A Manual of Poisonous Plants

Chiefly of Eastern North America, with Brief Notes on Economic and Medicinal Plants, and Numerous Illustrations

[Part I]

By

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THE TORCH PRESS
CEDAR RAPIDS, IOWA
1910

FOREWORD

During the last decade, there has been much interest manifested in regard to plants injurious to live stock. Numerous contributions have been made along this line, notably by Dr. Chesnut, formerly of the United States Department of Agriculture, Drs. True and Wilcox and their co-workers also of Washington, Dr. Schaffner of the Ohio State University, Dr. Jones of the Vermont Agricultural Experiment Station, Dr. Kennedy of the Nevada Experiment Station, Dr. Nelson of the Wyoming Station, Dr. Nelson of Washington, and Drs. Peters and Bessey of Nebraska. Other station botanists have also contributed to the same line of work. Much of the literature is scattered, however, hence an effort has been made to bring together in the following pages the results obtained.

Much information on this line of investigation has also been obtained from such works as Millspaugh's Medicinal Plants of North America, Dr. Johnson's Manual of the Medical Botany of North America, Lloyd's Drugs and Medicines of North America, Winslow's Veterinary Materia Medica and Therapeutics, Sayre's Organic Materia Medica and Pharmacognosy, Flückiger and Hanbury's Pharmacographia, Greenish's Materia Medica, Ellingwood's Materia Medica, Therapeutics and Pharmacognosy, Pereira's Materia Medica, Luerssen's and Czapek's publications and many others which give details in regard to the effects of poisonous plants.

Many persons may object to the great number of plants which are here regarded as poisonous or described as such in this work. I have placed the broadest interpretation on the subject and have, therefore, included all plants that are injurious although many of these are not known to produce poisons, some even being most useful economic plants and yet injurious to some people.

It has been thought best to arrange the manual so as to consider the plants in the same order as that given in Engler and Prantl's Die Pflanzenfamilien.

The Schizomycetes were contributed by my colleague, Dr. R. E. Buchanan, who has also favored me in many other ways.

The parts concerning the blue-green algae and algae, taking up the higher algae and their relation to the water slimes are given in their sequence under the Schizophyceae and Euphyceae.

The Eumycetes or true fungi are considered chiefly from the pathogenic standpoint; while other fungi are referred to and briefly considered under their respective groups. In regard to the higher fungi, such as the toadstools, much valuable information may be obtained from the works of Dr. Farlow, Prof. Peck and Prof. Atkinson.

The so-called Blastomycetic fungi have been arranged under the group of the imperfectly known forms. There are also brief characterizations of other groups of the cryptogams such as lichens, mosses, ferns and their allies.

The flowering plants or Spermatophyta are described more fully than the previous groups, and under the various orders and subdivisions we have also added notes on economic and medicinal plants. Those who desire fuller information in regard to other American species of the different orders described should consult the latest editions of Gray's or Britton's Manuals, in which the descriptions are full and complete. Of course, one cannot expect to add much to the excellent descriptions given in these treatises.

I have freely made use of published literature in the systematic portions as well as in the more technical matter pertaining to poisons, and I wish to acknowledge my assistance from these sources.

In order that the species named may be more readily recognized, a large number have been figured.

In addition to the descriptive part of the work there has been added a chapter on the active principles of plants, by my colleague, Prof. A. A. Bennett. I am also greatly indebted to Miss Harriette S. Kellogg who has carefully read the manuscript and assisted me in other ways besides preparing the bibliography. To Dr. Trelease of the Missouri Botanical Garden, I am indebted for the use of a number of books on the subject. Miss Charlotte M. King, Miss Ada Hayden, Mr. W. S. Dudgeon, and my daughter, Lois, have made drawings especially for this work. Other illustrations are taken from special works, due credit being given in each case.

I am indebted for the use of cuts to the following persons: Dr. C. F. Curtiss of the Iowa Agricultural Experiment Station, Dr. B. D. Halsted of the New Jersey Agricultural Experiment Station, Profs. S. B. Green and Washburn of the Minnesota Station and Prof. A. D. Selby of the Ohio Station, and to the United States Department of Agriculture. Some have been reproduced from Baillon's Dict., and from Bentham's Handbook of British Plants. I have endeavored in each case to give credit for the drawing or cut.

I am indebted to Drs. R. R. Dykstra and C. H. Stange, Profs. L. G. Michael, C. V. Gregory and A. A. Bennett for proof reading and to Dr. W. H. Stuhr for some matter in Part I.

The work does not pretend to be complete; we hope, however, that it may prove useful to the Veterinarian, Physician and Layman.

Ames, Iowa, June 1, 1909.

CONTENTS

CHAPTER I

POISONS AND STATISTICS ON POISONS

A poison has been defined as "Any substance that, when taken into the system acts in a noxious manner by means not mechanical, tending to cause death or serious detriment to health."

Kobert and other physicians define a poison as "A non-organized body, either organic or inorganic, which under certain conditions, affects temporarily or permanently one or more organs of the body, when in a state of health or in a healthy condition." Such poisons may develop in the body or may come from without. Some substances act injuriously in a mechanical way, that is, they may set up disturbances by irritating some parts of the body. Other substances, while poisonous to one who is ill, may be entirely harmless to persons or animals in a state of health.

Kobert also defines poisons from a pharmacological standpoint as "All pharmacological agents which, in a given case, do not act beneficially but injuriously."

Toxicology is the science of poisons, the word being derived from the ancient word "tox," meaning bow, or arrow, probably from the ancient use of the arrow to kill.

In tracing the application of the word "tox;" "arrow," to its later application, poison, Blyth says: "Perchance the savage found that weapons soiled wth the blood of former victims made wounds fatal; from this observation, the next step naturally would be that of experiment,—the arrow or spear would be steeped in all manner of offensive pastes, and smeared with the vegetable juices of those plants which were deemed noxious; and as the effects were mysterious they would be ascribed to the supernatural powers, and covered with a veil of superstition."

The different tribes of Indians in South America have from early days been skilful in preparing arrow poisons, the majority of which contain strychnin in some form. The following plants have, at various times, furnished poisons for arrow tips, not only in South America, but also in other countries: *Strychnos toxifera* (Strychnine), perhaps the most generally used of any; *Antiaris toxicaria,* an arrow poison of Java, Borneo, and North Africa; various *Leguminosae,* as *Erythrophloeum* in Angola, Sierra Leone, and Seychelles, a different species being used in each place; *Pithecolobium, Afzelia,* and *Derris elliptica* of Borneo; of the Menispermaceae, two species of *Abuta* are used.

Perhaps in this connection, it would not be out of place to mention several fish-poisons, many of which are also legumes. Of this order are *Albidizzia, Afzelia, Bauhinia, Enterolobium, Leucaena, Milletia, Piscidia, Acacia, Abrus precatorius, Clitoria, Mundelia, Derris, Lonchocarpus,* and *Tephrosia.* In preparing the last named, the leaves are crushed and mixed with quicklime before using. Among the *Menispermaceae* are the Indian Berry, Fish-berry or Levant Nut (*Anamirta paniculata*) of the East Indies which contains picrotoxin; and

Pachygone ovata, used also by the Malays to poison crocodiles. Of the *Rhamnaceae*, Kraemer mentions *Tapura, Gouania*, and *Zizyphus*. Among the Tiliaceae, Kraemer, also mentions species of *Grewia* which are used as fish-poisons. The same authority adds species of *Barringtonia* of the order *Lecythidiaceae* and *Laportea stimulans* of the *Urticaceae* to the above list.

ANCIENT USE OF POISONS

In his work on "Poisons: their Effects and Detection," Blyth writes an excellent account of their history from which a few of the following more important data have been taken for the present work.

Their early history is involved in myth. Hecate was said to have been the discoverer of poisonous herbs and her knowledge passed in turn to Medea. The Egyptian kings, Menes and Attalus Philometer, not only had a knowledge of plants but the latter was also familiar with the uses of such plants as hyoscyamus, aconite, conium, and others of similar character. He experimented with poisons and compounded medicines. The Egyptians knew prussic acid, which was extracted from the peach and by means of which those who revealed religious secrets were put to death. The ancient Romans also must have been familiar with this poison, since a Roman knight once took poison and fell dead immediately at the feet of Samolus. The ancient Greeks knew about poisons and it was not considered a dishonorable thing to commit suicide.

Nicander of Colophon (204-138 B. C.) wrote two treatises on poisons, in one of which he described the effect of snake venom; in the other, henbane, aconite, conium, and fungi, were discussed. As antidotes for poisoning from any of these substances, he recommended such remedies as lukewarm oil, in order to excite vomiting.

Dioscorides (40-90 A. D.) divided poisons into (1) Animal poisons, as cantharides, poisonous snakes, the blood of an ox (probably putrid); (2) Poisons from plants, as opium, hyoscyamus, conium, aconite (the latter coming from Akron in Heraclea), and colchicum; (3) Mineral poisons like arsenic and mercury (cinnabar).

Pliny mentions that the Gauls dipped their arrows in a preparation of veratrum.

Toffana of Naples sold under the name of Acquetta di Napoli a solution of arsenious acid, by which, it is said, 605 persons were poisoned, among them the popes, Pius III and Clement XIV.

Poisoning was much practiced in India for the purpose of revenge, robbery and suicide, every little quarrel being liable to end in assassination of one of the parties. Such poisons as arsenic, aconite, opium, and extracts derived from plants of the Solanum family, were also used in India to destroy cattle. It is said that gipsies used *Phycomyces nitens*, having knowledge of its properties from the same country. The spores of the fungus were administered in warm water and death, accompanied with all the symptoms of tuberculosis, followed in a few weeks. The Hebrews seem to have been familiar with certain poisons, as arsenic, aconite and, possibly, ergot.

The deaths of Socrates, Demosthenes, Hannibal, and Cleopatra, were due to the administration of poisons.

In the early part of the Christian era, there were many professional poisoners and their business flourished, kings, emperors, popes, and members of the nobility being among their victims. There were two great criminal schools

in Venice between the tenth and seventeenth centuries, the government secretly recognizing the operations of these criminals and paying a sum of money for the execution of prisoners of note. However, these efforts were not often successful. J. Baptisa Porta, in the sixteenth century, wrote under the title of "Natural Magic," a work devoted partly to cookery and partly to poisons and how to use them.

The early methods of detecting the presence of poisons were crude; the surroundings were always noted; the suspected poison was generally administered to an animal and, if it died, poison was sure to be diagnosed without further investigation, since the early church forbade postmortem examination. Later, however, doctors were permitted to dissect and thus become familiar with pathological changes.

THE RISE OF CHEMISTRY AND POISONS

At the close of the eighteenth and the beginning of the nineteenth centuries, chemistry had advanced sufficiently to test for arsenic and the more important mineral poisons. Scheele discovered prussic acid; other chemists, as Berthollet, Lavoisier, and Stahl, added to the chemical knowledge of poisons The father of modern toxicology, however, was Bonaventura Orfila, whose work was published in 1814. Derosne discovered the alkaloids of narcotin and morphin in 1818. Pelletier and Cavantou discovered strychnin in 1818. Giesecke discovered coniine in 1827, and Geiger and Hesse separated atropin and hyoscyamin in 1833.

The modern aspect of the subject began with treatises on poisons by such workers as Vogel [1] and Richard Mead,[2] and writings on the subject of chemistry through the works of Stahl, Scheele, Berthollet, Priestly and Lavoisier. Botanists, too, at this time began to be greatly interested in a study of poisonous plants. Thus we have the work of Bulliard [3] and the work of Gmelin.[4] The work of Bulliard discusses a large number of poisonous plants with excellent illustrations, and Gmelin treats quite fully of the then known poisonous plants of Europe.

The works of Gmelin, Bulliard and Plenck [5] on Toxicology, and Buchner's Toxicology [6] were frequently quoted by the older writers. The greatest of the older writers, however, was Orfila [7] whose great work on toxicology became the recognized authority on toxicology. This work was first published in 1814, and passed through many editions. Orfila conducted actual experiments with different plants. This work of Orfila was also translated into different languages,[8] Orfila was preceded by Fodere.[9] About that period other toxicologies were published in France and Germany, such as those of Sobernheim and Simon,[10]

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1 The Usefulness of Natural Philosophy. 1654.

2 Mechanical Theory of Poisons.

3 Historie des plantes veneneuses et suspectes de la France. Paris 1784. Folio X, 177 p., 72 tab. col.—Ed. II: Paris 1798.

4 Abhandlung von den giftigen Gewächsen. Ulm. 1775. Allegemeine Geschichte der Pflanzengifte. Nurnberg 1777.

5 Toxicolgia, seu doctrina de venenis et antidotis. Viennae, Graeffer. 1785, 338 p.

6 Toxicologie. Nurnberg. 1827.

7 Traite des poisons, ou Toxicologie generale. Paris, 1813. Ed. III, ib. 1826. Orfila and Bonaventura. Traite des Poisons ou Toxicologie Generale. Paris, 1814. (Ed. 5) 1852.

8 Toxicologie. Seemann & Karls. 2 Vols. Berlin 1829-1831.

9 Nedec leg. Ed. 2.

10 Handbuch der praktischen Toxicologie. Berlin, 1838. Toxicologie. Nurnberg, 1827, 2nd Ed.

Taylor,[4] Hoffman,[5] Th. Husemann & A. Husemann,[6] von Pragg,[7] and Opwyrda Rebuteau,[8] Selmi,[9] Böhm and von Boeck,[10] Dragendorff,[11] Falck,[12] and more recent works by Joshua Nunn,[13] Smith,[14] Hutyra and Marck,[15] to say nothing of the recent contributions occurring in the American Veterinary Review, the Journal of the Chemical Society, American Journal of Pharmacy, besides treatises in many chemical and pharmaceutical journals.

The modern work of Blyth, though a somewhat exhaustive treatise on the subject of poisons, is not comprehensive so far as a large number of the poisonour plants are concerned. Many popular treatises on the subject of poisonous plants have appeared in nearly every European language both in ancient and modern times, but perhaps no one has contributed more to the subject of poisons than Kobert, who has published several treatises, and one of his works "Practical Toxicology for Physicians and Students" was translated into English by Dr. Friedburg. Such men as Dr. M. Greshoff of Haarlem, published a number of extended treatises on the subject "Poisons, especially Hydrocyanic Acids and Saponins, in Plants." His monograph on fish poisons and subsequent monographs two and three, really survey most of the poisonous plants of the world. Nor should we omit to mention the many treatises by Prof. Power of Wellcome laboratory and his students who have investigated a large number of poisonous plants or the work of Prof. Maiden of New South Wales, or of Cornevin of France.

Ratzeburg on Poisonous Plants.

Between 1834 and 1838 there appeared the first part of the work of Brandt and Ratzeburg on Phanerogamous poisonous plants of Germany, and in the year 1838 in the same work the poisonous Cryptogams by Phoebus. This like other works of the time contained numerous fine colored plates. This work pertaining to the flowering plants, lists the following plants of Germany as poisonous.

Darnel (*Lolium temulentum*) *Juniperus Sabina,* Yew (*Taxus baccata*) *Arum maculatum, Colchicum autumnale, Fritillaria imperialis, Narcissus pseudonar, sissus, Paris quadrifolia, Veratrum album,* Aconite (*Aconitum Anthora, A Lycoctonum, A. Cammarum, A. altigaleatum, A. variable*) *Anemone nemorosa, Caltha palustris, Helleborus niger, H. foetidus, H. viridis, Pulsatilla vulgaris,* Crowfoot (*Ranunculus sceleratus, R. acris, R. alpestris, R. repens, R. bulbosus, R. flammula, R. Thora*), *Papaver somniferum, Euphorbia palustris, E. Cyparissias, Rhus Toxicodendron, Acthusa Cynapium, Cicuta virosa, Conium maculatum, Coronilla varia, Oenanthe fistulosa, Sium latifolium, Ledum palustre, Cyclamen curopaeum, Daphne Mezereum, Nerium, Oleander, Cynanchum, Vincetoxicum, Atropa Belladonna, Datura Stramonium, Hyoscyamus niger, Mandragora vernalis, Scopolina atropoides, Solanum nigrum, Digitalis purpurea, Gratiola officina-*

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4 Principles and Practice of Medical Jurisprudence. 3 vols. London, 1873.
5 Hoffmann, Lehrbuch der gerichtlichen Medicin. 5th ed. Wien, 1890-91.
6 Husemann & Husemann, Handbuch der Toxicologie. Berlin, 1862.
7 Leerboek voor practische giftleer. In Swee Theilen. Utrecht, 1871.
8 Elemens de Toxicologie et de Medecine Legale, appliquees a 'lEmpoisonnement. Paris, 1873. 2nd ed. by Ed. Bourgoing, Paris, 1888.
9 Studi di Tossicologio Chimica. Bologna, 1871.
10 Böhm and von Boeck. Handbuch der Intoxicationen. (Bd. 15 of the German edition of Ziemssen's Cyclopaedia).
11 Die gerichtlichchemische Ermittelung von Giften in Nahrungsmitteln, Luftgemischen, Speiseresten, Korpertheilen, etc. St. Petersburg, 1868. 3rd ed. Gottingen, 1888.
12 Die Klinischtigen Intoxicationen (Handbuch der spec. Pathologie u. Therapie red. von R. Virchow, Bd. 2.) Erlangen, 1854.
13 Veterinary Toxicology. Wm. R. Jenkins & Co., N. Y., 1901, 1907.
14 A Manual of Veterinary Hygiene. 5th ed. Wm. R. Jenkins & Co., N. Y., 1035 pages.
15 Specielle Pathologie and Therapie der Hausthiere.

lis, and Lactuca virosa. Many poisonous fungi are enumerated in the second part.

