration of liver, kidney, etc. Tumors in the brain. All organs and cavities of the body may smell of acetone. Urine will frequently turn a deep wine-red with ferric chlorid. Furunculosis diffusa. Corpse in some cases remarkably lean, in others very fat.	Grating masses of urates imbedded in kidneys, joints, cartilage of ears. Urate-calculi in pelvis of kidneys and bladder. These calculi appear to less extent in the cortex of the kidneys than in the parenchyma, particularly in and around the ducts. The tophaceous masses are imbedded in the connective tissue of all the organs involved, and frequently cause necrosis.	A characteristic finding does not exist, since in all cases accessory diseases have eventually led to death. The gist of the disease might be approached by careful bacteriological study of the cases.
The urine contains oxy-butyric acid, lower fatty acids, acet-acetic acid, sugar, increased amount of ammonia. In regard to the reagents for the detection of sugar, see table at the end of this book.	Detection of uric acid by means of the murexid-reaction; evaporate uric acid or its salts with HNO_3 until dryness; the yellowish-red residue will be colored purple-red by ammonia, blue-violet by KOH.	The alkali-test, mentioned above, and the strongly reducing action suffice. In contrast to glycosuria, the urine is not fermentable.

B.—Review of Toxicologically Interesting Orders and Genera of Plants Indigenous to Germany.

To my knowledge there is no book within easy reach of the physician that enumerates those orders of plants, growing in Germany, which are of toxicological importance. On page 155 we give a review, in the sequence of the natural system, of all orders growing in Germany. Those of particular pharmacological interest are italicised. We see from this review that poisons are to be found in the lowest as well as in the highest families of the system. It is the same with LINNÉ'S system (for which we give the key), where we find the poisons scattered all through the classes most varyingly.

The physician should at least know as much of the natural system, and of LINNÉ'S artificial system, as to enable him to determine a plant with the aid of a botanical work.

KEY TO THE SEXUAL SYSTEM OF LINNÉ.



Phænogamus plants.

Angiospermous plants.

Dicotyledonous plants.

Flowers perfect.

- Leaves of corolla separated from each other, hypogynous: *Ranunculaceæ*, Crassulaceæ, Rosaceæ, Berberidaceæ, *Papilionaceæ*, *Papaveraceæ*, Fumariaceæ, Cruciferæ, Nymphæaceæ, Resedaceæ, Violaceæ, Droseraceæ, Cistaceæ, Tamariscaceæ, Silenaceæ, Alsinaceæ, Paronichyaceæ, Portulaceæ, *Amygdalaceæ*, Terebinthaceæ, Rhamnaceæ, Lythraceæ, Tyliaceæ, Malvaceæ, Geraniaceæ, Linaceæ, *Oxalidaceæ*, Balsaminaceæ, Hypericaceæ, Elatinaceæ, Hypopityaceæ, Empetraceæ, Aceraceæ, Hippocastanaceæ, Ampelidaceæ, Calastraceæ. *Rutaceæ*.
- Leaves of corolla separated from each other epigynous: Grossulariaceæ, Loranthaceæ, Saxifragaceæ, Onagraceæ, Umbelliferæ, Hallorrhagidaceæ, Alariaceæ, Cornaceæ, Pomariæ, Philadelphaceæ.
- Corolla united into one piece, epigynous: *Cucurbitaceæ*, Campanulaceæ, *Lobeliaceæ*, Siphomandraceæ, Ericaceæ, *Rhodoraceæ*, *Compositæ*, *Rubiaceæ*, Caprifoliaceæ, *Valerianaceæ*, Dipsaceæ.
- Corolla united into one piece, hypogynous: *Boraginaceæ*, *Labiatæ*, Plumbaginaceæ, Globulariaceæ, Plantaginaceæ, Primulaceæ, Lentibulariaceæ. *Scrophulariceæ*, Verbenaceæ, Polygalaceæ, Oleaceæ, Aquifoliaceæ, *Convolvulaceæ*, Polemoniaceæ, *Solanaceæ*, *Gentianaceæ*, *Apocynaceæ*, *Asclepiadaceæ*.

Flowers imperfect, involucre simple or missing: Aristolochiaceæ, Hippuridaceæ, Santalaceæ, Callitrichaceæ, *Euphorbiaceæ*, Acaliphaceæ, *Buxaceæ*, *Polygonaceæ*, Urticaceæ, *Cannabaceæ*, Moraceæ, Ulmaceæ, Thymelæaceæ, Elæagnaceæ, Scleranthaceæ, *Chenopodiaceæ*, Amaranthaceæ, Ceratophyllaceæ, Platanaceæ, Inglandaceæ, Cupuliferæ, Betulaceæ, *Salicaceæ*, Myraceæ.

Monocotyledonous plants.

- Several ovaries: Alismaceæ, Butomaceæ, Juncaginaceæ, *Colchicaceæ*, Potamieæ.
- One ovary, hypogynous: *Orchidaceæ*, Hydrocharitaceæ, Iridaceæ, *Dioscoreaceæ*, Amaryllidaceæ.
- One ovary, epigynous: *Liliaceæ*, Juncaceæ, *Araceæ*, Typhaceæ, Naiadaceæ, Lemnaceæ, Cyperaceæ, Gramineæ.

Gymnospermous plants. Coniferæ.

Cryptogamous plants.

- Cormophytes (acrogens): *Equisetaceæ*, Marsiliaceæ, Salviniaceæ, *Lycopodiaceæ*, Ophioglosseæ, Osmundaceæ, Hymenophllaceæ, Polypodiaceæ.
- Thallophytes: Characeæ, *Muscineæ*, Algæ (Lichenes), Enzymes (splitting fungi).

The following table contains those orders with which the toxicologically cultured physician should be familiar. Lack of space forbids mention of the species. We refer to the particular works on botany that we have ourselves followed in regard to nomenclature, including AUG. GARCKE'S "Flora von Deutschland" (17. Aufl. Berlin 1893). Naturally, the choice of orders is more or less arbitrary, as we do not find in the pertaining literature that it really has been proven, for a large number of them, that they contain pharmacologically active agents. However, we are partly supported in this case by personal orientating experiments. Any improvement upon this table will be welcomed by us.