It is interesting here to note their classifications of poisons. Sobernheim classifies the poisons into, A. vegetable poisons, B. nervous poisons, C. blood poisons. Orfila clasifies poisons into four classes, (1) irritant, acrid, corrosive, (2) narcotic, (3) narcotic acrid, (4) septic poisons. In 1834 Brandt and Ratzeburg classified poisons as to their origin into (1) mineral, (2) plant, (3) animal. Brandt and Ratzeberg in their treatise on plant poisons make three divisions (1) narcotic (stupefying), (2) acrid (inflammatory), (3) narcotic (inflammatory). They use the classification of Buchner which is as follows:- (1) narcotic, *a* HCN Prunus, *b*, volatile narcotic, Lolium, *c*, narcotic alkaloidal, Poppy; (2) acrid narcotic, Cicuta, Conium, Ruta, Digitalis; (3) irritant narcotic, *a*, Aconite, Oleander, Rhus, Smartweed, *b*, more volatile, hot acrid, Dirca, Pepper; (4) acrid, *a*, drastic resins, Bryonia, Hypericum, Melia, *b*, drastic coloring matter, Abrus, Spartium, Pokeweed, *c*, emetic alkaloids, Iris, Colchicum, Narcissus, *d*, unknown poisons, Agaricus, Boletus, Phallus, Lycoperdon. Fodere divided poisons into septic, narcotic, narcoacrid, acrid, irritant and astringent.

Statistics of Poisoning.

The use of poisons for criminal purposes, although not nearly so extensive at the present time as during the middle ages, still plays an important part in criminal law. The following statistics afford some indication of the use of poisons for suicidal and homicidal purposes.

According to the last census of the United States, the number of persons reported as poisoned was as follows:

	By Active Poisons	By Gas [1]
1902	1374	950
1903	1551	1715
1904	1632	2167
1905	1269	1306
1906	1734	1276

Intentional cases of poisoning in live stock are not nearly so frequent as are those in the human family, although there are many cases of the former on record. Poisoning of live stock is generally accidental, caused by consuming plants that are poisonous. Large losses occur annually in this way. In 1900 Prof. Chesnut and Dr. Wilcox investigated the conditions in Montana relative to this subject and published the following statistics resulting from their studies. They state that probably not more than one fourth of the actual cases occurring came under their observation.

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1 These numbers include deaths during, or as a result of conflagrations. The annual average was 1412 from active poisons and 1365 from gas. From 1900-1904, the number of deaths by poison averaged 4.5 per 100,000.

Poisoning cases among cattle, horses, and sheep in Montana observed during the season of 1900:

	SHEEP		CATTLE		HORSES	
	Poisoned	Died	Poisoned	Died	Poisoned	Died
Zygadenus venenosus	3030	636				
Zygadenus elegans	40	15	6	2	4	3
Lupine	3000	1900			4	3
Delphinium bicolor			2	2		
Delphinium glaucum			100	56		
Cicuta occidentalis	105	80	36	30		
Loco weeds	3550	700	3		150	3
Total	9725	3331	147	90	154	6

Nearly every veterinarian has frequent calls to attend cases of poisoning from obscure causes. These can often be traced to plants that occur in the pasture or feed lot.

Accidental cases of poisoning from such wild plants as jimson weed and others are frequent in the United States, several cases occurring annually from cowbane in Iowa. Statistics in regard to such cases are, however, difficult to obtain.

H. W. Cattell, as senior coroner physician in Philadelphia, performed 799 postmortems, in 155 of which, death was due to poisoning. The poisons used were listed as follows: aconite, 1; ammonia, 1; arsenic, 5; carbolic acid, 10; chloroform, 1; creosote, 1; cyanide of potassium, 1; hydrocyanic acid, 2; illuminating gas, 12; lead, 1; oil of merbane, 1; opium, 11; oxalic acid, 1; phosphorus, 1; silver nitrate, 1; stramonium, 1; strychnin, 2; sulphuric acid, 1.

In his work on poisons, Blyth states that the deaths from poisons in England and Wales during the ten years ending December, 1903, were 11,035. Deaths from laudanum were 1,505; cocaine, 12; atropin, 96; prussic acid and oil of almonds, 328; potassic cyanide, 207; strychnin and nux vomica, 244; aconite, 45; alcohol, 87; petroleum, 23; belladonna, 95; cocain, 12.

STATUTES ON POISONING AND ACTION OF POISON ON DIFFERENT ANIMALS.

The statutes do not as a general rule define poisons, but in most of the codes the sale of certain poisonous substances is regulated by law. The following extract from the Iowa code illutrates this: Sale regulated of substances under Schedule A. Arsenic and its preparations, corrosive sublimate, white precipitate, red precipitate, biniodide of mercury, cyanide of potassium, hydrocyanic acid, strychnia and other poisonous vegetable alkaloids and their salts, essential oil of bitter almonds, opium and its preparations, except paregoric and other preparations of opium containing less than two grains to the ounce. Schedule B. Aconite, belladonna, colchicum, conium, nux vomica, henbane, savin, ergot, cotton root, cantharides, creosote, digitalis, and the pharmaceutical preparations, croton oil, chloroform, chloral hydrate, sulphate of zinc, mineral acids, carbolic acid, and oxalic acid.

Not all poisons act in the same way, some acting more quickly than others. Quality and quantity are prime factors in the results obtained. As an illustration of this fact, we may mention ricin which is obtained from the cas-

tor oil bean, one gram of which, if properly diluted, is estimated as sufficient to cause the deaths of 1,500,000 guinea-pigs. The characteristics of the animal affected by the poison is also an important factor in the result. For instance, a fatal dose of strychnin in case of ruminants, when given by mouth is varying; when given hypodermically, it is a little larger than for horses; the minimum fatal dose for a horse being 1½ to 3 grains when given hypodermically, and 3/5 grains (or ½ ounce of nux vomica) when given by mouth, but as much as 2 grains is permissible.

The snail is said to be capable of withstanding more strychnin than an adult man. The minimum dose for man is ½ a grain, while 4/7 grains constitute a lethal quantity.

Cardiac poisons produce no action upon insects. The rabbit can stand more morphin than a man. Kobert says: "*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 on the toad." "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, *crotonilic acid* is the poison thus freed; in the second, ethereal *mustard oil*; and in the third, *hydrocyanic 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 intestines."

Persons may become accustomed gradually to the use of poisons. Thus individuals who consume opium or its alkaloids may take large doses without apparent injury, although children are particularly susceptible. The former statement is equally true of those who daily use such poisons as hashish, nicotin, caffein, cocain, alcohol, or morphin. They must have the drug in order to keep up their condition. Many people exhibit idiosyncrasies with reference to food substances or drugs. Some people cannot inhale the odor of morphin, turpentine, or tobacco without becoming ill. Others are uncomfortably affected if the flowers of the common bird cherry or the haw are left in the room. Others become sick when in the presence of the flowers of the tuberose. Coming in contact with the castor-oil plants sometimes causes illness.

CHAPTER II

BACTERIAL POISONS

Impure Water the Source of Disease.

In all ages great stress has been laid upon the value of a pure water supply. In ancient times, wherever there were great centers of population, a large amount of labor as well as of money was employed to furnish water. Of this the Claudian aqueduct, built in Rome in the year 50 A. D., is an illustration. Prof. W. P. Mason says: "Not only was a generous daily per capita allowance sought for, but we note in the centuries gone by unmistakable evidences of a keen appreciation of the dangers lurking in a polluted supply; and upon this point many of the ignorant consumers of our day and generation would be benefited did they consult the wisdom of the past."

Of the value placed by the ancients upon the quality of water, Prof. Mason also says: "In ancient times, the valleys of the Euphrates and Tigris, now almost a desert, were densely populated. Four thousand years ago the rulers of Assyria had converted those sterile plains and valleys into gardens of extreme productiveness by the construction of immense artificial lakes for the conservation of the flood-waters of the rivers, and as great distributing canals for irrigation. One of these canals, supplied by the Tigris, was over 400 miles long and from 200-400 feet broad, with sufficient depth for the navigation of the vessels of that time." "In India, tanks, reservoirs, and irrigating canals were constructed many centuries before the Christian era, and a great part of that country was kept in the highest state of cultivation. Some of the tanks or artificial lakes covered many square miles, and were often fifty feet in depth.

A great deal of interest has been manifested recently in all parts of the United States concerning water supplies. This has not been confined to the cities but the interest is manifested in the villages and rural districts as well. We are now demanding more than ever before, not only that a good wholesome supply of water be provided to the citizens of a city or village, but also that as good a supply be furnished the farmer. That such diseases as typhoid and cholera are water borne can not be doubted. Many others, as anthrax, hog cholera, and tuberculosis may also be conveyed by water. Animal parasites are also water borne. In addition to these, there are some poorly defined intestinal disorders that are caused by poor water.

That typhoid fever is quite as prevalent in the country as in the city admits no denial. A record of the cases of typhoid occurring during the fall and winter in any of our rural communities shows that the disease is as widely spread in the country as in the city.

A certain class of animal diseases is produced not by the invasion of micro-organisms, but is caused by the water supply being contaminated by the decomposing products of animals. The water may, for example, be highly charged with colon bacilli or other bacteria that produce poisonous products.

Several years ago Dr. Stalker traced an epidemic of horses, cattle, and pigs, all of which had been affected with similar symptoms, the animals uniformly dying after an illness of about two days. The disease was not contagious; the farm buildings were fairly comfortable and clean, and the trouble was, evidently, not due to the food consumed by the animals on the farm. Most of them, however, had been in the habit of drinking from a small creek which ran through the premises. The stream was supplied by a series of springs, and in ordinary seasons flowed for a portion of its course over a gravelly bed. This season the rainfall was light, and it so reduced the supply of water that it ceased to flow. Investigation made on these premises and on the adjoining farms indicated that dead animals were thrown down the steep bluffs into the bed of the stream. During the summer, chickens which had died from cholera and hogs dead from hog cholera had been dumped into the creek. In addition, the creek received the drainage from manure heaps. This was the kind of water that these animals had had to drink. Stock which did not have access to the creek but were watered from a well escaped the disease, while stock on other farms having access to the creek water suffered from the disease.

Dr. Lewis and Mr. Nicholson, in Bulletin 66 of the Oklahoma Agricultural Station, refer to certain troubles of live stock due to faecal contamination. In many cases the pond from which stock is watered is situated where plowed debris is carried into it by heavy rains, partially or completely filling it up, while the stock tramping down the banks soon complete the process. Stock standing in the pond also foul the water with excrement, and in hot weather, when the water is low, such a pond certainly can not afford a very satisfactory water supply. During the winter and spring months, when the rainfall is abundant, this condition is not so noticeable since the water is being continually changed by fresh water running in.

One of the dangers that follow allowing stock of all kinds to stand in a pond is that when the water is at a low stage, and foul, as it becomes in summer seasons, the cattle will not drink a quantity of the hot, foul, surface water, sufficient to prevent certain derangements of the digestive system such as impaction, "dry murrain," and other conditions that are usually ascribed to dry feed, but which are, in a large measure, brought about by insufficient water.

A type of injury resulting from the use of polluted water is illustrated in volume 19, page 74, of the "Journal of Comparative Pathology and Therapeutics." This record is in the form of evidence given in a case in which the plaintiff is the tenant of a farm on which is kept a dairy herd of from 30-35 cows. In 1903, there was no complaint but in 1904 the cows were put to grass in the middle of May and their condition became unsatisfactory at the end of July. Early in September, one of the cows aborted, six others lost their calves between that date and the 7th of October. On the 19th of November, another cow aborted; seventeen of the remaining ones carried their calves the full term and four were barren. The cows drank water from a small lake about one and a half acres in extent, which the town council of Maybole, who were the defendants in the action, used for the deposit of rubbish from the town. About the 18th of October, the cows were removed to another pasture with different water supply, and only one cow slipped her calf. (This occurred Nov. 19). The expert testimony was very conflicting. The plaintiff and expert

testimony held that septic poisoning resulted from the use of this water and thus caused abortion, Prof. Williams maintaining that water holding a large amount of vegetable matter is dangerous to pregnant cows, while the defendants held that this would not be a sufficient cause for the action. Judgment was rendered for the plaintiff.

Bacterial Poisons.

Bacterial poisons are produced by two classes of bacteria: the first includes such as are parasitic or pathogenic; the second, those which form poisonous products by the breaking down of dead animal or plant tissues. An illustration of the first class is seen in the products resulting from the tetanus bacillus and diptheria bacillus which produce an extra-cellular toxin. Another type is the toxin known as endo-toxin. In the extra-cellular form, toxin exudes through the bacterial cell-wall and is found in the body; while in the endo-toxin form, the toxin remains wholly, or in part, in the cell during the life of the organism and is liberated only on the death of the bacteria.

There are on record numerous cases of poisoning as a result of eating certain foods of animal origin, and the same statement may be made in regard to foods of plant origin. Such foods as meats, fish, cheese, and milk, sometimes become injurious because of the products of bacterial growth which they contain. These products are classified as either ptomains or toxins; a third class, the leucomains, result from the breaking down of tissues of the living animal body, being proteid bodies which have been broken down by enzymes, secreted by the cells of the body. These leucomains produce auto-intoxication. Ptomains are soluble, basic substances formed by the action of bacteria on protein material. Dr. Holland illustrates the action as follows: "The amino-acids, ornithin and lysin, constituents of pure protein, subjected to bacterial action, split off CO_2, and change to putrescin and cadaverin." Some of these products, as methylamin, are harmless, while others are active poisons. The ptomains are strongly basic, combining with acids to form salts. They are precipitated with chlorides of mercury and are of various kinds. Some are free from oxygen, while others contain that element; some, as typhotoxin, tetanin, pyocyanin, are unclassified; several are injurious in foods; some are produced in fresh oysters and mussels.

Ptomain poisoning. Symptoms: Gastro-enteritis is the most prominent symptom, with depression and nervous disturbances. In most cases, there are, also, marked thirst, salivation, nausea, and vomiting, diarrhoea, cramps in the legs, great prostration, feeble pulse, dilated pupils, delirium, paralysis, and collapse. The postmortem examination generally, but not always, shows inflammation of the stomach and bowels. Cholin, in large doses, nervin, diamin, amanitin, muscarin, all act as poisons; nervin is much more powerful than cholin, the symptoms being those accompanying obstruction of the bowels, together with nausea, pain, and depression; the diamins are all actively poisonous, dilated pupils, convulsions, diarrhoea, and paralysis being prominent symptoms. Muscarin, found in Fly Agaric and certain putrid products, is a much more powerful poison than cholin or nervin and produces vomiting, griping pains in the stomach and intestines, slow pulse, arrested action of the heart, contraction of the pupils and fatal collapse.

Toxins. These are poisonous bases produced by living bacteria or by saprophytic bacteria in the animal body and in higher plants.

Holland arranges the food toxins in two classes: (1) The poisonous

CHAPTER III

DERMATITIS

Skin diseases are produced by a variety of causes, some resulting from pathogenic organisms and others through the ingestion of food. On account of these differences, we have two classes sometimes given: parasitic skin diseases and urticarial diseases. Under the second class are placed such eruptions as those produced by buckwheat and smartweed, known technically as *fagopyrismus* and *rhus venenata* (*dermatitis*), and *urticaria*, the latter being produced by a large number of plants, especially the nettles. Or there may be internal causes due to innervation of vasomotors. Of the parasitic skin diseases known under the general name of *dermatomycoses*, we have several types, the co-called *Tinea tonsurans* and the *Favus* organisms, the former occurring in cattle, dogs, horses, sheep, swine, and poultry.

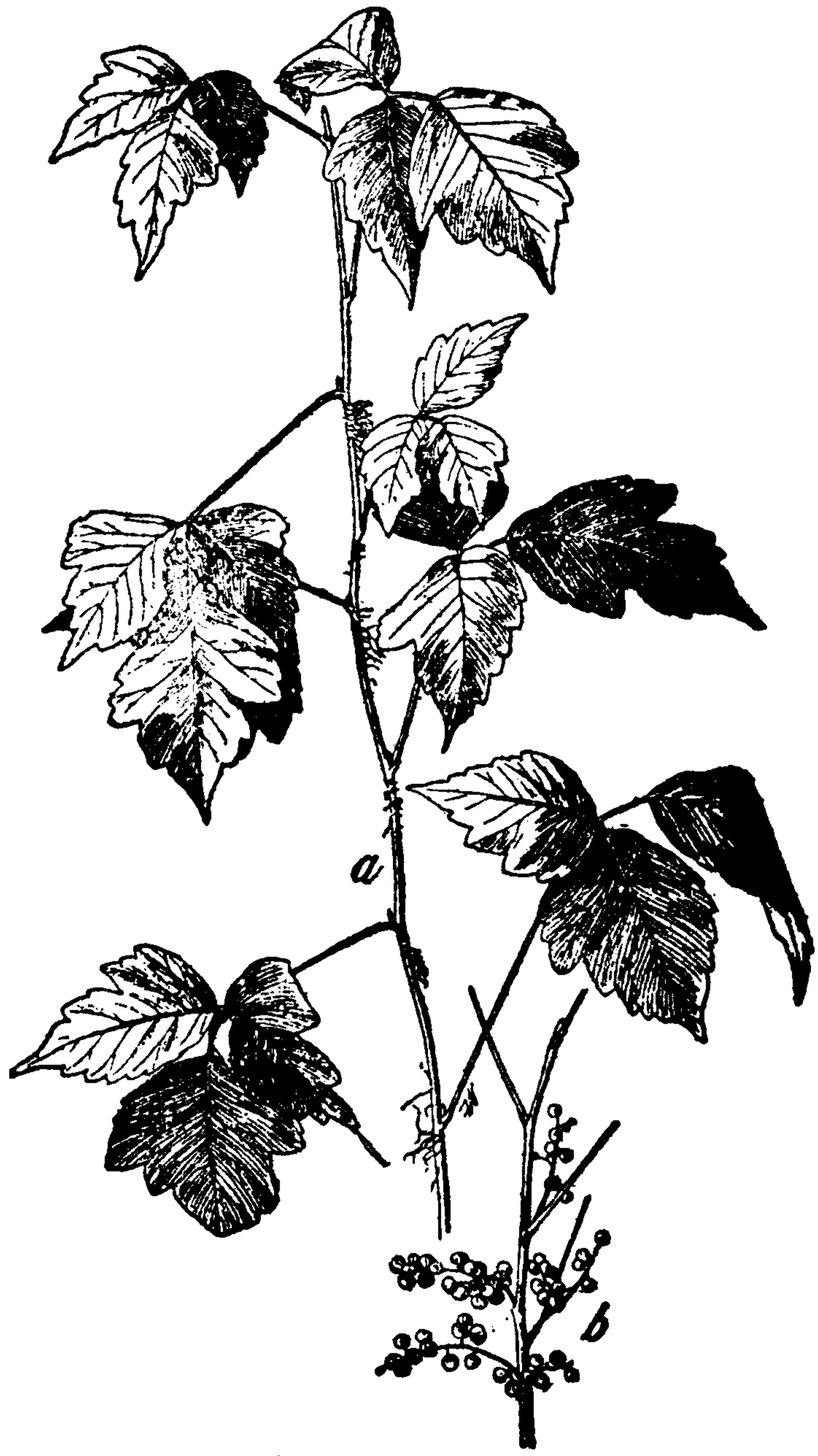

Fig. 1. Poison Ivy (*Rhus Toxicodendron*) cause of dermatitis or Rhus poisoning (U. S. Dept. Agrl.).

Dermatomycosis. This term is derived from two Greek words meaning skin and fungus. The classification of the fungi concerned is not at all satisfactory; at present, however, they are generally included in the groups known as *Fungi Imperfecti*, the *Mucoraceae* and *Ascomycetes*.

The Fungi Imperfecti include a large group of fungi whose life history has not been worked out completely. The fungi of this class are form-genera, such as the favus fungus. The so-called *Achorion* and *Trichophyton* of various authors represent such form genera, the *Oidium albicans* being another type; of these, some, perhaps, never produce any other kind of spore than the one commonly seen.

Many of these genera undoubtedly belong to the Ascomycetes, in which the spores are produced in little sacs called asci, the spores being known as ascospores. A kind of ringworm of the dog (*Eidamella spinosa*) belongs to

this group, also the forms that produce aspergillosis, one type of which occurs in the ear.

The *Mucoraceae* produce an unsegmented mycelium and septa only where the reproductive bodies are formed; the spores usually occur in sporangia, or occasionally small spores may be found in the mycelium; zygospores which result from fertilization also occur in some species. Several species produce surface lesions.

According to Neumann, the dermatophytes of domestic animals belong to six distinct genera of fungi as follows: Trichophyton, Eidamella, Microsporon, Achorion, Lophophyton, and Oospora. These genera are not, however, all accepted by botanists.

Trichophyton was established by Malmsten in 1848 and is characterized by having a mycelium consisting of simple or dichotomously branched filaments and producing spores from 4-9 μ long. Sabouraud, however, divides the genus into several species depending on the position of the fungus with regard to the invaded hair. The *T. endothrix* lives inside the hair; the *T. ectothrix* develops outside of the hair, forming a sheath around it, and the *T. endo-ectothrix* develops both inside and outside the hair. This classification is scarcely tenable from either clinical or cultural characters. We have placed this genus with *Sporotrichum.*

The *Eidamella spinosa* described by Matruchot and Dassonville in 1901, has a much branched mycelium, 1.5 μ in diameter, divided into short segments and splitting into somewhat squarish oval bodies; it is found on the dog.

The *Microsporon* discovered by Gruby in 1843, has a branched mycelium, the latter branches bearing conidia from 2-3 μ in diameter. This fungus has also been placed with *Sporotrichum.*

The *Lophophyton*, described by Matruchot and Dassonville in 1899, produces a mycelium with some tortuous filaments, others short curved, with thick curved walls; no spores produced; it occurs on fowls and is also referred to *Sporotrichum.*

The *Achorion* was described by Remak in 1833. The filaments of the mycelium are from 2-3 μ in diameter, flexuose or straight, variously branched; finally breaking up into spores. This fungus has been placed with the genus *Oospora.*

Oospora was described by Wallroth in 1833. Its mycelial threads are 2-3 μ in diameter, arranged in irregular chains.

The best expert account of dermatomycosis so far as it affects lower animals will be found in the treatise by Neumann[1] who includes also an excellent bibliography on the subject. Hutyra and Marek have a German text which devotes considerable space to the subject. The work by Hyde and Montgomery treats the subject from a human standpoint. The work by Plaut is also an exhaustive treatise.

Etiology. The cause has been ascribed to various fungi which will be described later in the present work. The predisposing causes are uncleanliness, weakness of animals (those that are worn out may offer a favorable medium for the attacks of the fungus). In the case of cows, Fleming observes that the disease is common in the winter when the stables are dirty, and disappears in the spring when the animals are turned out to

1 "A Treatise on the Parasites and Parasitic Diseases of the Domesticated Animals." English Translation by George Fleming. Second Edition Revised and Edited by James MacQueen. 697. *365 f.* 1907. New York.

pasture, the new condition being opposed to contagion. In the case of the cat, early age seems to be an essential condition. In the case of the rabbit, a similar condition seems to hold true. In the dog, early age is not an especially predisposing influence but inoculation is successful in young dogs only, according to Horand, so far as trichophyton is concerned. This latter statement does not hold true for favus.

Contagion. In ringworm of the horse, infection may occur from horse to horse. Mégnin states that in one locality 200 horses became infected in this way, a saddle from an infected horse having carried the disease to other animals. In each case, the disease occurred on the left side of the back. It has also been transmitted from an ox to a horse. The tinea tonsurans has been transmitted from the horse to calves, and the tinea of the horse from horse to dogs, sheep, and pigs, and even to man. Neumann says: "The infection of man is exceptional when the frequency of tinea tonsurans in the horse is considered, as there is scarcely a regiment in which it is not always on some young horse." Grooming is the usual way in which the infection is carried and rubbing facilitates inoculation. The ease with which infection occurs on man depends on the character of the fungus, some forms adapting themselves to the conditions present more readily than others. In the case of the bovines, the contagion may be direct. The virus may be preserved a long time in parts of stables where calves were affected with the tinea tonsurans. The infection spreads less readily to sheep and pigs but may be transmitted from bovines to man. In the case of the dog, it is transmitted from dog to dog, from rats and mice to the dog, and, occasionally, from dogs to man. In cats, favus is largely transmitted from mice and it is certain that this form can be transmitted to man. In general, it may be said that the transmission of favus from the rat or mouse is frequently brought about through the domestic cat. The tinea of the fowl is transmitted by contact with a diseased fowl. The favus fungus of the fowl[1] cannot be inoculated on the rat or dog but when inoculated on man, it produces lesions similar to favus. Man may be inoculated very easily by handling a fowl on which large erythematous patches occur. Similar patches have occurred in man when inoculation from a fowl was very probable.

Symptoms. Two forms of skin dermatomycosis in the horse have been recognized: (1) called microsporosis, and (2) trichophytosis. The more important symptoms of the first as given by Neumann are: "It appears in patches which are more especially seated on the upper part of the body — on the shoulders, back, loins, croup, sides, and flanks. These patches may, however, be met with on any part of the body, though they are rare on the lower parts of the legs. What are first noticed are the circular patches, the diameter of which is generally about that of a shilling; they are distinguished from the healthy skin by the dullness and erectness of the hairs covering them. Some time before the circular patches appear, a very small tuft of hairs — probably from half a dozen to a dozen — may be seen slightly, but markedly, raised in the form of a fine pencil, and feeling as if they had a somewhat hard base, or were matted together at the bottom, when the finger is passed over them. These tufts may be several in number, and are usually best seen in hindquarters at the very commencement of the disease, or in the vicinity of the patches, of which they are the initial symptom. The hairs fall off in a

1 Lophophyton gallinae, Mégnin — Trichophyton Mégnini, Blanchard. — *Sporotrichum.*

few days, and this is often the first symptom that attracts attention. The epidermis of the patch falls off at the same time as the hairs; it appears to be softened, and the surface of the skin has then a dark-grey tint and is slightly moist, which might be attributed to the rupture of vescicles, though their presence has never been demonstrated. It cannot, therefore, be said, as Raillet remarks, that the disease presents itself in the form of *herpes,* as what is so called in human pathology includes a phase marked by the appearance of vesicles. The humidity of the patch is ephemeral. Its surface generally soon dries, and is covered with epidermic scales of varying thickness, which are agglutinated into flat crusts that are shed and renewed incessantly. These crusts have—more frequently than in the ox—a shinning appearance and a grey or yellowish color like flax. At the same time, the lesion progresses by peripheral extension until it attains the diameter of a five-shilling piece or more, and on each zone invaded, successive symptoms are observed. Pruritis is nearly absent in Microsporosis, and is scarcely even shown to exist, except by movements indicating satisfaction on the part of the animal when the patches are gently scratched.

"2. Trichophytosis (*Sporotrichum*). A.—*Trichophyton flavum.*—The lesions consist of large patches, at least 8 to 10 cm. broad, of a more or less regular form, greyish, and quite smooth. The hairs, raised and matted at their base by a greyish-yellow crust, fall away very rapidly with the crust. The naked surface is not prominent, and shows no trace of suppurative folliculitis (Bodin).

"B. *Trichophyton equinum* occurs usually in numerous patches, some isolated, scattered over the croup and shoulders, and attaining at the most 3 cm. in diameter. At first they can be detected only by touch, but later they become visible by the flattening of the hair. The least traction or slight friction carries off a scaly crust which brings away the diseased hairs. The skin then appears smooth, moist, pinkish, or light grey. Very soon the patch becomes dry, scaly, or powdery, and at its base a slaty grey. The lesions spread by the falling out of the marginal hairs (Matruchot and Dassonville).

"C. *Trichophyton verrucosum,* var. *equi,* occurs in numerous patches which average 5-6 cm. in diameter, and are localized on the shoulders, neck, withers, flanks, in fact, everywhere where the harness touches. By their confluence, these patches may produce large, irregular lesions. At first the hairs are raised, not broken, and are matted together at the base by a soft grey crust. This crust falls off in less than a week, carrying away the hairs, and having an absolutely bald, grey surface covered more or less with dry, greyish scales, and without follicular suppuration. In young horses, however, the shedding of the crust leaves a vesicle or pustule, then the surface of the patch is slightly raised, red, and indurated (Bodin).

"D. *Trichophyton verrucosum,* var. *asini.*—The lesions are the same as in the preceding Trichophytosis of the Horse, but are generally confined to the neck, head, and ears (Bodin).

"E. *Trichophyton mentagrophytes.*—This Trichophytosis occurs usually on the nostrils or head. It forms patches up to 5 or 6 cm. in diameter, which may be mistaken for pustules of horse-pox undergoing regression. Over the whole of these patches the hairs are matted together at their base by a soft, brownish crust of unequal thickness. Slight traction on the hair brings away the crust, exposing a bare, slightly raised surface, which is red, inflamed, and pitted with

small depressions, grey at their base. These result from the opening of the pustules, of which some may be found at the margin of the patch. The hairs are not broken, but shed, and the condition is, in fact a suppurating folliculitis."

The symptoms observed in the bovine, as described by Neumann, are as follows: "The commencement is manifested by a slightly salient ring, on the surface of which the hairs are erect. An active proliferation of the epidermis causes the rapid formation of scales more or less adherent to each other, and crusts of 2 mm. to 7 mm. thick—hence; the name *dartre crouteuse* was given to the affection by the older (French) veterinarians. According to Gerlach, the crusts are thicker on dark skins, on which they have a greyish-white, fibrous appearance, resembling the amianthus (*porrigo asbestinea*) on white skins, which are usually finer, the crust is thinner and a little yellow in color."

Diagnosis. The diagnosis based on clinical symptoms should be verified by microscopical examinations. It is best to take material from the younger and deeper parts of the crust which may be moistened with water, or a better examination can be made if it is boiled with a 40 per cent solution of potash after which the particles can be dissected and the fungus threads and spores made out. The different forms cannot readily be distinguished except by cultural methods. The organism grows readily on nutrient media that are neutral or with slightly alkaline reaction. Sabouraud recommended the following:

Pure glycerine, glucose, lactose, or maltose.....	4	grammes
Granulated petone.....	1	gramme
Distilled water.....	100	grammes
Gelose	1.50	grammes

Solid media like potato, agar, and peptonized bouillon are favorable media while liquid media are less favorable. Growth may occur at 15°C, the optimum being 30°C.

Prognosis. The duration of the disease depends on circumstances, it gradually diminishes and may disappear without medical aid. The average length of time of the disease is 40-50 days. Cleanliness has much to do with its disappearance. It lasts longer in thick coated animals than in those with thin coats.

Treatment. Cleanliness and sanitary surroundings, disinfection of stables, careful and regular grooming (all articles used in this process having been thoroughly disinfected, especially when they have previously been used on a diseased animal), avoiding any substance that causes irritation. The following preparations have been used with success: Mercury 1-500; carbolized glycerine; alcohol; an ointment composed of 1 part of carbolic acid, hard and soft soaps, each 20 parts. Fourie and La Calve recommend pure carbolic acid, tincture of iodine, and chloral hydrate in equal parts. The applications should be made once or twice a day or every other day depending upon the irritant properties of the preparations used.

For bovines, the remedies named above will prove efficacious; for the dog, application of an ointment prepared from 1-5 per cent of nitrate of silver, is satisfactory.

Favus

Dr. Stuhr has contributed the following account of favus in animals: "Favus is a contagious, vegetable-parasitic disease of the skin, characterized by the formation of more or less circular, cup-shaped crusts, varying in size from very small up to that of a dime. It has been observed in almost every species of domestic animals. The disease is quitc rare among horses and cattle although dogs and cats are frequent sufferers. The domestic fowl and pigeon are also susceptible.

Of the laboratory animals, mice, rats, rabbits, and guinea pigs, harbor the

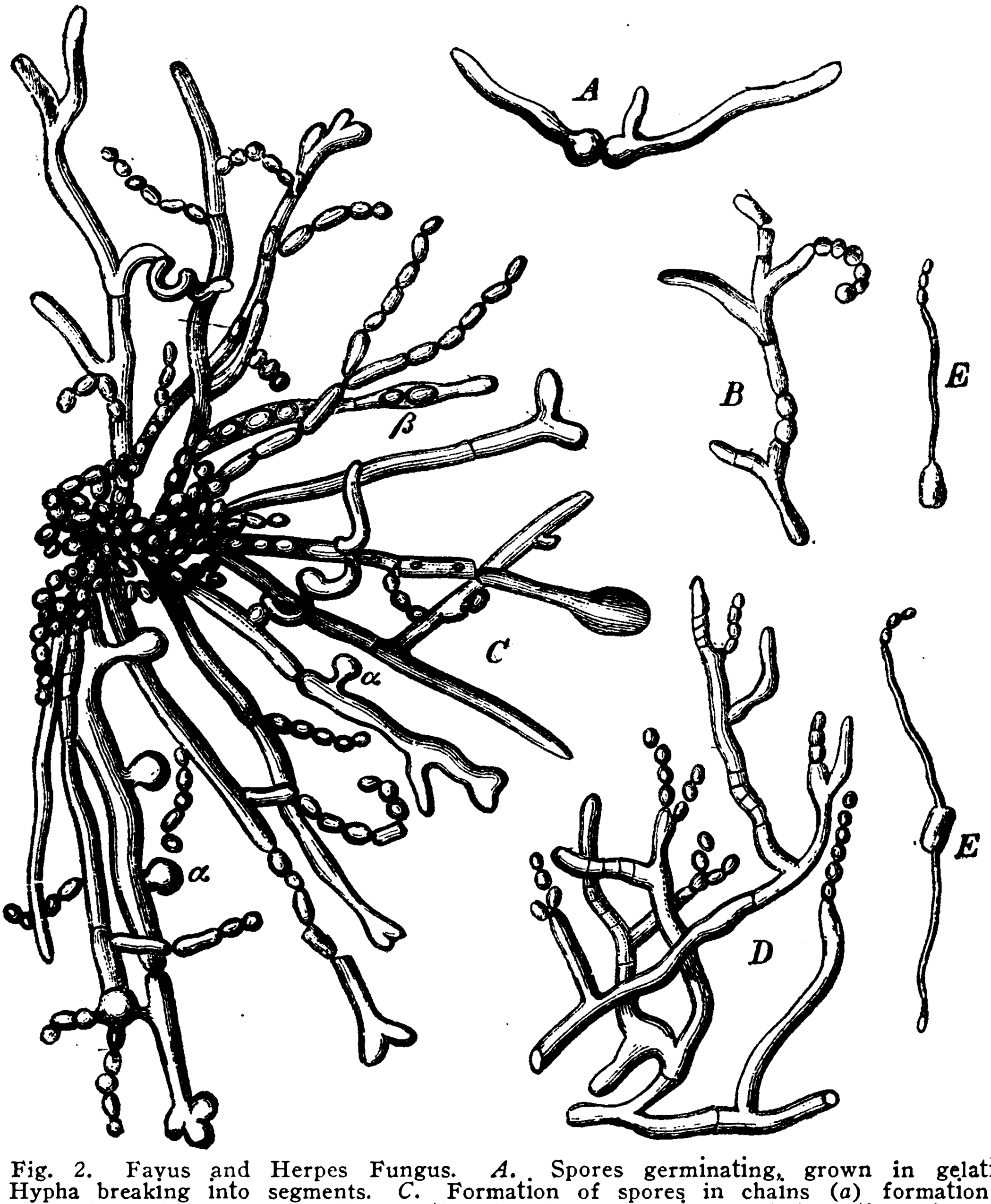

Fig. 2. Favus and Herpes Fungus. *A.* Spores germinating, grown in gelatine. *B.* Hypha breaking into segments. *C.* Formation of spores in chains (*a*) formation of buds (*b*) chlamydospores. *D.* Herpes, threads of the mycelium and formation of spores. *E. Oidium lactis* spore with germ tubes. After Grawitz.

disease. Young age, thin skin, and debility predispose. Favus is communicable from lower animals to man and vice versa. Man frequently contracts the disease from cats, the latter becoming infected from eating mice and rats.