No.	ORDER.	GENUS.
1	Ranunculaceæ.	Clematis, Thalictrum, Hepatica, Pulsatilla, Anemone, Adonis, Ranunculus, Ficaria, Caltha, Trollius, Eranthis, Helleborus, Isopyrum, Nigella, Aquilegia, Delphinium, Aconitum, Actæa, Cimicifuga.
2	Papaveraceæ.	Papaver, Glaucium, Chelidonium.
3	Fumariaceæ.	Hypecoum, Corydalis, Fumaria.
4	Cruciferæ.	Cheiranthus, Nasturtium, Barbaræa, Turitis, Arabis, Sisymbrium, Alliaria, Erysimum, Brassica, Sinapis, Alyssum, Cochlearia, Camelina, Thlaspi, Iberis, Lepidium, Capsella, Bunias, Raphanistrum, Raphanus.
5	Violaceæ.	Viola.
6	Polygalaceæ.	Polygala.
7	Silenaceæ.	Gypsophila, Dianthus, Saponaria, Vaccaria, Cucubalus, Silene, Coronaria, Melandryum, Agrostemma.
8	Linaceæ.	Linum.
9	Hippocastanaceæ.	Æsculus.
10	Geraniaceæ.	Geranium, Erodium.
11	Oxalidaceæ.	Oxalis.

No.	ORDER.	GENUS.
12	Rutaceæ.	Ruta, Dictamnus.
13	Celastraceæ.	Evonymus.
14	Rhamnaceæ.	Rhamnus, Frangula.
15	Terebinthaceæ.	Rhus.
16	Papilionaceæ.	Ulex, Sarothamnus, Genista, Cytisus, Lupinus, Ononis, Melilotus, Robinia, Coronilla, Lathyrus.
17	Amygdalaceæ.	Amygdalus, Prunus.
18	Rosaceæ.	Spiræa, Aruncus, Ulmaria, Geum, Rubus, Fragaria, Potentilla, Alchemilla, Rosa.
19	Cucurbitaceæ.	Bryonia.
20	Paronychiaceæ.	Herniaria.
21	Crassulaceæ.	Sedum, Sempervivum.
22	Umbelliferæ.	Cicuta, Apium, Petroselinum, Carum, Pimpinella, Sium, Œnanthe, Æthusa, Fœniculum, Levisticum, Archangelica, Imperatoria, Conium, Coriandrum.
23	Araliaceæ.	Hedera.
24	Loranthaceæ.	Viscum, Loranthus.
25	Caprifoliaceæ.	Sambucus, Viburnum, Lonicera.
26	Rubiaceæ.	Asperula.
27	Valerianaceæ.	Valeriana, Valerianella.
28	Compositæ.	Artimisia, Achillea, Anacyclus, Matricaria, Tanacetum, Arnica, Cnicus, Cichorium, Leontodon, Taraxacum, Lactuca, Picris.
29	Lobeliaceæ.	Lobelia.
30	Siphonandraceæ.	Vaccinium, Arctostaphylus, Andromeda, Cassandra.
31	Ericaceæ.	Erica, Calluna.

No.	ORDER.	GENUS.
32	Rhodoraceæ.	Ledum, Azalea, Rhododendron.
33	Aquifoliaceæ.	Ilex.
34	Asclepiadaceæ.	Vincetoxicum.
35	Apocynaceæ.	Vinca.
36	Gentianaceæ.	Menyanthes, Gentiana, Erythræa.
37	Convolvulaceæ.	Convolvulus, Cuscuta.
38	Boraginaceæ.	Heliotropum, Cynoglossum, Anchusa, Symphytum, Echium, Lithospermum.
39	Solanaceæ.	Lycium, Solanum, Physalis, Nicandra, Scopolia, Atropa, Hyoscyamus, Nicotiana, Datura.
40	Scrophulariaceæ.	Verbascum, Antirrhinum, Gratiola, Digitalis, Melampyrum, Alectorolophus, Orobanche.
41	Labiatæ.	Lavandula, Mentha, Salvia, Origanum, Thymus, Satureja, Melissa, Hyssopus.
42	Primulaceæ.	Anagallis, Primula, Cyclamen.
43	Globulariaceæ.	Globularia.
44	Plumbaginaceæ.	Armeria.
45	Chenopodiaceæ.	Chenopodium.
46	Polygonaceæ.	Rumex, Polygonum.
47	Thymelæaceæ.	Thymelæa, Daphne.
48	Aristolochiaceæ.	Aristolochia, Asarum.
49	Euphorbiaceæ.	Tithymalus.
50	Buxaceæ.	Buxus.
51	Urticaceæ.	Urtica.
52	Cannabaceæ.	Cannabis, Humulus.
53	Salicaceæ.	Salix, Populus.

No.	ORDER.	GENUS.
54	Araceæ.	Arum, Calla, Acorus.
55	Iridaceæ.	Crocus, Gladiolus, Iris.
56	Amaryllidaceæ.	Narcissus, Leucojum, Galanthus.
57	Dioscoreaceæ.	Tamus.
58	Liliaceæ.	Tulipa, Scilla, Allium, Paris, Polygonatum, Convallaria, Majanthemum.
59	Colchicaceæ.	Colchicum, Veratrum.
60	Gramineæ.	Lolium.
61	Coniferæ.	Taxus, Juniperus, Sabina, Thuja.
62	Equisetaceæ.	Equisetum.
63	Polypodiaceæ.	Aspidium, Phegopteris, Asplenium.
64	Fungi, mushrooms.	Amanita (or Agaricus), Lactarius (or Galorrhœus), Russula, Inocybe, Hypholoma, Boletus, Helvella, Clavicèps.

It is desirable to investigate anew many of the plants contained in the foregoing table, since they have not as yet been studied in a satisfactory manner. *We must not for a moment believe that the partaking of relatively small particles of the different plants mentioned, will at once produce intoxication.* Many of them contain active principles capable of toxic action when in a pure state, but they are contained in the plant in small quantities only. We have consequently referred to them as "pharmacologically important plants," instead of using the word "toxic" in this connection. The plants containing *mucous substances* (orchis, althæa) we omitted, because up to date no detrimental action of these substances is known. The *lichens* viz.: the parasitic fungi of algæ, are not mentioned in the column "order" for the reason that their toxic representatives do not occur in Germany.

C. Review of the Pharmacologially Interesting Subkingdoms and Species of Animals.

We include this review of animals, according to the natural system, in spite of the fact that we have treated the most

important animal poisons in special chapters of this book. We thus gain the advantage, at least for the in-vertebrates, of appending a few details, which otherwise we could not well rubricate. Regarding the species, we refer to the works on zoology, *e. g.*, Julius v. Kennel, Lehrbuch der Zoologie, Stuttgart, 1893.

TABLE OF POISONOUS ANIMALS, ARRANGED BY SUB-KINGDOMS.

SUB-KINGDOM.	GENERA AND SPECIES.
I. Cœlenterata	They are also called Knidaria, viz.: nettle-animals (from κνιδάω—to whip with nettles), because almost all representatives of this family possess nettle-capsules in the epidermic cells which serve as apparatus of defense, and of aggressiveness in securing prey. They possibly contain *formic acid* and an *enzyme*, causing violent local excitation in man. We mention physalia, cyanea, actinia, corallium.
II. Plathelminthes.	Of the three groups, viz.: the turbellaria, the trematodes and the cestodes, only the last two are of pharmacological interest. Distoma hæmatobium causes hæmaturia; it is, however, not proven that this is occasioned by a poison. It is more likely that it originates in a mechanical manner. Distoma hepaticum causes the gravest disturbances of the liver (icterus, acholia etc.), but also apparently by mechanical means. Under certain conditions bothriocephalus, and tænia, belonging to the cestodes, produce pernicious anæmia. It is questionable whether this takes place by vital products of metabolism or by ptomaines formed on putrefaction of the dead animals.
III. Nemathelminthes.	Trichina spiralis causes hyperidrosis and stiffness as well as terrible pain, but probably in a merely mechanical way. Dochmius duodenalis causes the severe anæmia known by the name of Egyptian chlorosis or Gotthard-tunnel disease. This is most likely brought about by deprivation of blood in the duodenum, which it causes.
IV. Mollusca.	Some snails, *e. g.*, dolium galea, secrete free sulfuric acid in such a concentrated form, that in man corrosion is produced. The sea-hare, aplysia, was supposed in ancient and mediæval times to be exceedingly poisonous; we know to-day that it only becomes poisonous in a diseased state or on putrefying. The same is true of the common mussel, mytilus (edulis), and of the oyster, ostrea. The true pearl-oyster (oriental pearl-mussel), Meleagrina margaritifera, the pearly turbo and haliotis (the ear-shell, ormer or abalone) produce a disease in the polishers who handle these shells. This disease causes an inflammation of the bones resembling the necrosis by phosphorus. It is unknown what poison is involved in this case.

SUB-KINGDOM.	GENERA AND SPECIES.
V. Annelida.	Some species of hæmentaria, used as leeches in the tropics, produce an inflammation of the skin emanating from the bitten spot. It is an open question what causes this. Our leech, hirudo, secretes a poison (enzyme?) which is not destroyed on boiling. This prevents the blood from coagulating.
VI. Tracheata.	This great family has three sub-classes, all of which interest us here: 1. Myriapoda (centipedes, millipedes). Scolopendra possesses a poison-gland the secretion of which makes the bite dangerous. The nature of the poison is unknown. Fontaria, when touched, secretes hydrocyanic acid. Julus, centipede. 2. Hexapoda, insects. Of the many sub-divisions of interest to us, are: (a) the orthoptera, on account of periplaneta or blatta, a cockroach (Schabe or Tarakane) used in Russia as a diuretic (antihydropin); properly speaking, it is not poisonous. (b) To the rhynchotes (weevils) belongs huechis sanguinea, used in China for the same purposes as our Spanish-fly (lytta vesicatoria). The plant-lice (aphis, etc.), contain a locally excitating substance. The same is to be said of the human lice, pediculus and phthirius, as well as of pentatoma rufipes, the hydrocores and cimex (acanthia). (c) Of the beetles, coleoptera, we have already mentioned (pages 106 and 18) lytta, meloë (oil-beetle), and gyrinus, the whirligig. It has not been accurately investigated whether or not cetonia acts in a similar manner. The same is to be said of carabus. (d) The caterpillars of some of the lepidoptera or butterflies are endowed with nettling hair, each hair emanating from a poison-gland, *e. g.*, cnethocampa processionea, and arctia, the caterpillar of arctia nais. This poison appears to contain an enzyme besides formic acid. The caterpillar of the garden-white or cabbage-white, pieris, though possessed of only short hairs, produces in animals eating it inflammation of the stomach, paralysis, etc. (e) Of the hymenoptera we mention the ants (formica, myrmica, atta), wasps (vespa), hornets (vespa crabro), humble-bee or bumble-bee (bombus), and bees (apis, xylocopa). The poison of these animals has not been satisfactorily investigated; in each case there is a locally excitating action. (Compare page 106). The poison of the ants contains formic acid, undecane, and an enzyme. (f) Of the diptera we mention culex and simulia, tabanus (horse-cattle-breeze-flies), and the flea (pulex). The poison of these animals is of composition similar to that of the foregoing. 3. Arachnida. Under this heading belong the genuine and non-genuine spiders, mentioned on page 107, as well as the scorpions (euscorpins, buthus, androctonus), whose poison is very dangerous. It contains a peculiar toxalbumin. Finally, we must mention in this place the mites, of which argas and sarcoptes (sarcoptes scabei, the itch-mite) appear to possess peculiar poisonous substances.
VII. Vertebrata.	The poison of fishes, snakes, toads and salamanders has been discussed on pages 102 and 106.