Etiology. Favus is caused by a vegetable parasite, (*Oospora porriginis*) which invades the cutaneous structures, especially the epidermal portion. The *Achorion Schoenleinii* was first discovered by Schoenlein in 1839,

although Remak was the first to demonstrate its pathogenic character by direct inoculation. It consists of mycelium and spores, existing in such profusion that it is readily detected. Skin abrasions are an important accessory cause.

Symptoms. The disease is characterized by dry scabs, brownish or yellowish, gray or silver white on the surface, and white or sulphur yellow in their deep layer. These scabs have a circular form, with a diameter not to exceed that of a dime, and a thickness varying up to one-fifth of an inch. They occasion atrophy of the hair and a slight depression of the skin. These scabs are usually found on the forehead, cheeks, ears, face, abdomen, external side of the hind legs and in the neighborhood of the claws. At first the scabs are perforated by hairs which soon fall out. Later the skin exfoliates under the scab and leaves a pit. In the horse the scabs may become confluent and form bands as wide as the finger. Itching is observed in the dog. In most cases the progress of the disease is quite rapid although the prognosis is favorable unless the disease has become too far advanced.

Lesions. According to Robinson the parasite first obtains a lodgment in the funnel-shaped depression in the epidermis, through which the hair shaft emerges upon the surface. It grows luxuriantly in the upper part of the hair-sac and insinuates itself on all sides between the superficial layers of the epidermis. When it reaches a short distance on all sides of the hair follicle it breaks up the looser layers and appears on the surface producing the characteristic cup-shaped bodies. It also invades the hair shaft itself, penetrates between the cellular layers of the root sheath and by its mechanical pressure upon the papillae interferes with the nutrition of the hair and causes it to fall out. If the pressure is sufficient to cause atrophy of the papilla, a new growth does not occur.

In the skin the parasite usually confines itself to the upper corneous cells and does not extend to the living tissues. In cases where the surface is covered by irregular confluent masses of the parasites, the entire upper layer of the epidermis will be found infiltrated with the achorion.

The corium itself is usually in a state of chronic inflammation, and suppuration, which may be quite abundant, often occurs under crusts. Even in the absence of pus, the pressure of the parasite causes atrophy of the skin, and at last pit-like depressions or more extensive reddened scars are left. The disease ends with the destruction of the glandular structures of the skin.

Treatment. This is purely local except when debility complicates the disease, in which instance tonics should be administered to build up the system. As for the local treatment, its aim is to destroy the parasite and relieve the cutaneous irritation. The dry scabs may be softened and removed by thoroughly washing with soft soap and water. The removal of the hairs, by extraction, from the affected part has been attended with good success since, in so doing, many of the parasites are disposed of. A liniment composed of liquid tar and green soap two parts and alcohol one part will prove beneficial since it is antiparasiticide, disinfectant, dessicating, emollient and cleansing.

Mercuric chlorid in one to two per cent aqueous solution tincture of iodine, sulphur iodid ointment, red iodid of mercury ointment 1-8, sulfur ointment, etc., are all useful applications. It is advisable to clip the hair from unaffected parts adjacent to the diseased foci, so that any spread of the malady may be immediately detected. Whatever the treatment, it is a good plan to wash daily with soft

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CHAPTER IV

FORAGE POISONING, ERGOTISM, AND ASPERGILLOSIS

Forage Poisoning

We have several excellent illustrations of how other external known parasitic organisms may produce disease. Catarrhal stomatitis, for instance, may be produced by the ingestion of fodder which has become infected with any one of several fungi belonging to distinct orders. Among these are the rust of clover, bacteria, mildew of grass, and the rape-destroying fungus, *Polydesmus exitiosus*; even the common grass rust and other rusts upon grasses as well as the bunts and smuts are known to produce this form of disease. Among higher plants, such products as the pungent spices of pepper and of the roots of horseradish and radish are treated at length in such pathologies as the Friedburger and Frohner Veterinary Pathology.

Serious diseases of the stomach are caused not only by pathogenic germs but also by the ingestion of various foods. Many foods, such as unclean, or damaged fodder, poor water, musty hay, mouldy corn, decomposing potatoes, are responsible for gastro-intestinal catarrh; many fodders, also, contain irritant substances. There are several forms of gastro enteritis. Among forms of the third class (including those caused by ingestion of lower organisms such as fungi or poisonous substances) we may mention botulism, fish poisoning, injuries produced by mould fungi, smuts, rusts, and, finally, the so-called toxic gastro-enteritis produced by numerous poisons. These have sometimes been classed as irritant poisons and narcotic irritant poisons. The vegetable poisons under this head are numerous and have been treated under the different plants. Some pathologists, however, mention especially lupinosis of sheep and equisetosis.

The terms applied to this disease are Cryptogamic Poisoning, Forage Poisoning, Enzootic Cerebritis, Epizootic Cerebro-Spinal Meningitis, Leuco-Encaphalitis, etc.

Characterization. So-called forage poisoning among horses and mules is a non-communicable disease, which undoubtedly belongs to a group of cryptogamic poisonings. Horses seem to be slightly more susceptible than mules, although it usually terminates fatally in both species.

The disease is characterized by symptoms which are referable to a disturbance in the central nervous system, and by lesions which, if present, are also found there. The course of the disease may be very acute, or it may be greatly lengthened, depending upon the suddenness of the onset. The mortality is very high and but few well developed cases ever recover. Suckling foals do not contract the disease.

History. This disease has prevailed quite generally throughout the Eastern and Central parts of the United States for many years, but until recently has not attracted any considerable attention. During the past few years, however, it has occurred with unusual frequency in the Central West, and, because of the extensive losses directly attributable to it, has

become of great economic importance. In various parts of Iowa, for instance, individual stock-owners have lost several thousand dollars from its ravages. In the different localities the disease has been known by various names, such as "grass staggers," "choking distemper," and "putrid sore throat," and because it apparently presents some of the distinguishing characters of a specific infectious disease, has been frequently recognized as "infectious cerebro-spinal meningitis."

A noteworthy fact however is, that thus far no evidence has been discovered which would indicate that the disease is transmissable from animal to animal, or that it is even inoculable.

On the other hand, an outstanding feature in every outbreak is, that the affected animals have had access to unwholesome food, either while at pasture or in the stable.

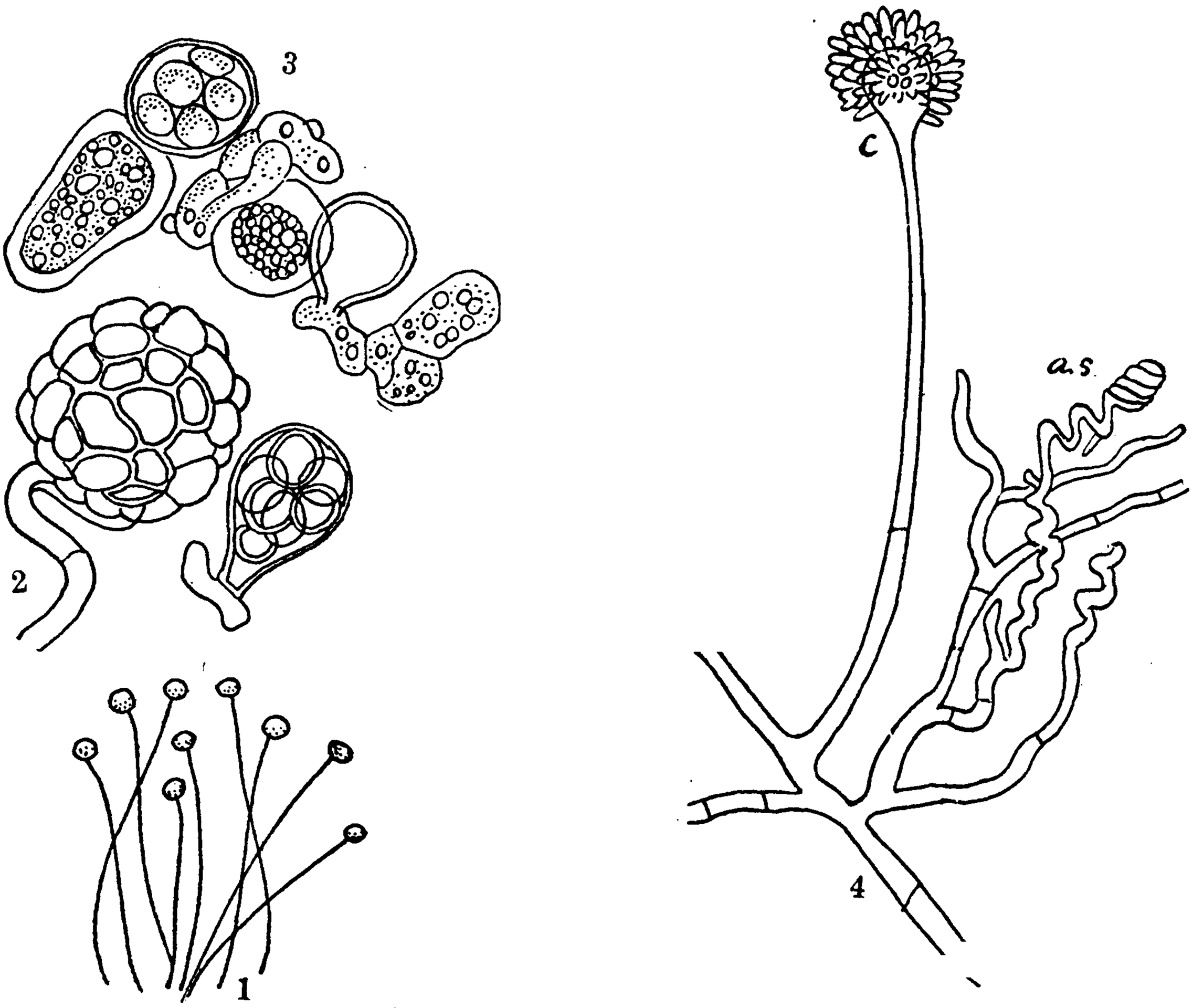


Fig. 3. Common Aspergillus on mouldy corn. 1. General appearance, showing long conidiophore and sterigmata on end. 2. Perithecium with one of its asci and ascospores. 3. Contents from an unripe perithecium. 4. A small part of the mycelium with conidiophore *c* and spore bearing sterigmata; young ascogonium *as*. 2, 3, 4 after DeBary.

Geographical distribution. The disease has been reported from nearly every part of the United States. It never becomes epizootic, but is usually confined to isolated localities.

While forage poisoning is not necessarily peculiar to low, poorly drained districts, it is at least most frequently observed in those places where conditions are most favorable for the development of cryptogamic growth.

Etiology. The disease seems especially likely to appear when horses or mules are fed on grain or fodder which has become overgrown with

moulds, or when at pasture, they have had access to grass which, for various reasons, has become fermented or mouldy. Various micro-organisms have been found to be associated with the disease, but as yet none have been proven to possess any etiological significance. Cultural and histological studies have all proved negative. Dr. Moore has in one instance succeeded in obtaining a pure culture of the colon bacillus from the brain.

Symptoms. Depending upon the severity of the attack, the disease may manifest itself in any one of three forms, namely; acute, subacute and abortive. It is possible to observe all of these forms in a single outbreak, as the suddenness of the onset is apparently regulated by the amount of the poison laden food which the animal has ingested.

The acute type is characterized by the abruptness of its appearance, and the grave general disturbances which immediately manifest themselves. There is sometimes violent trembling and twitching of the muscles over the entire body, but most commonly the acute form is ushered in by stupor.

There is manifested a weak, staggering gait and the pharynx is either partially or completely paralyzed. The tongue may also be partially paralyzed and protrude from the mouth, and saliva falls in strings from the lips. The pupil is dilated and the conjunctiva is, as a rule, highly congested. The pulse is variable and may be very rapid and hard, or scarcely perceptible; the respiration is hurried and jerky. The temperature may be slightly elevated, but is most frequently subnormal. Intestines and bladder are paralyzed.

In this form there may be slight muscular rigidity affecting the muscles of the back, neck and jaws, although in many cases this symptom never manifests itself. There is no rigidity of the ocular muscles. The animal soon becomes so weak that he is no longer able to support himself and falls. Delirium may manifest itself, in which the patient may perform a series of movements as if trotting, or become so violent as to do himself serious injury, but most often coma and complete paralysis supervene and death results in from four hours to two days from the commencement of the attack.

The subacute form is much the same as the preceding, except that it developes more slowly and the symptoms are not so violent. It is first noticed by a slowness in mastication and a difficulty in swallowing. A further indication of approaching paralysis is seen in the frequent knuckling and the loss of control over the tail. The temperature is subnormal and the pulse and respiration are but slightly altered. The bowels and bladder are inactive and it is seldom that voidance of urine and faeces occurs voluntarily. There is but slight rigidity of the muscles if indeed there is any, and no evidence of pain is apparent. These symptoms may last two or three days, when gradual improvement takes place, or the paralysis becomes more complete, the general weakness more marked, paroxyms of delirium develop, with inability to stand, breathing becomes more labored, coma comes on and death results apparently without a struggle. This form lasts from six days to two weeks.

In the abortive form there are no well marked constitutional symptoms. The appetite may be somewhat lessened, the ability to swallow slightly impaired, and the animal's movements a little uncertain, but no very noticeable symptoms appear to attract the attention. Improvement usually takes place on the third or fourth day, and recovery is the usual result.

Lesions. As a rule, post-mortem examination reveals no naked eye changes in the tissues of animals dead of forage poisoning. There

may be congestion of the brain and cord with extensive effusion into the ventricles and subarachnoid spaces. Few small hemorrhages and parenchymatous degenerations within the various organs have been mentioned. MacCallum and Buckley have found in the brains of horses dying of this disease, areas of softening "in the frontal region on each side, anterior to the motor region of the cortex." This lesion was practically confined to the white matter immediately under the cortex. In the affected areas there was "complete destruction of the brain substance, in which the anatomical structures are disintegrated and largely replaced by a colloid-like material. The neighboring blood vessels were acutely inflamed, with cellular infiltration of leucocytes and red corpuscles into the perivascular spaces and tissues. In a later outbreak these writers failed to find the brain lesion, but did observe the vascular changes above described.

McCarthy and Ravenel, in a study of fifteen animals found certain lesions in the upper gastro intestinal tract and in the central nervous system. These were:

(1) In the intervertebral and Gasserian ganglia, where a pericapsular, small round cell accumulation was present. The cells were all of the same type, the nucleus and protoplasm being about the size of a red corpuscle. There was no evidence that these cells were the result of proliferation of the original layer of capsular cells.

(2) Cortical lesions. — These consisted of congestion of the cerebellar and cerebral cortex. There were also capillary hemorrhages. The meninges were normal.

(3) Changes in the choroid plexus. — In three cases the choroid plexus was changed into a triangular tumor-like mass, of a yellowish red color and of a firm consistency. The increase in size was found to be due to a proliferation of the elastic tissue surrounding the vessels.

(4) Changes in the nerves. — There was a distinct degeneration of the nerves supplying the larynx and neck. This was present in the nerve up to the ganglion, but was not found in the posterior roots. Other slight changes were detected.

Moore failed to find any gross lesions in the nervous system and other organs in the cases examined by him. In one case the brain, spinal cord, and organs were studied histologically with negative results.

Differential diagnosis. A very important point in the recognition of forage-poisoning is the history which has been referred to previously. It must be distinguished from inflammations of the brain and meninges, and from rabies.

Treatment. In the acute cases this is seldom successful, although quick-acting stimulants to arouse the patient may be tried. In the subacute cases a purge should always be given to rid the intestines of the poison. Strychnin in large doses, to overcome the extreme depression of the nerve centres, and atropin to support a failing circulation may be administered hypodermically at frequent intervals with benefit.

In the very mild cases, all that is necessary is to empty the bowels with a purge.

It is of the utmost importance, in all cases, with the return of the appetite, to supply only such food and water concerning the wholesomeness of which there can be no question.

Prevention. Since it seems to be quite generally accepted that this disease is brought about by the ingestion of mould-contaminated food

the prophylaxis is apparent. Whenever the disease makes its appearance either in a stable or a pasture, the animals should be immediately removed from further exposure by changing the food supply. The food should come, preferably, from a clean, new source and the water should not be contaminated by surface drainage. It is also well to thoroughly disinfect the mangers and feed-boxes, and render inocuous the soiled litter.