D. Review of the More Important Pharmacological Reactions, which are occasionally Quoted by the Author's Name.

A great number of chemical reactions in common use are occasionally—and some of them almost always designated by the name of persons who either discovered or modified them, or even who only popularized them. We have in this work scarcely mentioned any author. A supplementary enumeration therefore, of such reactions, which are synonymous with certain proper names, should be welcomed by the physician. While busy in arranging a table correspondingly, our endeavors were anticipated by the publication of such a one, prepared by C. DÜNNENBERGER, in "Schweizer Wochenschrift für Chemie und Pharmacie," (1894, Nos. 5-11). From this we have abstracted such reactions as will be considered of importance to the reader.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
1	Allen.	Phenol.	A mixture of HCl and HNO_3 produces a carmine-red color.
2	Almén-Schönbein-van Deen	Blood or hæmoglobin.	A mixture of newly prepared tincture of guaiacum and spirits of turpentine, of equal parts; turns blue. The color passes into alcohol on agitation.
3	Arnold.	Atropin and homatropin.	Both alkaloids turn, first red-violet, then pale rose-red, if rubbed with conc. H_2SO_4, and then addition of a trace of sodium nitrite and 30 to 40 p.c. KOH.
4	Baumann and Udransky.	Diamins and poly atomic alcohols.	A mixture of sodium hydrate solution and benzoyl chlorid. White precipitate.
5	Beissenhirtz.	Anilin.	On dissolving in conc. H_2SO_4 and addition of a little potassium dichromate, first red-, then blue-coloration.
6	Bettendorf.	Arsenic or arsenic acid.	Addition of stannous chlorid in conc. HCl to the solution, strongly acid of HCl, on warming turns brown. The reaction is disturbed by the presence of much H_2SO_4 or by oxidizing- or by organic substances.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
7	Bloxam.	Alkaloids.	A mixture of HCl and $KClO_3$ will produce colors with some of them.
8	Boddé.	Resorcinol.	Sodium hypochlorite colors first violet, then yellow.
9	Boedeker.	Albumin.	Potassium ferro-cyanid and acetic acid give a white precipitate.
10	Boettger.	Glucose.	On warming with sodium carbonate and bismuth sub-nitrate, black.
11	Boettger.	Ozone.	Acid-free auric-chlorid paper turns violet.
12	Bouchardat.	Alkaloids.	10 J + 20 KJ + 500 H_2O, brown precipitate.
13	Brand.	Quinin and quinidin.	Addition of chlorin-water and then of NH_3 produces a green flocculent precipitate (thalleiochin), soluble with emerald-green color in excess of NH_3.
14	Brandt.	Alkaloids.	Some of them are colored by a mixture of H_2SO_4 and selenic- or telluric-acid.
15	Brociner.	Alkaloids.	Coloration produced by H_2SO_4 + ammonium-niobate, or -uranate, or -tellurate.
16	Brouardel and Boutmy.	Morphin, various ptomaines, etc.	A mixture of potassium-ferri-cyanid and ferric chlorid is turned blue with partial reduction.
17	Buckingham.	Alkaloids.	See page 51.
18	Capranica.	Bile-pigments.	Urine is agitated with a solution of bromin in chloroform, when green coloration ensues, which on sequent agitation with HCl passes into the latter.
19	Davy.	Phenol.	Molybdic acid dissolved in H_2SO_4 turns blue.
20	Dittmar.	Alkaloids containing the pyridin-nucleus.	A solution of chlor-iodin produces yellowish-brown precipitates.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
21	Dragendorff.	Alkaloids.	Potassium-bismuth-iodid precipitates in acid solution kermès-red (crimson).
22	Drechsel.	Bile-acids.	On heating with cane-sugar and H_3PO_4, purple-red coloration.
23	Duflos.	Anilin.	Dilute H_2SO_4 + plumbum peroxid produces green color.
24	Dusart and Blondlot.	Phosphorus.	Free P, phosphin, phosphorous acid and argentum-phosphid turn the hydrogen-flame green.
25	Eboli.	Cantharidin.	Heating with H_2SO_4 and potassium-chromate, green coloration.
26	Ehrlich.	Bile-pigments.	A solution of bilirubin in chloroform is mixed with 1 to 2 vols. of a 0.1 p.c. solution of diazo-benzene-sulfonic acid, strongly acidified by HCl, and as much alcohol added as to produce a homogeneous mixture. Turns red, and, upon addition of conc. HCl, violet, blue-violet, and finally pure blue. On cautious addition of KOH, two layers form: the lower one greenish blue, the upper pure blue; between the two we find a red streak.
27	Einhorn.	Glucose.	Yeast is added to the urine, and this is allowed to ferment in the fermentation saccharometer. The amount of sugar is calculated from the volume of CO_2 obtained.
28	Erdmann.	Alkaloids.	Conc. H_2SO_4 with KNO_3 or with a little HNO_3 will give colorations with individual alkaloids.
29	Essbach.	Albumin.	1 picric acid + 2 citric acid + water ad 100 produces yellow precipitate.
30	Fehling.	Glucose.	34.639 $CuSO_4$, $5H_2O$ are dissolved in water to 500 c.c. Then 173.0 Rochelle salts + 50.0 solid NaOH are dissolved in water to 500 c.c. In using, equal volumes of each are mixed. 10 c.c. correspond to 0.05 glucose. The blue solution will be decolorized under formation of a red precipitate, either before or while boiling.

No.	REAGENT OF;	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
31	Em. Fischer.	Sugars.	Phenylhydrazin with sodium acetate will produce crystalline compounds on boiling.
32	Fresenius.	Phenol.	On boiling with acidulated mercurous nitrate, Hg separates and the odor of salicylic aldehyde obtains.
33	Fröhde.	Alkaloids, etc.	See page 51. Protein-substances turn dark blue.
34	Gaglio-Gruber.	Carbon monoxid.	Blood, containing CO, is boiled, for hours, with KOH, while, by means of a current of air, the escaping gas is quantitatively passed into palladious chlorid, which it will blacken.
35	Gardiner.	Tannic acids.	A conc. solution of ammonium molybdate produces a yellow coloration.
36	Gautier.	Tannic acids.	Precipitation by aqueous cupric acetate solution (1:30).
37	Gerrard.	Atropin and hyoscyamin.	A 5 p.c. solution of corrosive sublimate in alcohol of 50 p.c. causes red precipitation; homatropin will not be precipitated.
38	Girard.	Coloring matter of wines.	4 c.c. 10 p.c. aqueous KOH solution + 20 c.c. 5 p.c. H_2SO_4 produces with natural wines a colorless, in presence of tar-colors, a red filtrate.
39	Gmelin.	Bile-pigments.	A layer of urine on top of fuming nitric acid produces simultaneously green, blue, violet, red and yellow rings of colors. The green ring is uppermost.
40	Griess.	Nitrous acid and nitrites.	Meta-diamido-benzene hydrochlorid turns the solution brown.
41	Günzburg.	Free hydrochloric acid.	2.0 phloroglucin + 1.0 vanillin + 30.0 alcohol. A few drops will turn red, *e.g.*, on evaporation with gastric juice.
42	Gunning.	Acetone.	On addition of iodin and NH_3, iodoform is produced. Alcohol will not react in this manner.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
43	Gutzeit.	Arsenic.	Moist, argentic nitrate paper held at the mouth of the delivery tube of AsH_3 turns black or yellow with black edge.
44	Hager.	Arsenic.	A drop of its solution in HCl produces a dark-grey or black spot when heated on a bright strip of copper.
45	Hammarsten-Jaffe.	Indican.	Urine mixed with fuming hydrochloric acid and bleaching powder ($Ca(OCl)Cl$), when agitated with chloroform will turn this the color of indigo-blue.
46	Heller.	Blood in urine.	The phosphates precipitated will be of brownish-red color if the urine is boiled with KOH.
47	Herapath.	Quinin.	The alcoholic solution upon addition of iodin-tincture, drop by drop, will produce a crystalline precipitate of iodo-quinin-sulfate.
48	Hlasiwetz.	Hydrocyanic acid.	On heating with free alkali and picric acid, blood-red coloration.
49	Hoffmann.	Phenol.	Conc. H_2SO_4 + KNO_3 produce violet coloration.
50	Hoffmann.	Tyrosin.	The hot aqueous solution is turned dark-red by mercurous nitrate + potassium nitrite.
51	Hofmann.	Anilin.	On heating with fuming nitric acid, first yellow coloration, then red, then dark-blue.
52	Hofmann.	Primary amines.	Heating with alcoholic KOH and chloroform generates odor of carbylamin. (Iso-nitril-reaction.)
53	Hoppe-Seyler.	Carbon monoxid-blood.	Mixing to equal parts with conc. NaOH will not produce a brownish-green, but a cinnabar coloration.
54	Hoppe-Seyler.	Phenol.	Shavings of pine-wood moistened with HCl will turn blue with phenol.
55	Hüfner and Knop.	Urea.	Sodium hypobromite solution, newly prepared, develops free nitrogen.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
56	Jacquemin.	Anilin.	Upon addition of ammonium sulfid, first rose-red, then yellow coloration.
57	Jacquemin.	Phenol.	Upon addition of anilin and then of sodium hypochlorite, blue coloration, changed by acids into red.
58	Johnson.	Glucose.	On heating with KOH + picric acid, dark-red coloration, caused by formation of potassium-iso-purpurate.
59	Ittner.	Hydro-cyanic acid.	The solution is made alkaline, mixed with ferrous- and ferric-hydrate, and acidulated with HCl. Prussian blue will form.
60	Kjeldahl.	Nitrogen in organic compounds.	The organic substance is thoroughly destroyed by heating with fuming sulfuric acid; water and NaOH are added, and the ammonia produced is distilled off into normal acid, and titrated.
61	Knapp.	Glucoses.	Black mercury will be deposited on heating with a colorless solution of mercuric cyanid ($Hg(CN)_2$).
62	Kobert.	Hydro-cyanic acid.	Brown met-hæmoglobin solution will be reddened by CNH. This reaction serves also for the detection of met-hæmoglobin.
63	Kobert.	Hæmoglobin.	Agitation with zinc-dust or addition of zinc-sulfate or -acetate furnishes a precipitate of zinc-par-hæmoglobin, soluble in alkali with red color.
64	Ladendorf.	Blood.	On mixing the liquid with tincture of guaiacum and then with Eucalyptus-oil, the lower layer will turn blue, the upper one violet.
65	Landolt.	Phenol.	Bromin-water produces a white, crystalline precipitate of tri-brom-phenol bromid.
66	Lassaigne.	Nitrogen in organic compounds.	The organic substance is heated with metallic sodium, a slight explosion must follow. The mass is extracted with water, in order to dissolve the NaCN produced. The filtrate is mixed with ferro-ferri-sulfate, acidulated with HCl, when Prussian blue will be formed.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
67	Lieben.	Acetone and alcohol.	The distillate, *e. g.*, of urine, will furnish iodoform with KOH and iodin in potassium iodid solution.
68	Liebermann.	Cholesterin and fats containing cholesterin.	The solution in acetic acid anhydrid, treated with conc. H_2SO_4, turns rose-red, then blue, and finally green.
69	Liebig.	Hydrocyanic acid.	The solution, cautiously evaporated with ammonium sulfid, will produce a blood-red coloration of ferrum-sulfo-cyanate.
70	Liebig.	Cystin.	Cystin boiled in sodium hydrate solution containing plumbic oxid, turns black from plumbic sulfid formed.
71	Lugol (Wagner-Fresenius).	Alkaloids, starch.	Lugol's solution is a solution of iodin in potassium iodid. It turns starch blue and precipitates alkaloids.
72	Mandelin.	Alkaloids.	Colorations are produced with some alkaloids upon addition of a solution of ammonium vanadate in conc. H_2SO_4.
73	Marmé.	Alkaloids.	Potassium-cadmium-iodid acts as a precipitant in acid solutions.
74	Marsh.	Arsenic.	See page 94.
75	Mayer (Ferd.)	Alkaloids.	Mercuric iodid-potassium-iodid acts as a precipitant in acid solutions. Compare page 50.
76	Méhu.	Albumin.	A mixture of 1 phenol + 1 acetic acid + 2 alcohol acts as a precipitant in presence of HNO_3.
77	Meyer (Vikt.)	Thiophene and its homologues.	A solution of isatin in conc. H_2SO_4 produces blue coloration.
78	Millon.	Albumin and many aromatic substances.	Mercury dissolved by warming in an equal weight of HNO_3 (1,4 spec. grav.), and diluted with water to twice the volume, produces brick-red coloration on warming.
79	Mitscherlich.	Phosphorus.	The vapors, on acid distillation, are luminous in the dark. Compare page 44 and page 99.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