There is no known means of artificial protection, and the disease will recur if the animals are again allowed access to spoiled food. (Stuhr).

Poisoning from Silage

History. During the winter of 1908-1909, several cases of poisoning from spoiled silage were reported to Dr. Stange of the Iowa State College. Other cases have no doubt been encountered. In every instance, as in the case reported by Dr. Beaumont, below, moulds occurred in the silage. Dr. Beaumont says: "I am sending you under separate cover by mail a specimen of corn silage upon which you will notice is growing some form of mould which in my opinion is accountable for a very peculiar disease, existing among a herd of young horses and mules belonging to a farmer living here."

Dr. R. E. Buchanan found these moulds occurring in spoiled silage to be a species of *Monascus.* Other moulds, *Mucor, Penicillium glaucum,* and *Verticillium* were also present; but there was a preponderance of *Monascus.*

Symptoms. "The first animal, a three year old filly, was taken sick about April 1st, showing symptoms as follows: Gaunt, depressed, stiffness of gait. When lying was unable to rise, but when assisted to rise would stand and show inclination to eat but was unable to masticate and swallow food. Temp. 103.5 F. Pulse 86, Respiration 36; friction sounds distinctly heard at each heart beat. A whistling sound was emitted during expiration and there was also a suppressed painful cough. Animal died in about five days.

"A two-year-old mule and one two-year-old filly were attacked with disease. The mule is improving and will recover but the two-year-old filly shows exactly the same symptoms as Case No. 1, aside from being especially stiff and lame in one fore shoulder, and I think will die within two days."

Treatment. The treatment as followed by Dr. Beaumont is described in detail in his paper before the Missouri Valley Veterinary Association, June 16-17, 1909. Briefly, the method was as follows:

Tincture Strophanthus in two-dram doses, every two hours (given as a cardiac stimulant, the heart action being very weak). 1 quart of raw linseed oil given in two doses, six hours apart (as general laxative). Potasii Nitras in half to one ounce doses, dissolved in water and given as a drench, every three hours (alterative diuretic, and respiratory stimulant). After the first twenty-four hours the Tr. Strophanthus was discontinued and he began giving Iron Quinine and Strychnin tonic in one-ounce doses three times daily. This was continued with the Potasii Nitras until the animal showed marked improvement when both remedies were discontinued and he prescribed Fowler's Solution (Liquor Potasii Arsenitis) in half-ounce doses three times daily during convalescing stage of the disease which lasted about ten days or two weeks.

Dr. C. H. Stange has contributed the following on forage poisoning and especially with reference to silage:

"Numerous cases have been reported of an affection of the central nervous system, the symptoms being in general quite similar but different and varying

causes are assigned. Dr. Francis reports that in the fall of '03, spring of '04, four to five thousand horses and mules died with a nervous disorder characterized by structural changes in the brain which cause incoordination, delirium, coma and usually death. He concludes that the disease is not caused by moulds but is the result of animals having free access to a labor diet when kept in idleness. He was unable to find the germ described by Wilson and Brimball.

"Professor Harrison of the Ontario Agricultural College reported several cases and as a result of his investigations he concluded that the disease was due to a coccus insolated from the meningeal fluids. Pearson studied an outbreak in seven horses, five of which died. The outbreak occurred soon after opening a new silo, the ensilage from which was mouldy. The symptoms observed were very similar to those observed by Professor Harrison and he emphasized the paralysis of the pharynx and great muscular weakness. He concluded as a result of feeding experiments that the so-called cerebro-spinal meningitis was a forage poisoning. Dr. Dow of Connecticut describes two cases which were attributed to watering from a tub containing a mouldy slime. Dr. Ferguson of Texas describes three cases of forage poisoning due to smutted corn. There was vertigo, coma, low temperature, pulse in later stages rapid and irregular. In 1901 Dr. Hickman investigated an outbreak among horses in North Carolina in which a large number of horses died. In 1906 another outbreak occurred at the same place (Hyde Co.) in which about forty horses and mules died in about three weeks. The cause in these cases seemed to be moulds on vegetation. On the whole the country is low and swampy. The pathological changes of Epizootic Leuco-Encaphalitis were described by McCallum and Buckley in 1902. Muller of Germany reported an outbreak among horses, cattle and sheep due to mouldy straw. (Berliner-Tierärztliche Wochenschrift). Drs. McCarrol and McMullen describe an outbreak of cryptogamic poisoning in horses due to feeding mouldy beet tops. Dr. Lockhart describes several cases in Canada. The prominent symptom seemed to be the inability to swallow.

"Two outbreaks have come under our observation during the past year. The first consisted of eleven head of horses, two horses were being fed for market, the others were fed in the same manner during the nights and turned out during the day. The first animal affected was one being fed for market. It ate part of its feed in the morning but in a few hours showed symptoms of ptyalism, depression and paresis of the hind quarters. By noon the animal was down, unable to rise and struggling some, and died that night. The next animal to show symptoms was its mate. The symptoms shown in this case were similar to acute cases of the so-called cerebro-spinal meningitis, coming on with trembling and weakness causing the animal to stagger. An early symptom in all cases coming under our observation is the ptyalism due to inability to swallow. (Dyspagia). As a result the saliva collects in the mouth and hangs from it in strings. Muscles of different regions of the body are liable to contract. The breathing is rapid and in some cases may be of the Cheyne-Stokes variety. The temperature in this case was sub-normal. In some of the more chronic cases and when the animal has been down for some time with considerable struggling the temperature was somewhat elevated. The pulse was variable, being about normal in some cases and rapid and almost imperceptible in others. The animal became quite violent at times and finally died living but a few hours longer than the first animal. The other seven animals showed

a more chronic course, showing inability to swallow, slow, weak pulse, difficult, noisy respiration, weakness and paralysis, spasm of muscles of head, neck and back, death taking place in from two to six days. The other two animals showed a mild type of the disease as slight loss of control, some exophthalmia, loss of appetite and thirst and loss of condition. These animals were placed on potassium iodid and nux vomica and recovered.

"This outbreak was attributed to mouldy silage, which was being fed to the horses, but in order to be more certain 150 pounds of silage were shipped to the college and fed, first to one horse which died in two days from an acute form of the disease. Another horse was fed but would not eat the silage so well, consequently did not die quite so soon, living for several days. In both cases the symptoms resembled those seen in the original outbreak. Post mortem revealed no changes except a few petechia along the small intestine, a few infarcts in the kidney and slight softening of the brain. This however was not very marked, probably due to the fact that they were acute cases. Microscopic examination revealed the presence of mould in the mucosa and submucosa of the intestine, also mycelial threads growing between and around the renal tubules.

"The other outbreak consisted of four horses, three of which died of an acute form of the disease, the symptoms being similar to those already described. The fourth being of a more chronic nature was placed on potassium iodid and nux vomica and recovered. In this outbreak the hay was found to contain a fine mould and was cut from an old pond which had been plowed up and seeded. The water had overflown this, however, and stood for some time. The symptoms and post mortems were similar to those described in the first outbreak, with the exception that no histological examinations were made.

"A form of cerebro spinal meningitis is quite common in Germany. It has also been described in Australia, Great Britain and Russia. It may be that these outbreaks are due to other causes than those already described. Sidamgrotzky and Schlegel found a form of coccus in the sub-arachnoid fluid, but it was necessary to make sub-dural injections of cultures of this organism to cause meningo encephalitis. Johne found a diplococcus in the cerebro-spinal fluid of affected horses.

"Ostertag found a diplococcus similar to the one found by Johne in the cerebro-spinal fluid in the so-called Borna's disease. They were pathogenic for horses and sub dural injections produced symptoms and death similar to cases of Borna's disease. Hutyra and Marek call attention to the fact that bacteriological investigations have not been followed by the same result but possibly the several investigators were working with the same organism. Nevertheless it remains to be shown whether all cases of cerebro-spinal meningitis are due to the same cause and resemble Borna's disease. On the other hand it is possible that epizootic cerebro-spinal meningitis of domestic animals has no specific cause.

"It is apparent that mouldy food and water has caused several outbreaks in this country. Natural infection in European outbreaks is also supposed to take place through infected food and drinking water. The disease is not transmitted from one animal to another. Mohler calls attention to the very interesting work of Schlegel and the Berliner Tierärztliche Wochenschrift who

associates with the affection an organism which he termed Streptococcus melanogenes. Mohler states, however, that whether the disease is of microbian origin or an intoxication has not yet been definitely established."

Ergotism

Ergotism is a disease of bovines caused by the ingestion of considerable quantities of food contaminated by ergot. Equines are apparently less susceptible than bovines, although the horses have been known to suffer severely from the disease. Ergotism in man is not an uncommon occurrence, and in nearly every instance it has resulted from eating bread made of ergotized grain. The disease makes its appearance among cattle chiefly in the winter and spring seasons and has at times been the cause of serious losses throughout the central and western states.

Ergot is the sclerotium of a parasitic fungus, *Claviceps purpurea*, which infests many species of native and cultivated grasses, and appears on some of our grains, especially rye. The sclerotium represents a stage in the life history of the fungus, which is intermediate between that of the mycelium or spawn, and that of the spore-bearing thallus. It flourishes particularly well on rich soil and in warm, damp seasons. The chemistry of ergot is not exactly known, although Kobert succeeded in separating three bodies; namely, ergotinic acid, cornutin, and sphacelinic acid.

Ergotinic acid is a protoplasmic poison, and when injected intravenously produces inflammation of serous and mucous membranes, disintegration of red blood cells, and wide-spread ecchymoses; cornutin excites the central nervous system and causes general convulsions; and sphacelinic acid induces gangrene.

Symptoms. Ergotism manifests itself among animals chiefly in the chronic form, since, as a rule, the poison is acquired in small amounts and accumulation takes place slowly. Two distinct types of the disease are recognized, namely: spasmodic and gangrenous. Symptoms referable to the digestive tract, such as nausea, vomiting, colic, diarrhoea or constipation appear in both forms. Pregnant animals very frequently abort.

In the spasmodic type of the disease, symptoms due to over stimulation of the central nervous system, appear. These are tonic contraction of the flexor tendons of the limbs, anaesthesia of the extremities, muscular trembling, general tetanic spasm, convulsions and delirium. Death usually occurs from secondary causes.

Gangrenous ergotism is attributed to prolonged constriction of the arterioles, and more directly perhaps to degenerative changes in the vessel walls, and the consequent formation of hyaline thrombi.

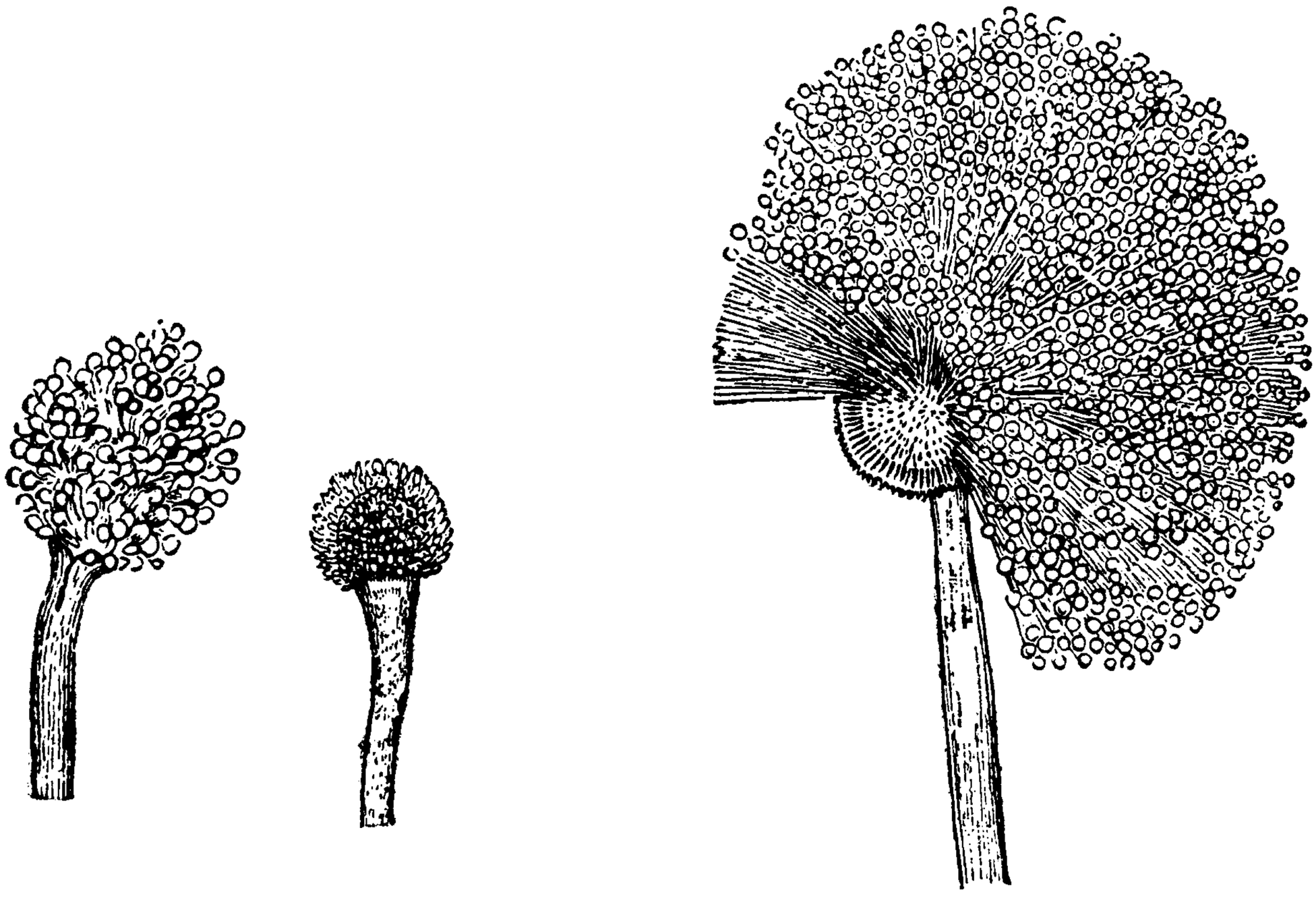
It is characterized by coldness and anaesthesia of the extremities, followed ultimately by dry gangrene of these parts. The effects of this dry gangrene are often very serious and amount to sloughing of the feet, tips of the ears, tip of the tail, shedding of the hair, teeth, etc. Death takes place from exhaustion.

Lesions. With the exception of the gangrene which may vary greatly in severity, there are no lesions of especial significance. Degenerative changes in the sensory area of the cord and in the vessel walls have been observed in animals slowly poisond with ergot.

Treatment. The first essential in the treatment of ergotism is to remove the cause. In well established cases treatment does not as a rule prove satisfactory.

Tannic acid is the chemical antidote, and should be given to neutralize the unabsorbed portion of the poison. Chloral is the physiological antidote. In

addition to giving the antidote, the treatment is entirely symptomatic. (Stuhr).



A *B*

Fig. 5. A. *Aspergillus fumigatus* showing coniliophore on right with sterigmata and spores attached on left. B. *A. niger* showing conidiophore, sterigmata, and spores attached in chains. After Siebenmann.

Aspergillosis

Pneumonomycosis is a not uncommon disease of domestic animals caused chiefly by the mould, *Aspergillus fumigatus*, although the *Aspergillus niger* is also pathogenic for birds. This disease is most frequent in birds, both domestic and wild, occasionally observed in horses and cattle, and rarely in man. Respiratory diseases and lowered vitality predispose. In all species the disease is characterized by purulent local inflammations in the lungs or other tissues, and a purulent and necrotic pseudomembrane upon the bronchial, tracheal, and other mucous membranes upon which it grows. The appearance of the pulmonary lesions sometimes resembles tubercle, sometimes actinomycosis.

Pneumonomycosis has been experimentally produced in birds (pigeons and geese) by compelling them to inhale aspergillus spores for a few minutes, after which they usually die of pneumonia in a few days. Rabbits have also been successfully inoculated by intravenous injection of spores.

Etiology. In mammals the *Aspergillus fumigatus* and in birds the *Aspergillus fumigatus, niger* and *flavescens* seem to be pathogenic species.

Infcetion takes place most commonly by inhalation of the spores which often are suspended in the air, or by taking them in with the food. Intestinal infection has not been observed. The spores are widely distributed in nature and exist in vegetable matter and grain abundantly. They possess remarkable vitality and exhibit considerable resistance to destructive agencies. The pathogenic power of the mould does not depend upon any product which it elaborates but upon the reactions which result from its penetration into the tissues. Peck observed the disease in seven subjects, in a stable where horses were fed on mouldy hacked hay.

Symptoms. The disease is of slow development in the larger animals and may not be observed until well advanced. In general the symptoms are of a pneumonic nature and in addition there is progressive emaciation. A case in a

Jersey cow, described by Pearson and Ravenel presented the following symptoms; the animal had been in poor condition for six months before it was examined. It was weak and depressed, did not eat, breathed with difficulty and, at times, coughed violently. Percussion of the chest gave sounds clearer and louder than normal and auscultation revealed the lung and bronchial sounds much intensified. Six days later these symptoms became more pronounced, the respiration and pulse very rapid. The animal grew rapidly weaker and died ten days after first being seen. The symptoms in birds are much the same as those in mammals except, that the disease runs a more rapid course. Emaciation advances rapidly and fetid diarrhoea may set in and continue until death in from a week to two months. At times emaciation is the only symptom. Fowls emit a glairy discharge from the nostrils which may contain the spores. In the prevention of the disease in fowls therefore, it is necessary to isolate or destroy the sick fowls together with the carcasses and fumigate the poultry houses. The roosts may be whitewashed.

Lesions. The lesions take the form of a miliary suppurative process, the foci varying in size from very small up to that of a pea. These may exist in large numbers and be scattered throughout the entire lung. Sometimes they become confluent and produce large areas of disease. The process starts in the bronchial mucous membrane, and later involves the bronchioles and alveoli. A very important feature is the intense amount of emphysema which is apparent on external examination of the lung. The lobules are often widely separated and can be readily seen in outline when a portion of the tissue is examined by transmitted light. In these emphysematous interlobular spaces, and in the air passages are seen whitish, mouldy looking patches. They are composed of denuded epithelium, inflammatory exudate, fruit hyphae and spores.

The lesions spread by penetration of the mycelium causing a destruction of tissue. Spores are not found within the tissues. In rare cases there is diffuse pneumonia characterized by hepatization and interstitial infiltration. On this latter account the disease has been described as being similar to contagious pleuro pneumonia of cattle. There may be pulmonary gangrene from secondary invasion of putrefactive organisms acting upon the devitalized tissue. An interesting feature is that this disease may interfere with the tuberculin test. This was shown in the case, above referred to, in which the test was used without success, and lesions of tuberculosis found in the lung on postmortem examination.

Treatment. This must of necessity be unsatisfactory since it is quite impossible to destroy the moulds which have penetrated the lungs. (Stuhr).

CHAPTER V

POISONING FROM FUNGI

That fungi of various kind are injurious, was known to the ancients. Prof. Ford[1] says, "The most interesting cases of mushroom or, as commonly described, toadstool poisoning and one of the first authentic cases on record, occurred in the family of the Greek poet, Euripedes, who lost in one day, wife, daughter, and two sons, who in the poet's absence partook of the deadly species. Among the great ones whose lives were sacrificed to the same ignorance may be mentioned Pope Clement VII., the Emperor Jovian, the Emperor Charles VI., Berronill of Naples and the widow of Tsar Alexis. The death of the Emperor Claudius is also assigned to this cause, but the reason and manner of the accident are not certain.

In addition to poisoning from toadstools, it has long been known that Ergot (*Claviceps purpurea*) is injurious to man and lower animals. In recent years Ergotism has not been so serious as formerly.

Other fungi also may be responsible for the death of animals by poisoning. The Fly Agaric (*Amanita muscaria*), a beautiful species, is common in many parts of the United States. I have described it in detail in another part of this work. In this connection I shall quote freely from the detailed and excellent account of poisoning as given by Prof. V. K. Chesnut, and the excellent report given of *A. phalloides* by Prof. Ford, who has written the most recent account of poisoning from this fungus.

The symptoms and treatment are thus described by Mr. V. K. Chesnut:

"The symptoms of poisoning from the fly amanita, as deduced from a number of cases, are varied. In some instances they begin only after several hours, but usually in from one-half to one or two hours. Vomiting and diarrhoea almost always occur, with a pronounced flow of saliva, suppression of the urine, and various cerebral phenomena beginning with giddiness, loss of confidence in one's ability to make ordinary movements, and derangement of vision. This is succeeded by stupor, cold sweats, and a very marked weakening of the heart's action. In case of rapid recovery the stupor is short and usually marked with mild delirium. In fatal cases the stupor continues from one to three days and death at last ensues from the gradual weakening and final stoppage of the heart's action.

"The treatment for poisoning by *Amanita muscaria* consists primarily in removing the unabsorbed portion of the amanita from the alimentary canal and in counteracting the effect of muscarin on the heart. The action of this organ should be fortified at once by the subcutaneous injection, by a physician, of atropin in doses of from one one-hundredth to one-fiftieth of a grain. As a stimulant emetic, mustard is particularly valuable. If this is not effective apomorphin should be administered by a physician. In case of profound stupor, however, even this may not produce the desired action. Tannin is of little

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1 Science N. S. 30: 97, 98.

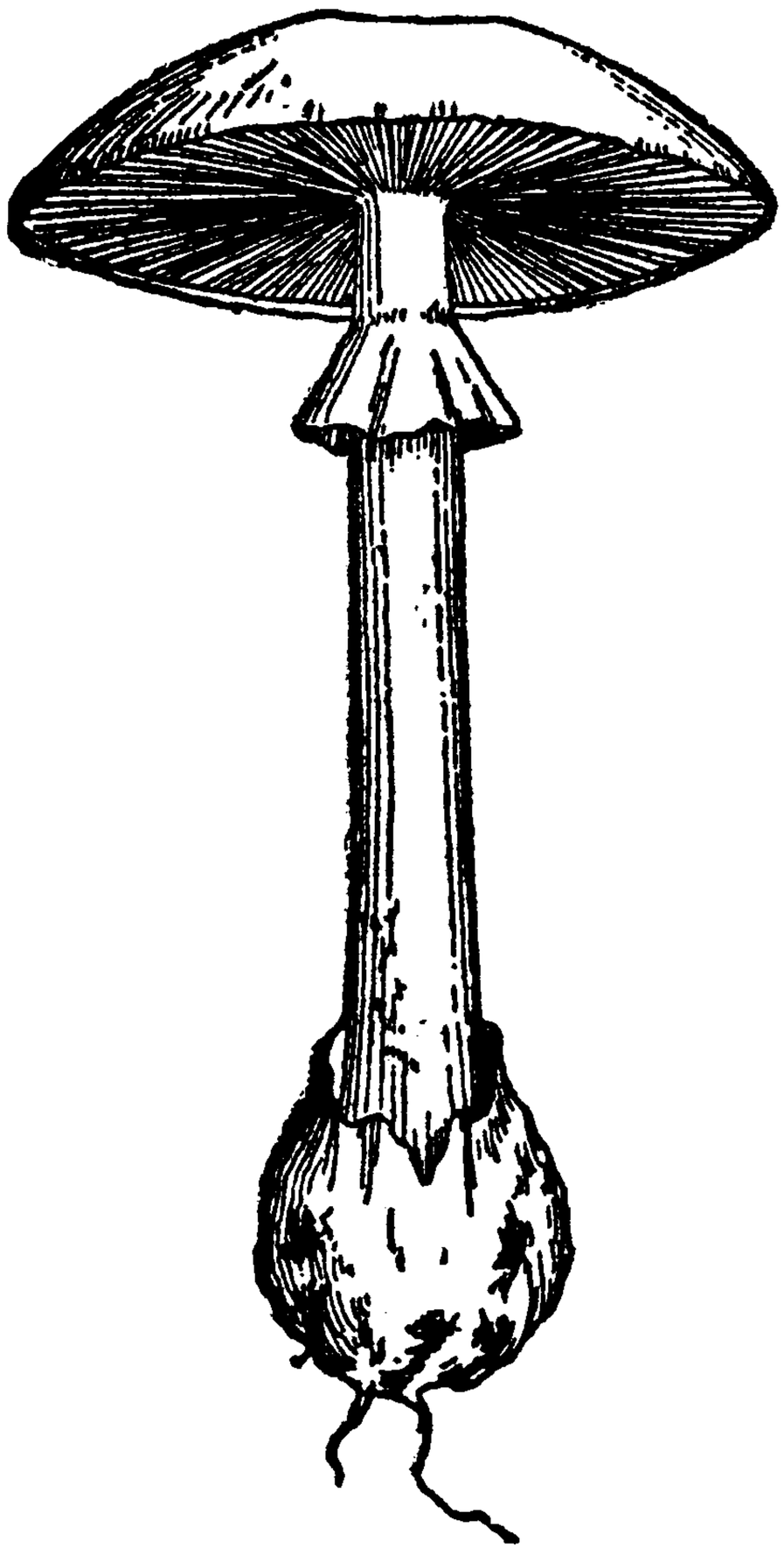

Fig. 6. Deadly Amanita (*Amanita phalloides*). U. S. Dept. Agrl.

or no value in rendering the muscarin insoluble in the stomach. If vomiting has not taken place, recently burned charcoal or two grains of a one per cent alkaline solution of permanganate of potash may then be administered, in order, in the cases of the former substance, to absorb the poison, or, in case of the latter, to decompose it. This should be followed by oils and oleaginous purgatives, and the intestines should be cleaned and washed with an enema of warm water and turpentine.

"Experiments on animals poisoned by the fly amanita and with pure muscarin show very clearly that when the heart has nearly ceased to beat it may be stimulated to strong action almost instantly by the use of atropin. Its use as thus demonstrated has been the means of saving many lives. We have in this alkaloid an almost perfect physiological antidote for muscarin, and therefore in such cases of poisoning its use should be pushed as heroically as the symptoms will warrant. The presence of *phallin* in *Amanita muscaria* is possible, and its symptoms should be looked for in the red color of the blood serum discharged from the intestines. Its treatment, which is difficult, is discussed under *Amanita phalloides.*

"It is well known that in some parts of Europe the fly amanita, after the removal of the poison by treatment with vinegar, is a common article of food. It was interesting to discover not long since that among some of our own people a similar practice prevails. Though most of the colored women of the markets look upon the species with horror, one of them recited in detail how she was in the habit of cooking it. She prepared the stem by scraping, the cap by removing the gills and peeling the upper surface. Thus dressed the mushrooms were first boiled in salt and water, and afterwards steeped in vinegar. They were then washed in clear water, cooked in gravy like ordinary mushrooms and served with beefsteak. This is an exceedingly interesting operation from the fact that although its author was wholly ignorant of the chemistry of mushroom poisons, she had nevertheless been employing a process for the removal of these poisons which was scientifically correct. The gills, according to various pharmacological researches, are the chief seat of the poisonous principles in this plant and their removal at once takes away a large part of the poison. The salt and water would remove phallin or any other toxalbumin the mushroom contained, and although the presence of phallin or any of this class of poisons has not been demonstrated in *Amanita muscaria,* there is a strong suspicion that it may occur in slight amount. The vinegar, secondly, removes the alkaloid poison, muscarin, and the mushroom after the two treatments is free from poisons. This process is cited, not to recommend its wider use, but as a matter of general interest. The writer's recommenda-

tion is that a mushroom containing such a deadly poison should not be used for food in any form, particularly at a season when excellent non-poisonous species may be had in abundance.

"It is surprising that cases of poisoning are not more frequent. At Tacoma Park, D. C., on November 9, of last year, a lady who has a thorough knowledge of edible and poisonous mushrooms met a family, consisting of a man, woman, and two children, who had just completed the gathering of a basketful of the fly amanita and the death cup, described below, which they were taking home to eat. In reply to questions the woman stated that they had often eaten this kind purchased dry at an Italian store, but that they had never gathered fresh ones before. Of course they had mistaken the species, or possibly the dried ones were fly amanitas from which the poison had been removed by treatment with vinegar. After considerable persuasion the people consented to throw the lot away.

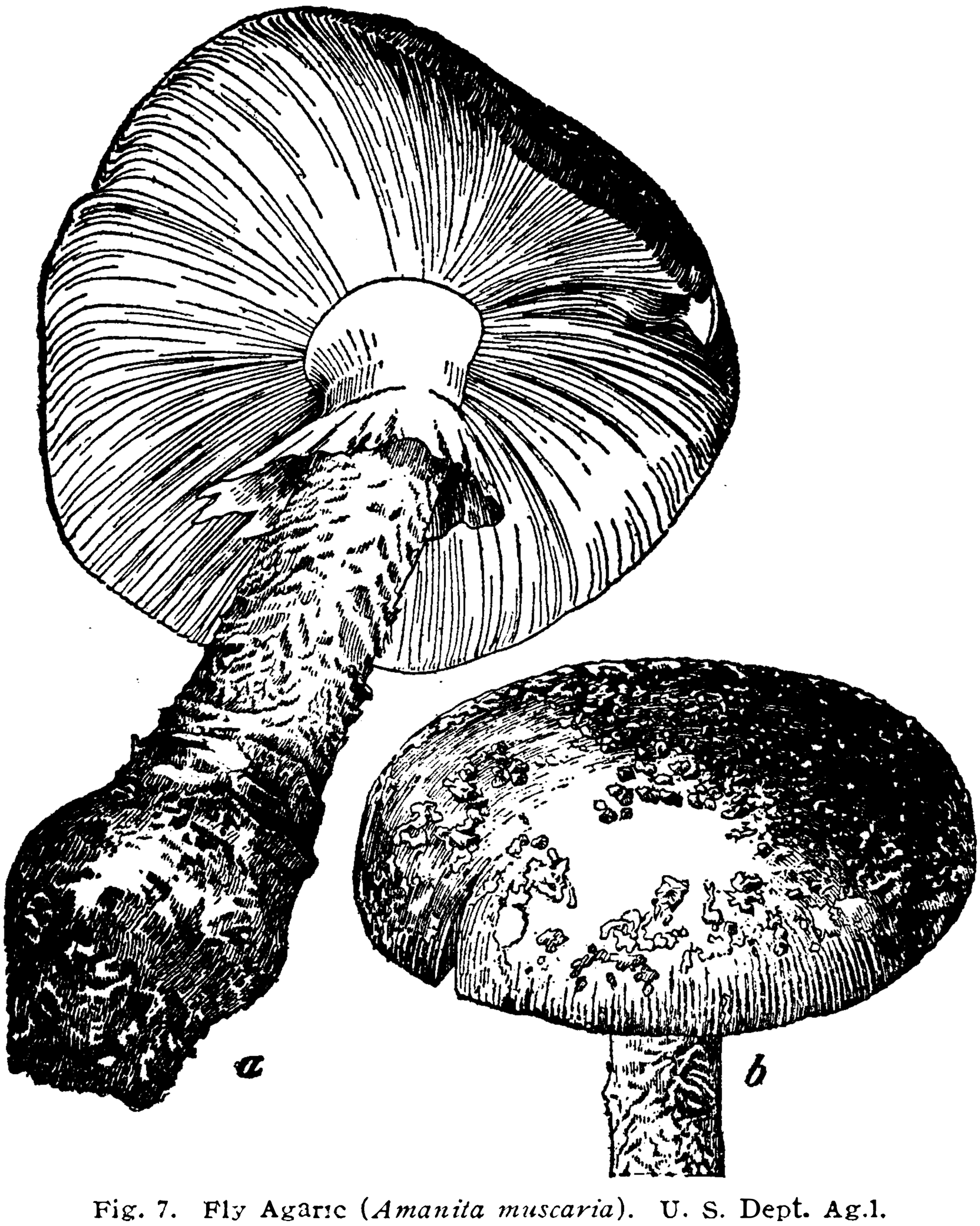

Fig. 7. Fly Agaric (*Amanita muscaria*). U. S. Dept. Ag.1.

"It is impossible to say what amount of the fly amanita would prove fatal, but in this connection it is of interest to note the custom reported by Krasheninnikoff, a Russian who travelled in Siberia and Kamchatka from 1733 to 1743, namely that the natives of the latter country, particularly the Koraks, used the fly amanita as an intoxicant, three or four specimens constituting a moderate dose for one habituated to its use, but ten being required for a thorough drunk.

The same observations, with varied details, have been made by others, particularly by Langsdorff, who traveled around the world with the Russian navigator Krusenstern from 1803 to 1806, and in more recent times by Kennan in his first Siberian journey of 1865-67.

"The plant may be taken fresh, but its taste is so disagreeable that only with great difficulty can a sufficient amount be eaten to produce the intoxicating effect. The Koraks have two principal methods of taking it: First, by swallowing pieces of the dried caps without chewing them; second, by boiling the dry caps in water and then drinking the liquor thus produced mixed with the juice of berries or herbs to disguise the taste. The intensity of the poisonous character of the fly amanita undoubtedly varies at different ages, with different individuals, and with different methods of preparation. The amount of the poison that can be taken into the system with impunity varies, too, with the person who takes it. The fact that a Korak, who has long used the plant as an intoxicant, can eat ten specimens and merely become drunk, does not prove that a similar number would not be fatal to an American who had never eaten it before.

"Very diverse statements concerning the properties of this fungus have been recorded. While some have attributed to it edible qualities, others have asserted that it is a most active poison and has caused numerous accidents by being confused with the Orange amanita. It is said to have caused death even when eaten in small quantities, and again it is said to have been eaten in abundance without any evil results. According to Quelet, it acts as a cathartic if eaten in small quantity, but causes death if eaten freely. One of my own correspondents assures me that he has eaten of the yellow variety, Var. *formosa*, without evil results, and that he regards it as very good. But there is no disputing the fact that the species possesses intoxicating and poisonous properties. It has long had the reputation of possessing properties fatal to flies that sip its juice. This suggests the names muscaria, Fly amanita, Fly agaric and Fly killer by which it is known. I have myself seen the cap of a single specimen surrounded by a circle of lifeless flies that had sipped the viscid juice from its moist surface and fallen victims to its virulent properties before leaving the place of their fatal repast.