80	Molisch.	Carbohydrates	Alcoholic solution of α-naphthol or thymol will produce, when mixed with the solution in question and conc. H_2SO_4, a violet color, and upon addition of water a bluish-violet precipitate. (Depends on furfurol formation.)
81	Moore.	Glucoses.	Brown coloration on heating with KOH, and, on subsequent acidifying, odor of caramel.
82	Mulder.	Glucoses.	The blue solution of indigo-carmine, to which sodium carbonate had been added, will be decolorized on warming with glucoses, but on agitation with air the blue color reappears.
83	Mulder.	Albumin-substances.	Conc. HNO_3 produces a lemon-yellow coloration; alkali intensifies the color, which is due to xanthoprotein.
84	Nessler.	Ammonia.	2 KJ + $5H_2O$ + $4HgCl_2$ are warmed together. After cooling, dilute with 40 H_2O, filter and add 10 KOH. Even traces of ammonia produce a yellow tinge, larger amounts cause a brown precipitation.
85	Nylander.	Glucoses.	A solution of 2 bismuth sub-nitrate + 4 rochelle-salts + 100 NaOH of 8 p.c. turns black on boiling with glucoses.
86	Pellagri.	Morphin.	Purple-red coloration on evaporation with HCl + H_2SO_4.
87	Piria-Städeler.	Tyrosin.	The substance in question is gently warmed with a little conc. H_2SO_4, the solution, temporarily of deep-red color, diluted with H_2O, neutralized with $BaCO_3$, boiled, filtered, and Fe_2Cl_6 added drop by drop. In case tyrosin is present, a violet coloration will obtain.
88	Plugge.	Phenol, etc.	The aqueous solution will turn intensely red on boiling with a mercurous nitrate solution, containing traces of nitric acid.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
89	Preyer.	Hydrocyanic acid.	Very diluted, newly prepared tincture of guaiac resin, containing traces of $CuSO_4$ will be colored blue by CNH.
90	Reinsch.	Arsenic acid and arsenious oxid.	Metallic copper reduces the solution in HCl to form a grey film of cupric-arsenid.
91	Robinet.	Morphin.	Very dilute neutral ferric chlorid, containg oxy-chlorid, produces a blue evanescent coloration, in neutral solutions of the salts of morphin.
92	Roussin.	Nicotin.	On mixing ethereal nicotin solution (1:500) with ethereal iodin solution (1:75) well developed crystals of nicotin iodid form.
93	Rubner.	Glucose.	Glucose or diabetic urine, when warmed with ammoniacal plumbic acetate, will produce a precipitate, white at first, then flesh-color to purple-red.
94	Rubner.	Carbon monoxid blood.	On mixing normal blood with 4 to 5 times its volume of plumbic acetate and agitating for a minute, brown discoloration ensues. CO-blood retains a fine light-red color.
95	Schlagdenhauffen.	Alkaloids.	On warming to 60–70° C with a mixture of equal parts of a 3 p.c. tincture of guaiac resin and saturated corrosive sublimate solution, the alkaloids will show a blue coloration, the glycosids not.
96	Scheibler.	Alkaloids and glycosids.	Phospho-tungstic acid (phospho-wolframic-acid) causes, in HCl solution, a copious white precipitation.
97	Schneider.	Bismuth.	A solution of 3 parts of tartaric acid + 1 part stannous chlorid in potassium hydrate solution will precipitate bismuth salts, black on heating.
98	Schönbein.	Ozone, etc.	Potassium iodid-starch solution (1:10:200) will be turned blue.
99	Schönbein.	Copper.	Turns blue by a mixture of potassium cyanid and tincture of guaiacum.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
100	Schönbein.	Hydro-cyanic acid.	Bibulous paper soaked in a guaiac-tincture of 1 p.c. and additionally moistened with cupric sulfate solution of 0.1 p.c. will turn blue with HCN.
101	Smith.	Bile-pigments.	A layer of iodin-tincture poured over urine produces a green coloration.
102	Soldaini.	Glucoses.	15 cupric carbonate dissolved in 1400 c.c. water and 416 potassium bi-carbonate added. Reduction on heating.
103	Sonnenschein.	Alkaloids.	In acid solutions phospho-tungstic acid will precipitate them.
104	Spiegler.	Albumin.	8 $HgCl_2$ + 4 tartaric acid + 200 H_2O + 20 glycerin. Urine is acidu'ated with acetic acid, to remove mucin; in contact with the reagent, a white ring appears.
105	Teichmann.	Blood.	Glacial acetic acid and a trace of NaCl added to the substance in question, evaporated on the objective plate will yield crystals of hæmin.
106	Trapp.	Veratrin.	On boiling with conc. HCl, purple-red coloration, not evanescent.
107	Trommer.	Glucoses.	On warming with sodium carbonate and cupric sulfate, the precipitate caused will dissolve with blue coloration of the liquid. Then the color changes to red and $Cu(OH)_2$ will be precipitated.
108	Ultzmann.	Bile-pigments.	Agitate 10 c.c. of urine with 3 c.c. KOH (1:3) and oversaturate with HCl; an emerald-green coloration will ensue.
109	Valentin.	Fuchsin.	Fuchsin solutions on agitation with ether will only produce a violet coloration after addition of ferric iodid.
110	Vitali.	Atropin, etc.	After evaporation with fuming HNO_3, the residue will turn violet with alcoholic KOH.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
111	Wayne.	Glucoses.	2 cupric sulfate + 10 KOH + 10 glycerol, dissolved in 200 water. On warming, reduction.
112	Wenzel.	Alkaloids.	Some of them will show color reactions with sulfuric acid containing potassium permanganate (1:200), *e.g.*, veratrin: light-red.
113	Weppen.	Veratrin.	Furfurol-sulfuric acid turns yellow, dark-green, blue, and finally a dirty violet.
114	Wetzel (Kunkel).	Carbon monoxid-blood.	10 c.c. of the blood are mixed with 15 cc. of a solution of 20 p.c. potassium-ferro-cyanate and 2 c.c. acetic acid. A red coagulum will be formed, and not a brown one, as in case of normal blood.
115	Wetzel (Kunkel).	Carbon monoxid-blood.	Blood is diluted to four times its volume with water. Three times of the total volume of 1 p.c. tannin-solution is added. Normal blood will turn gradually grey, carbon-monoxid-blood carmine-red.
116	Weyl.	Creatinin.	To urine some sodium nitroprussid ($Fe(CN)_5 . NO . Na_2$) + $2H_2O$ and NaOH is added. The color produced is ruby-red.
117	Wurster.	Active oxygen, etc.	Paper soaked in tetra-methyl-para-phenylene-diamin (so-called tetra-paper) will be turned blue.
118	v. Zaleski.	Carbon monoxid-blood.	On mixing 2 c.c. CO-blood with as much water and three drops of a $\frac{1}{3}$ saturated cupric sulfate solution, a brick-red precipitate will appear. With normal blood the precipitate will be of greenish-brown color.
119	Zeissl.	Colchicin.	3 milligrms. of the substance dissolved in 5 c.c. H_2O are boiled for 1–3 minutes with 5–10 drops fuming HCl and 4–6 drops Fe_2Cl_6. First a yellow, then a green coloration will ensue. If now the mixture is agitated with chloroform and admission of air, the chloroform turns red and the supernatant liquid green.