"Some have attempted an explanation of the contradictory statements concerning this plant by supposing that its poisonous properties are not always developed, that in some localities or under favorable circumstances it is harmless. This explanation violates our sense of the constancy of Nature, and is not at all satisfactory. In the case of my own correspondent, the caps were peeled before cooking. May it not be that much of the noxious quality resides in the epidermis and the viscid substance upon it, and that by discarding this the dish is rendered less dangerous? In some cases it is said that those who eat it freely and without harm boil it a long time in water and throw away the water. In this way, doubtless, much of the poison is abstracted. Long soaking in salt and water, also in vinegar, have been recommended as a means of rendering suspected or noxious species harmless, and may have been practiced in some of the cases in which this fungus has been eaten with impunity. Whatever may be the explanation of the contradictory statements, the only safe way is to consider this species as deleterious and avoid its use under all circumstances. There is no need of taking any risks, with suspected species,

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a toxalbumin is a glucoside. Prof. Ford has obtained an anti-poison or an anti-hemolysin with a high grade of immunity. According to Schlesinger and Ford[1] the Amanita-toxin in a purified state is one of the most powerful of organic poisons — four tenths of one milligram killing a guinea pig within twenty-four hours. Ford believes that the hemolysin plays no part in human intoxication, but that the toxin is the active principal which resists the action of the gastric juice and boiling. He finds that the *Amanita rubescens* considered an edible species by some, contains an hemolysin as powerful as the Deadly Amanita. He found a toxin and an hemolysin in *Amanita virosa.* The latter substance in a dilution of 1-200 killed a guinea pig. The *A. spreta* produced intoxication and according to Ford must be classified with the "deadly poisonous" as the *A. verna.*

The *A. strobiliformis, A. chlorinosma, A. radicata,* and *A. porphyria,* do not contain hemolysins but small quantities of a toxin probably identical with *amanita-toxin.* The *Amanita solitaria,* regarded as edible, causes the blood corpuscles to adhere in clumps much as agglutination occurs with typhoid bacilli when brought in contact with the blood of a typhoid patient.

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1 Jour. Biol. Chem. 3:279. 1909.

Anemonella and Stemless Loco Weed. Photo by Charlotte M. King.

CHAPTER VI

POISONING FROM OTHER PLANTS. EQUISETOSIS, LOCOISM, AND LUPINOSIS

Equisetosis. It has been recently proven by direct experimentation that the common horsetail (*Equisetum arvense*) when ingested in sufficient amount, is capable of producing fatal poisoning among horses. This discovery is of great importance since the plant has a wide distribution, and at times is the cause of extensive losses. The common horsetail thrives best in moist sandy soils or in low, damp meadows, which are not frequently cultivated, and often constitutes a large part of wild hay. The dried plant alone seems to be poisonous. Young horses seem to be the most susceptible. Sheep are supposed to be slightly susceptible although cattle eat the hay in which the plant occurs in large proportion, with impunity. The toxic principle of the plant has not been determined.

Symptoms. The effects of poisoning from eating horsetail appear at times varying from two to five weeks, depending upon the age of the animal, and the amount of contaminated hay ingested.

The first symptoms are usually unthriftiness, general bodily weakness and emaciation. The animal seems to have a depraved appetite, preferring the plant to wholesome feed. As the disease progresses the muscular weakness becomes more pronounced, the animal loses muscular control and exhibits incoordinate movements. During this stage the pulse and temperature are depressed, extremities are cold and the visible mucosae are pale. Appetite usually remains good until the end and consciousness is apparently retained. Finally the animal falls, manifests nervous excitement, paroxysms of convulsions appear and death results from exhaustion. In the final stage the pulse becomes accelerated and the temperature elevated.

Hypostatic pneumonia is a frequent complication.

Treatment. The first step in the treatment is the removal of the cause. A cathartic should be administered to rid the bowels of the irritant and nerve and heart stimulants given to combat the symptoms of depression. In case the patient is unable to stand, it would be advisable to give some support. When the animal is down it becomes necessary to guard against the development of hypostatic pneumonia.

Where cases are not too far advanced and appropriate treatment is instituted, recovery is the usual result. (Stuhr).

Locoism. Stock-poisoning by the loco weed is a frequent and serious condition with which the stock-owners of the western half of the United States have to contend. Montana and Colorado, especially, sustain heavy annual losses. Similar diseases occur in other parts of the world. In Australia other plants of the order *Leguiminosae* like *Gastrolobium* produce similar symptoms. Maiden[1] states that the "Nenta Lessertia disease of S. Africa is identical with a disease of the Pea-eating animals of Australia and

1 Miscell. Pub. Dept. Agrl. N. S. Wales, 477: 11.

with the Loco disease of the United States. Many forage plants of excellent repute such as white clover, alfalfa, lotus and other plants, may produce tympanites.

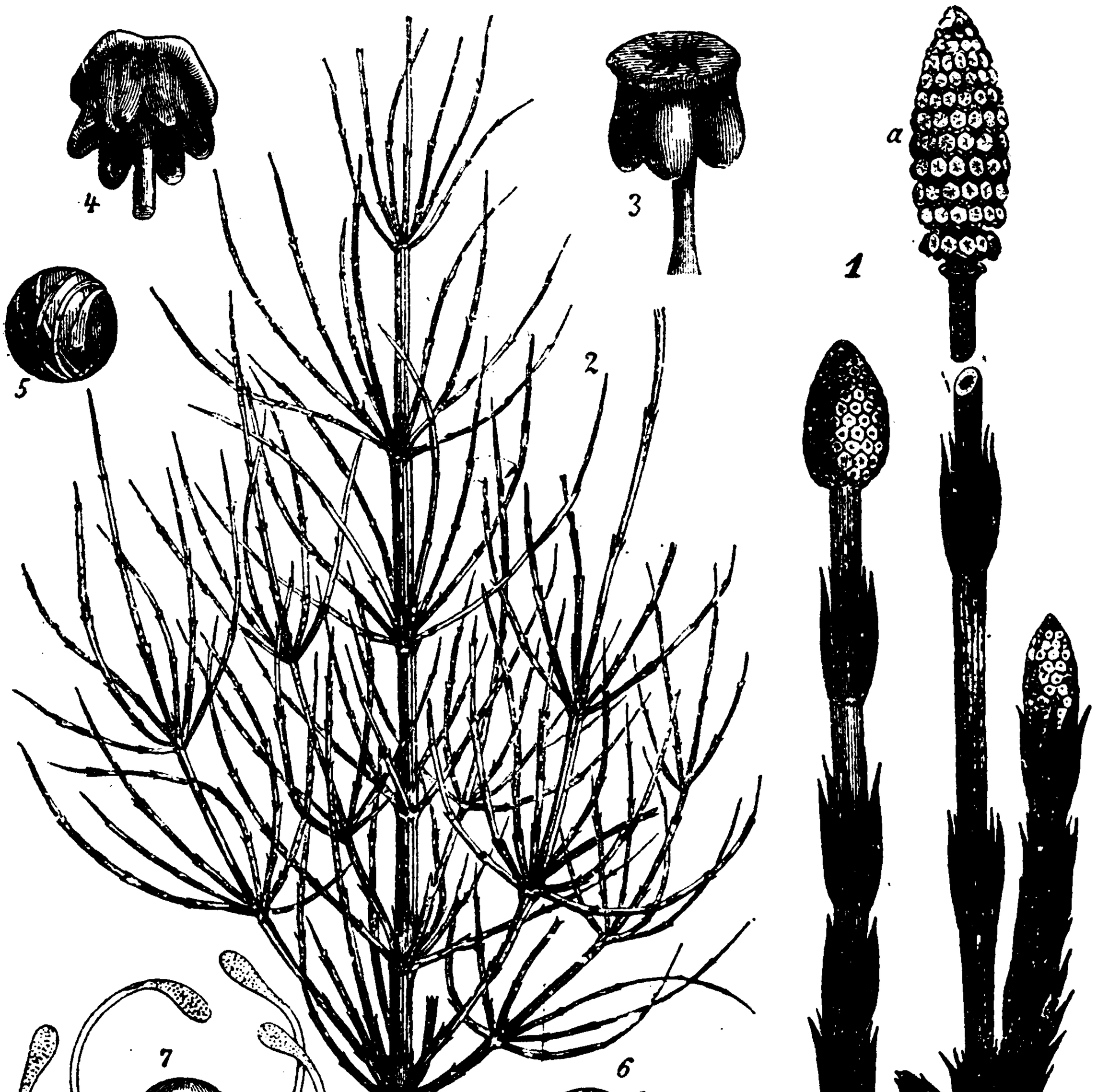

Fig. 8. Common Horsetail (*Equisetum arvense*), the plant causing Equisetosis. 1. Fertile stems terminating in cones *a*. 2, Sterile stem. *a*. rhizome tubers. 3. Sporophyl with sporangia. 4. Sporangia opened to discharge spores. 5, 6, 7. Spores with spiral elaters. After Wossidlo.

Symptoms: difficult breathing; the poison enters the circulation and stops the action of the lungs and heart when the animals stagger and die.

In the advanced stages the animals become frantic, hence the name "loco" or crazy. Horses and sheep are the most susceptible, although cattle are also affected.

Of the various species of loco weed, the stemless loco *Oxytropis Lamberti Pursh*), and the woolly loco weed (*Astragalus mollisismus Torr*), are the most injurious. These weeds grow luxuriantly on sandy ranges and appear early in the spring when other vegetation is scarce, and since they retain their fresh

Fig. 9. Loco Weed (*Astragalus mollissimus*), U. S. Dept. Agrl.

green color during the entire summer they prove especially attractive to stock.

Recently the poisoning has been attributed by Dr. Crawford and others of the U. S. Dept. of Agrl., to mineral salts in the plant.

The period of greatest danger is chiefly during the month of May.

Symptoms. The symptoms, which are referable to the nervous system, are attributable to the narcotic effect of the plant. They appear slowly and are apparently divisible into two stages. The first stage is characterized by the following symptoms: Stupor, defective vision, unnatural movements and apparent hallucinations. When excited the animals become frenzied. The coat becomes shaggy, the teeth grow long and become loose, and a depraved appetite which is very marked, is developed. The animals prefer the loco weed to wholesome food, and will dig up the roots and eat them to satisfy their craving.

In the second stage there is emaciation, exhaustion, feeble movements and finally death from starvation. The course of the disease is quite variable

and may last from a few months to one or two years. Sheep manifest symptoms very similar to those above described.

Treatment. When the disease has reached the advanced stage, treatment is of no avail as recovery does not occur. If, however, the afflicted animals are taken early in the course of the disease and placed on pasture where loco weeds do not exist, and are given good nourishing food, there is hope of recovery.

Prevention, which of course is the most desirable, is not always practicable. Animals do not as a rule become addicted to the loco habit when they have plenty of wholesome food and salt.

There are no demonstrable lesions other than emaciation. (Stuhr).

The recent investigations of Marsh and Crawford lay considerable stress on the presence of barium in the plants and the Bureau of Animal Industry, Washington, D. C., recommends the following treatment: for cattle, strychnin in doses of three-twentieths to four-twentieths of a grain daily, administered hypodermically; for horses, Fowler's solution of arsenic in half-ounce doses daily in the drinking water or in the grain. This treatment should be continued for at least a month. To correct the constipation which is almost universal in locoed animals, magnesium sulphate (Epsom salt) may be administered as a drench in two-ounce doses. Epsom salt may also serve to some extent as an antidote to the poison produced by the weeds. Beneficial results have also been obtained by giving horses daily a drench containing two ounces of Epsom salt with ten drops of dilute sulphuric acid, and by giving cattle tri-weekly three or four ounces of Epsom salt with a proportional increase in the quantity of dilute sulphuric acid.

As the foregoing treatments are in the experimental stage, the Bureau of Animal industry of Washington, D. C., would be glad to receive reports from their use.

The value of keeping stock away from these poisonous plants is indicated in some investigations that have been carried on by the Bureau of Forestry and the Bureau of Plant Industry. In many cases the ranges are becoming practically useless on account of these poisonous plants and if used the losses are so heavy as to materially reduce the profits of the business. In the Manta Forest Reserves in Utah for instance, it was found that the death of sheep was due to their browsing upon the chokecherry. Certain portions of the old trail were abandoned, and along other portions the chokecherry bushes were cut out. The method of handling the sheep was also changed. Instead of large bands which could be moved but slowly, smaller bands were trailed, and so far as possible they were allowed to fill up on healthy forage before entering the dangerous area. The trail was also improved wherever practicable and by this means it was possible to get the sheep through in much better shape and with little or no loss. The Department has also, in some instances, adopted the plan of flagging the area in which these injurious plants occur.

Lupinosis. This is a disease of sheep and horses especially, caused by eating the seeds and straw of the lupine. Cattle and goats are also susceptible and the dog has been poisoned experimentally. There are many species of the lupine growing in various parts of the United States, although the yellow lupine (*Lupinus luteus*) is the most toxic. The nature of the toxic agent found in the lupines has not as yet been determined. Arnold and Schneidemühl succeeded in isolating a chemical poison and

gave to it the name lupinotoxin. They described its physical properties but failed to determine its chemical composition. Attempts to associate a fungus with the plant have failed. Lupinosis is characterized by jaundice, acute yellow atrophy of the liver, and parenchymatous inflammation of other internal organs.

Symptoms in Sheep. The disease appears in either the acute or chronic form, depending upon the amount of poison ingested. These two forms have been experimentally reproduced by giving carefully regulated amounts of lupinotoxin.

In the acute form the disease appears suddenly. There is loss of appetite, fever, hurried and difficult breathing, rapid pulse, stupor, vertigo, and not infrequently swelling of the lips, ears or face. The initial temperature may be as high as 104° to 106° Fahr., but is intermittent and gradually falls just before death. The pulse may reach 130 per minute and the respirations 100. A bloody froth may issue from the nostrils. Icterus which may be detected in the conjunctiva and the urine, usually appears on the second or third day. In certain cases this latter symptom fails to manifest itself and therefore is not constant. There is grinding of the teeth and sometimes trismus. The animal apparently prefers the recumbent position, extends the head on the ground and seems entirely oblivious to all surroundings. At first there is constipation, the faeces being hard and scanty and covered with yellow mucous. Later diarrhoea may set in and the excreta be tinged with blood giving them a dark brown color. Emaciation develops rapidly. In case of recovery the symptoms gradually abate and improvement takes place slowly. Cachexia is a common sequel. In the chronic form the symptoms are not so violent. Jaundice may be entirely absent and emaciation and anemia may be the chief signs. Inflammatory tumefaction of the lips, eyelids, and ears with the formation of ulcers and scabs is described by various writers.

Course. Death may supervene within twenty-four to forty-eight hours, although frequently the disease lasts four or five days. The immediate cause of death is rapid emaciation and extreme weakness. Horses contract the disease from eating oats contaminated with the seeds or from eating the straw of the plant. The symptoms which they manifest are essentially the same as those above described. Horses seldom die from the effects of lupines.

Lesions. The cadavers are emaciated and decompose rapidly. The muscles are of a grayish yellow color, the fibers having become fatty and having lost their striations. The subcutaneous tissue of the abdomen and the omentum and mesentery are yellowish. The most important lesion in both the acute and chronic forms is found in the liver. The alterations in this organ are those of acute hepatitis. The liver cells have become swollen and granular on account of the parenchymatous change, or they may be more or less completely degenerated into fat. The gland is soft and friable and may be somewhat swollen. The interlobular connective tissue is greatly increased in amount due to inflammatory hyperplasia. In the course of a few days the liver undergoes acute yellow atrophy as a result of the absorption of the degenerated cells and the contraction of the hyperplastic stroma. In the chronic form the changes are those of chronic interstitial hepatitis. The icterus is of hepatic origin and due to catarrh of the bile ducts. The gall bladder is distended with bile and its lining membrane is congested and swollen. The kidneys and bladder may show changes, more or less marked, due to inflammation. The blad-

der is, as a rule, empty. In the digestive tract we observe frequently yellowish discoloration of the mucosa, hemorrhages in the small intestine with catarrhal lesions of the entire canal. The heart is pale and friable and the blood which it contains is dark and thick. Capillary hemorrhages are quite generally observed throughout all of the tissues.

Treatment. This is chiefly preventive since there is no specific antidote. Attempts should be made at once to prevent further absorption of poison by administering some acid as acetic or hydrochloric, well diluted with water. Alkalis should be strictly avoided as the poisonous principle is very soluble in alkaline solutions. It is advisable to evacuate the bowels by giving a purgative, preferably oil. Potassium permanganate is recommended by some as an antidote. Further than this the treatment is entirely symptomatic. (Stuhr).

A B

Fig. 11. A. Larkspur (*Delphinium tricorne*). B. *D. Carolinianum.* Larkspur poisoning (*Delphinosis*) is caused by various species of Delphinium. *A.* U. S. Dept. Agrl. *B.* Ada Hayden.

CHAPTER VII

DELPHINOSIS, LATHYRISMUS (LATHYRISM), ACONITISM, VERATRISM, UMBELLIFERAE, CONIUM, CICUTA.

Delphinosis.

The purple larkspur (*Delphinium Menziesii D C.*), and other species which are found in the northwestern part of this country and especially in Montana are plants very dangerous to stock. Drs. Chesnut and Wilcox have proven the toxicity of the above species by direct experiment and have called attention to the serious losses which they occasion annually.

The latter experimenter has fatally poisoned a yearling lamb within two hours by administering, *per os*, the extract made from less than an ounce of the dried leaves. The weed appears early in spring, in advance of the forage plants and it is during this period that the greatest harm results. The poison is found both in the leaves and the roots although the latter are not frequently eaten on account of their woody fibrous nature. The poisonous principle has not been isolated. Cattle and sheep are most susceptibl,e although horses frequently suffer.

Symptoms. The first indication of poisoning is a general stiffness and a straggling gait, especially in the posterior limbs. Walking appears to be difficult, and is evidently painful. At this stage the pulse and respiration are much depressed, and the temperature is lowered. The appetite is retained in most cases until the appearance of the final stage of the poisoning. This is manifested by irregular muscular twitching of all of the muscles which finally becomes frequent and violent, and by incoordinate movement. There are attempts at vomiting and the animal froths at the mouth. Finally all of the muscles of the body contract spasmodically, the animal falls and dies in violent spasms. The pulse and respiration become very weak and rapid just before death. There is no aberration of the special senses. The course of the poisoning is quite rapid and death usually takes place within a few hours.