No.	REAGENT OF:	FOR THE DETECTION OF:	PREPARATION, COMPOSITION, ACTION.
120	Dragendorff & Podwyssotzki.	Erythro-sclerotin, coloring matter of Secale cornutum.	Extract with alcohol containing H_2SO_4, filter and agitate the filtrate with ether. Transfer the coloring matter, by agitation of the ethereal solution with an aqueous solution of sodium bi-carbonate, into this latter. Put the liquid before the slit of a spectroscope. One absorption band in the blue and one in the green will be observed.
121	C. C. Keller (Blumberg, Tanret).	Cornutin, Ergotinin (alkaloids from Secale cornutum)	H_2SO_4 and ferric chlorid produce first orange-, then deep-red-coloration, while the peripheral zone will turn bluish to bluish-green.

RULES FOR THE SPELLING AND PRONUNCIATION OF CHEMICAL TERMS.

ADOPTED BY THE AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE IN 1891.

In 1887 a committee was appointed by the American Association for the Advancement of Science, to consider the question of attaining uniformity in the spelling and pronunciation of chemical terms. The work of this committee extended through the following four years. As a result of widespread correspondence and detailed discussion at the annual meetings of the Chemical Section of the American Association, the accompanying rules have been formulated and adopted by the Association. They are submitted to chemists generally, and especially to the large number of those engaged in teaching chemistry, with the request that a cordial and earnest effort be made to render their use general and thus obviate the many difficulties arising from the present diversities of style.

The following summary of the rules has been arranged in the form of a chart for general distribution to high schools and colleges, so that they may be kept permanently and prominently before the eyes of teachers and pupils.

Committe.

T. H. NORTON, Ph.D., Professor of Chemistry, University of Cincinnati.
EDWARD HART, Ph.D., Professor of Chemistry, Lafayette College, Easton, Pa.
H. CARRINGTON BOLTON, Ph.D., University Club, New York City.
JAS. LEWIS HOWE, Ph.D., M.D., Polytechnic Society, Louisville, Ky.

GENERAL PRINCIPLES OF PRONUNCIATION.

1. The pronunciation is as much in accord with the analogy of the English language as possible.
2. Derivatives retain as far as possible the accent and pronunciation of the root word.
3. Distinctly chemical compound words retain the accent and pronunciation of each portion.
4. Similarly sounding endings for dissimilar compounds are avoided (hence **-ĭd, -īte**).

ACCENT.

In polysyllabic chemical words the accent is generally on the antepenult; in words where the vowel of the penult is followed by two consonants, and in all words ending in **-ic,** the accent is on the penult.

PREFIXES.

All prefixes in strictly chemical words are regarded as parts of compound words, and retain their own pronunciation unchanged (as, ā′ceto-, ă′mĭdo-, ă′zo-, hȳ′dro-, ī′so-, nī′tro-, nītrō′so-).

ELEMENTS.

In words ending in **-ium,** the vowel of the antepenult is short if **i** (as īrĭ′dium), or **y** (as dĭdȳ′mium), or if before two consonants (as că′lcium), but long otherwise (as tĭtā′nium, sĕlē′nium, chrō′mium).

alū′minum
a′ntimony
a′rsĕnic
bā′rium
bĭ′smuth (biz)
bō′ron
brō′mĭn
că′dmium
că′lcium
ca′rbon
cē′rium
cē′sium
chlō′rĭn
chrō′mium
cō′balt
colŭ′mbium
co′pper
dĭdў′mium
e′rbium
flū′orĭn
gă′llium
germā′nium
glū′cinum
gold
hў′drogen
ĭ′ndium
ĭ′odĭn
ĭrĭ′dium
iron
lă′nthanum
lead
lĭ′thium
magnē′sium
(zhium)
ma′nganese
(eze)
me′rcury
mōlў′bdenum
nĭ′ckel
nĭ′trogen
ŏ′smium
ŏ′xygen
pallā′dium
phŏs′phorous
plă′tinum
potă′ssium
rhō′dium
rubĭ′dium
ruthē′nium
samā′rium
scă′ndium
sĕlē′nium
sĭ′licon
silver
stro′ntium
(shium)
sū′lfur
tă′ntalum
tellū′rium
te′rbium
thă′llium
thō′rium
tin
tĭtā′nium
tŭ′ngsten
ūrā′nium
vănā′dium
ytte′rbium
ў′ttrium
zinc
zircō′nium

Also: ămmō′nium, phosphō′nium, hă′logen, cўă′nogen, ămĭ′dogen.

Note in the above list the spelling of the halogens, cesium, and sulfur; **f** is used in the place of **ph** in all derivatives of sulfur (as sulfuric, sulfite, sulfo-, etc.)

TERMINATIONS IN **-ic.**

The vowel of the penult in polysyllables is short (as cўă′nic, fûmăric, arsĕ′nic, sillĭ′cic, ĭŏ′dic, būtў′ric), except (1) **u** when not used before two consonants (as mercū′ric, prŭ′ssic), and (2) when the penult ends in a vowel (as benzō′ic, olē′ic), in dissyllables it is long except before two consonants (as bō′ric, cĭ′tric). Exception: acē′tic or acĕ′tic.

The termination **-ic,** is used for metals only where necessary to contrast with **-ous** (thus avoid aluminic, ammonic, etc.)

TERMINATIONS IN **-ous.**

The accent follows the general rule (as plă′tinous, sŭ′lfurous, phŏ′sphorous, coba′ltous). Exception: acē′tous.

TERMINATIONS IN **-ate** AND **-ite.**

The accent follows the general rule (as ă′cetāte, vă′nadāte): in the following words the accent is thrown back: ă′bietāte, ă′lcoholāte, ă′cetonāte, ă′ntimonīte.

TERMINATIONS IN **-id** (FORMERLY **-ide**).

The final **e** is dropped in every case and the syllable pronounced **ĭd** (as chlō′rĭd, ĭ′odĭd, hў′drĭd, ŏxĭd, hўdrŏ′xĭd, sŭ′lfĭd, ă′mĭd, ă′nilĭd, mūrĕ′xĭd).

TERMINATIONS IN **-ane, -ene, -ine** AND **-one.**

The vowel of these syllables is invariably long (as mĕ′thāne, ĕ′thāne, na′phthalēne, a′nthracēne, prō′pīne, quĭ′nōne, ă′cetōne, kē′tōne).

A few dissyllables have no distinct accent (as benzēne, xўlēne, cētēne).

The termination **-ine** is used only in the case of doubly unsaturated hydrocarbons, according to Hofmann's grouping (as propīne).

TERMINATIONS IN **-in.**

In names of chemical elements and compounds of this class, which includes all those formerly ending in **-ine** (except doubly unsaturated hydrocarbons) the final **e** is dropped, and the syllable pronounced **-ĭn** (as chlō′rĭn, brō′mĭn, etc., ă′mĭn, ă′nilĭn, mo′rphĭn, quĭ′nĭn (kwĭ′nĭn), vanĭ′llĭn, alloxă′ntĭn, absĭ′nthĭn, emŭ′lsĭn, că′ffeĭn, cō′caĭn).

TERMINATIONS IN **-ol.**

This termination, in the case of specific chemical compounds, is used *exclusively* for alcohols, and when so used is never followed by a final **e.** The last syllable is pronounced **-ōl** (as glў′cōl, phē′nōl, crē′sōl, thў′mōl, (ti), glў′cerol, quĭ′nōl. Exceptions: ălcohŏl, a′rgōl.

TERMINATIONS IN **-ole.**

This termination is always pronounced **-ōle,** and its use is limited to compounds which are not alcohols (as ĭ'ndōle).

TERMINATIONS IN **-yl.**

No final **e** is used; the syllable is pronounced **ȳl** (as ă'cetȳl, ă'mȳl, cē'rotȳl, cē'tȳl, ĕ'thȳl).

TERMINATIONS IN **-yde.**

The **y** is long (as ă'ldehȳde).

TERMINATIONS IN **-meter.**

The accent follows the general rule (as hydrŏ'meter, barŏ'meter, lactŏ'meter). Exception: words of this class used in the metric system are regarded as compound words, and each portion retains its own accent (as cĕ'ntime"ter, mĭ'llime"ter, kĭ'lome"ter).

MISCELLANEOUS WORDS

which do not fall under the preceding rules.

Note the spelling: albumen, albuminous, albuminiferous, asbestos, gramme, radical.

Note the pronunciation: a'lkalīne, a'lloy (n & v.), a'llotropy, a'llotropism, ĭ'somerism, pŏ'lymerism, apparā'tus (sing. & plu.), āqua regia, barȳ'ta, cĕntigrade, co'ncentrated, crystallīn or crystallīne, electrŏ'lysis, lîter, mŏ'lecule, mŏlĕ'cular, nō'menclā"ture, olē'fiant, vā'lence, ū'nivā"lent, bī'vā"lent, trī'vā"lent, qua'drivā"lent, tĭ'trate.

A LIST OF WORDS WHOSE USE SHOULD BE AVOIDED IN FAVOR OF THE ACCOMPANYING SYNONYMS.

For—	*Use—*
sodic, calcic, zincic, nickelic, etc., chlorid, etc.	sodium, calcium, zinc, nickel, etc., chlorid, etc. (vid. terminations in **-ic** supra.)
arsenetted hydrogen	arsin
antimonetted hydrogen	stibin
phosphoretted hydrogen	phosphin
sulfuretted hydrogen, etc.	hydrogen sulfid, etc.

For—	*Use—*
beryllium	glucinum
niobium	columbium
glycerin	glycerol
hydroquinone (& hydrochinon)	quinol
pyrocatechin	catechol
resorcin, etc.	resorcinol, etc.
mannite	mannitol
dulcite, etc.	dulcitol, etc.
benzol	benzene
toluol, etc.	toluene, etc.
thein	caffein
furfurol	furfuraldehyde
fucusol	fucusaldehyde
anisol	methyl phenate
phenetol	ethyl phenate
anethol	methyl allyl-phenol
alkylogens	alkyl haloids
titer (n.)	strength or standard
titer (v.)	titrate
monovalent	univalent
divalent, etc.	bivalent, etc.
quantivalence	valence

Fāte, făt, fär, mēte, mĕt, pīne, pĭn, marîne, nōte, nŏt, mōve, tūbe, tŭb, rûle, mȳ, ȳ = ĭ.

' Primary accent; " secondary accent. N.B.—The accent follows the vowel of the syllable upon which the stress falls, but does not indicate the division of the word into syllables.

ALPHABETICAL INDEX.