Lesions. The direct cause of death from larkspur poisoning is probably failure of respiration due to paralysis of the centre and the alterations therefore are those of asphyxia. The lungs are congested and dark-colored and the right heart, veins and capillaries are distended with dark colored blood. Other organs and tissues are normal with the exception of the general venous and capillary congestion.

Treatment. Potassium permanganate is the chemical antidote and should be given as early as possible in doses of fifteen to twenty grains to horses, thirty to fifty grains to cattle, and five to ten grains to sheep, dissolved in a copious amount of water. To combat the extreme depression of the circulation and respiration, atropin sulphate has proven very efficacious. Even after the final convulsions have begun this drug has been of good service. It should be hypodermically administered in doses of three-fourths to one grain to the larger animals and one-twentieth to one-fifteenth of a grain to sheep. In the late stages sheep should be given as much as one-sixth to one-

fourth of a grain. In the convulsive stage when there is impending failure of respiration, inhalations of ammonia may be resorted to with good results.

A feature of the treatment quite as important as the medication is the care of the animals. Complete rest and freedom from excitement are very essential since exercise or fright is likely to induce fatal spasms.

Finally the danger from poisoning can be largely obviated by preventing access to the young plant in the early spring.

Lathyrismus.

In ancient times, this disease was quite common, affecting both man and the horse. It is very frequent today in Spain, Italy, Russia, and India.

Etiology. It is caused by eating bread made of flour derived from some species of Lathyrus or Vetch (*L. Cicera, L. Clymenum,* or *L. sativus*), or, in case of horses, by the consumption of Fodder Pea. The seeds of these species contain a toxic substance formed without the action of bacteria. Man, the horse and the pig are subject to this disease.

Symptoms. In domestic animals, the symptoms are debility of the rear and lower extremities, producing motor paralysis. In lower animals, the normal functions of the larynx become impaired because of paralysis.

Treatment. Change of fodder, providing good food, application of massage and electric treatment.

Fig. 12. Aconite (*Aconitum uncinatum*). Aconitism is caused by this and other species of Aconitum. Charlotte M. King.

Fig. 13. Green Hellebore (*Veratrum viride*). Common Eastward. (U. S. Dept. Agrl.).

Prognosis. Fatal cases are not frequent.

Post-mortem. The ganglion cells of the anterior horns are atrophied; also the recurrent nerves and the muscles of the larynx.

(Adapted from Friedberger and Fröhner).

Aconitism.

Various species of aconite (*Aconitum*) are known to be poisonous. One species, the *Aconitum Napellus*, has long been used in medicine. The most common species in North America is the Columbia Aconite (*A. columbianum*), which is found in the Rocky Mountains and on the Pacific coast. Several other species occur in North America, one extending into northeastern Iowa; the European Aconite (*A. Napellus*) is frequently cultivated in gardens. Cases of poisoning are largely due to the administration of over doses in medicine. In the Rocky Mountains accidental poisoning among live stock is confined to animals that graze at higher altitudes. All parts of the plant are poisonous; one tenth of a grain of the drug is a poisonous dose for some animals. The smallest fatal dose recorded in man is a teaspoonful of the tincture of aconite, which is equivalent to about XXX gr. of the crude drug. The minimum lethal quantity is 1-16 of a gr. for man.

Symptoms. The effects of the poisoning are a tingling sensation on the end of the tongue, which shortly gives rise to a burning sensation followed by pronounced constriction in the throat. It reduces the pulse and frequency of the cardiac pulsations. The action of the heart is lessened and the pulse is weak, irregular and intermittent, at first slow and then rapid; tingling and prickling over the entire body is characteristic; vision is confused, there are abdominal pains, vomiting and diarrhoea. Death is caused by the stoppage of respiration, but is preceded by numerous twitchings; in the case of the horse the animal falls and is unable to rise. The symptoms are different in cats and rabbits.

Treatment. No specific antidote is known, but physicians use atropin or digitalis and nitrite of amyl. The stomach should, however, be evacuated at once; cardiac and respirative stimulants are given subcutaneously.

Veratrism.

The Hellebores belonging to the genus Veratrum are common in the mountain regions of the west and one species is abundant in the east. They are found in swampy places, in wet meadows and along brooks. The most frequent cases of poisoning come through the administration of the drug; although in the Rocky Mountains considerable quantities of the plant are consumed by sheep where grazing is close. All parts of the plant are poisonous although the root is more poisonous than the seeds and leaves which contain several alkaloids.

Symptoms. Veratrin is a powerful irritant and when inhaled in minute quantities produces sneezing. When injected under the skin it causes restlessness, when consumed in large quantities it produces salivation, frequent vomiting with purging pain, and collapse, the temperature falls. Veratrin is a drug poisonous to the motor and sensory nerves. Death is caused by paralysis of the heart; 1/16 of a grain has produced alarming symptoms in man and 1 gr. injected subcutaneously produces poisonous symptoms in a horse.

Treatment. The stomach should be emptied immediately, then give stimulants, such as brandy and strong coffee.

Fig. 14. Larkspur (*Delphinium glaucum*) causes Delphinosis (U. S. Dept. Agrl.).

Fig. 15. Cowbane (*Cicuta occidentalis*), a deadly poisonous plant. (U. S. Dept. Agrl.).

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reported to the writer by an Iowa farmer, a cow which had eaten freely of the roots, fell into a spasm when brought into the barnyard. The animal, however, rose, walked one hundred feet and fell again, got up again, walked about thirty rods, fell and died in about thirty minutes. In a second case, a yearling owned by the same man had been in good healthy condition but began to display the same symptoms and died in twenty minutes.

Dr. E. S. McCord, on September 31, of the same year, gave an old horse six drachms, hypodermically, of a strong decoction of the root. In fifteen minutes the animal showed uneasiness; pulse was full and fast; in a short time the animal laid down, and the pulse decreased; the horse was in great pain and kept moving the extremities; the pulse was weak but the patient finally recovered. The botanist of the Oregon Experiment Station found that the root has less of the toxic substance in the summer than in the winter and spring, which may account for the failure in this last case to produce death. In frogs, frequency of breathing is increased, tetanic convulsions follow, gradually paresis of the extremities, and lastly full paralysis and death. Cicutoxin,

Fig. 16a. Poison Hemlock (*Conium maculatum*), native to Europe; naturalized in the U. S. (U. S. Dept. Agrl.).

the characteristic poison of Cicuta, acts especially upon the medulla oblongata; the brain and spinal cord are merely secondary seats of its action.

Treatment. The stomach should be effectually evacuated by the use of the stomach pump or by a strong emetic. External and internal stimulants such as whisky should be applied; anaesthetics and narcotics used to control the spasms; hypodermic injection of morphin aids in recovery. It usually hap-

pens, however, that the veterinarian or physician is called too late to accomplish much.

The Poison Hemlock.

The Poison Hemlock (*Conium maculatum*) is indigenous to Europe and has long been known as a poisonous plant. It is a fetid smelling herb from 2-5 feet high, with a spotted stem, compound leaves, and small, white flowers in umbels. The plant is not uncommon in waste places in the East and in the Rocky Mountains, especially in Utah. It has long been used as a poison.

Symptoms. In lower animals, there is observed a dilatation of the pupil, followed by weakness of the limbs, passing into paralysis; labored respiration, frequency of breathing diminished, heart action irregular; death preceded by convulsions. In man, there are weakness in the lower extremities, staggering gait, in two hours paralysis of upper and lower extremities and slight convulsions; death occurs in a few hours usually caused by cessation of respiration.

Treatment. The stomach should be evacuated by means of a pump or tube; or a hypodermic injection of 4-5 drops of a solution of apomorphin given; or emetics of sulphate of zinc or mustard administered. The temperature of the body should be kept up by hot applications.

Stimulants may be given, and, if necessary, artificial respiration applied. As a drink, strong tea, tannin, or any harmless vegetable decoction containing tannin may be administered.

CHAPTER VIII

FISH AND ARROW POISONS, HYDROCYANIC POISONING—TOXALBUMINS—BLACK LOCUST, CASTOR OIL, AND JEQUIRITY.

Fish and Arrow Poisons.

Fish and arrow poisons have played an important part with the aborigines of all countries and they are still used to a considerable extent by primitive people. Thus Merrill [1] mentions the use of the Antiaria toxicaria in the Philippines and other plants used in the same way which are being worked up by Dr. R. F. Bacon.

Radlkofer [2] some years ago published a long list of plants which are used to poison fish, and added a history of the earlier literature. He lists some 154 species which have been used in various parts of the world for this purpose and these plants belong to the following orders and genera. The species are listed under the poisonous species in another part of this work.

Dilleniaceae (*Tetracera*), Menispermaceae (*Anamirta, Abuta, Pachygone*); Cruciferae (*Lepidium*), Capparideae (*Cleome*), Bixaceae (*Pangium, Hydnocarpus*), Ternstroemiaceae (*Caryocar*), Tiliaceae (*Grewia*), Meliaceae (*Walsura*); Chailletiaceae (*Chailletia Tapura*); Rhamneae (*Gouania*); Sapindaceae (*Serjania, Paullinia, Sapindus, Dodonaea, Harpullia, Magonia*); Hippocastaneae (*Pavia*); Leguminosae (*Tephrosia, Milletia, Orobus, Abrus, Centrosema, Clitoria, Camptosema, Phaseolus, Lonchocarpus, Derris, Piscidia, Bowdichia, Cassia, Bauhinia, Leucaena, Albizzia*); Myrtaceae (*Barringtonia, Gustavia*); Compositae (*Clibadium, Ichthyothere*); Campanulaceae (*Tupa*); Ericaceae (*Rhododendron*); Primulaceae (*Cyclamen*); Myrsineae (*Aegiceras, Jacquinia*); Sapotaceae (*Bassia*); Ebenaceae (*Diospyros*); Apocyneae (*Melodinus, Thevetia, Cerbera, Aspidosperma*); Loganiaceae (*Buddleia, Strychnos*); Solanaceae (*Hyoscyamus, Nicotiana*); Scrophularineae (*Verbascum, Digitalis*); Bignoniaceae (*Bignonia, Tecoma, Jacaranda*); Labiatae (*Eremostachys*); Chenopodiaceae (*Chenopodium*); Polygoneae (*Polygonum*); Aristolochiaceae (*Aristolochia*); Piperaceae (*Piper*); Thymelaeaceae (*Daphne, Wilkstroemia*); *Euphorbiaceae* (*Euphorbia, Phyllanthus, Securinega, Piranhea, Croton, Joannesia, Manihot, Jatropha, Excoecaria, Hura*); Coniferae (*Taxus*); Liliaceae (*Veratrum*).

Ernst [3] lists only sixty species that are used as fish poison. There must, however, be considerably more as indicated by Radlkofer.

W. M. I. Brost Pauwels [4] in his contribution on the Surinamic fish poisoning [5] contributes an interesting article on the subject.

Pauwels who made an investigation of Nekoe (*Lonchocarpus violaceus*) states that it is a powerfully toxic substance. He found that Nekoeid will poison fish in proportion of 1,5,000,000, and that a second substance B.

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1 Philip. Journ. of Sci. 2:111, Sect. C.

2 Sitz., Math-Phys. Classe k. b. Akad. d. Wiss. München., 1886, 379.

3 Memorio Bot. el Embarbascar o sea la Pesca por media de Plantis venenosas.

4 Bijdrage tot de Kennis der Surinaamsche Vischvergiften. M. Greshoff has likewise published a number of works on fish poisoning plants.

5 Hart and Swatters found in the *Piscidia Erythrina* piscidin $C_{15}H_{12}O_4$, and Greshoff found in *Pachyrhizus angulatus*, pachyrhizid $C_{28}H_{18}O_8$ $(OCH_3)_2$.

Nekoeid will poison fish in proportion of 1-10,000,000. The poison will take effect in one hour. The water poisoned with the substance will cause the fish to make an effort to get away from the poison, they are in a horizontal position, breathe heavily, come to the surface of the water and try to jump out and finally breathing becomes increasingly difficult and at last they turn on their backs and die.

Hydrocyanic Poisoning.

Under poisoning from cherry, sorghum and a few other plants, an account has been given of poisoning from hydrocyanic acid. It may be convenient to bring together some of the plants from which the very poisonous substance, prussic acid, has been obtained.

Maurits Greshoff of the Colonial Museum in Holland has taken the pains in a paper on Cyanogenesis to give the distribution of Prussic acid in the vegetable kingdom, the Hydrocyanic acid being found in a great many different plants. The following list gives the orders in which this substance occurs.

Ranunculaceae (*Aquilegia vulgaris, Thalictrum aquilegifolium*). Berberidaceae (*Nandina domestica*). Cruciferae (*Lepidium sativum*), Bixaceae (*Gynocardia odorata, Hydnocarpus venenata, Kiggelaria africana, Pangium edule, Ryparosa caesia, Taraktogenos Blumei, Trichadenia zeylanica*); Sterculiaceae (*Sterculia*); Tiliaceae (*Echinocarpus*); Linaceae (*Linum usitatissimum*); Rutaceae (*Citrus medica*); Dichopetalaceae (*Chailletia cymosa*); Olacaceae (*Ximenia americana*); Celastraceae (*Kurrimia zeylanica*); Rhamnaceae (*Rhamnus Frangula*); Sapindaceae (*Cupania, Schleichera trijuga*); Anacardiaceae (*Corynocarpus laevigata*); Leguminosae-Papilionaceae (*Lotus arabiscus, Indigofera galegoides, Phaseolus lunatus, Vicia sativa, Dolichos Lablab*); Rosaceae (*Amelanchier vulgaris, Chamaemeles, Cotoneaster integerrima, Crataegus Oxyacantha, Eriobotyra japonica, Nuttallia cerasiformis, Osteomeles, Photinia, Pyrus, Prunus Amygdalus, Pygeum africanum, Spiraea Aruncus*); Saxifragaceae (*Ribes aurcum*); Combretaceae (*?Combretum constrictum*); Myrtaceae (*?Psidium montanum*); Melastomaceae (*Memecylon*); Samydaceae (*Homalium*); Passifloraceae (*Passiflora quadrangularis, Tacsonia*); Caprifoliaceae (*Sambucus nigra*); Rubiaceae (*Plectronia dicocca*); Compositae (*Chardinia xeranthemoides, Xeranthemum annuum*); Sapotaceae (*Isonandra, Lucuma bonplandia, Payena latifolia*); Asclepiadaceae (*Gymnema latifolium*); Convolculaceae (*Ipomoea dissecta*); Bignoniaceae (*Osmohydrophora nocturna*); Euphorbiaceae (*Bridelia ovata, Elateriospermum Tapos, Hevea brasiliensis, Jatropha augustidens, Manihot utilissima, Ricinus communis*); Urticaceae (*Sponia virgata*); Araceae (*Arum maculatum, Colocasia gigantea, Cyrtosperma lasioides, Lasia aculeata*); Gramineae (*Glyceria aquatica, Panicum, Sorghum vulgare, Stipa hystricina*); Fungi (*Hygrophorus agathosmus, Marasmius oreades, Phaliota radicosa, Russula foetens*). He makes the following statement with regard to the presence of this substance in plants:

"Many plant physiologists in Europe, with more experience with Prunus or amygdalin than with the tropical Pangium, incline to the view that hydrocyanic acid in these plants has nothing to do with either the building-up or the breaking-down of proteids, but that this substance is made by the plant from sugar and nitrate by a special process, and serves no other purpose than to defend the plants against the attacks of animals. It is above all the incompleteness of our physiological knowledge which makes decision between these theories difficult.

"In the study of this question it is important to remember the possible diversity of origin of this body, and every cyanogenetic plant will be required to be examined on the lines laid down by Treub."

The wide distribution of glucosides that yield hydrocyanic acid is evident from the list above. Dunstan and Henry [1] discovered three glucosides, *dhurrin* $C_{17}H_{17}O_7N$ in the common sorghum, *lotusin* $C_{28}H_{31}O_{16}N$ in a species of lotus of Egypt and *phaseo-lunatin* $C_{10}H_{17}O_6N$ in wild beans of *Phaseolus lunatus*, the common lima bean. Brunnich [2] attributed death from the feeding of immature sorghum to *dhurrin*. Power and Lees [3] isolated from the seeds of *Gynocardata odorata* a glucoside to which they gave the name *gynocardin* $C_{13}H_{19}O_9N$. All of the above glucosides yield on hydrolysis, hydrocyanic acid. The most important and best known of all the glucosides that yield hydrocyanic acid is *amygdalin.*

Greshoff discovered an amygdalin-like glucoside in two tropical trees, *Pygeum parviflorum* and *P. latifolium.* The same author found glucosides. in a member of the milkweed family *Asclepidaceae.* The *Pangium edule* of the tropics contains a large quantity of a glucoside capable of being converted into hydrocyanic acid and a large amount can be prepared from a single plant. The *Hydnocarpus inebrians* also contains a large quantity of a glucoside which yields hydrocyanic acid. It is used to destroy fish. The common linseed cake contains a glucoside which yields prussic acid. Francis found prussic acid in the sweet cassava root, .0168 per cent, and in the bitter cassava .0275 per cent. It is well known that fresh bitter cassava root is bitter poison. The above facts are brought together by Blyth in his work on poisons and may be consulted for more of the details.

The statistics on poisoning seem to indicate that it occupies third place among poisons in the order of frequency in Great Britain. In that country there are about forty deaths annually from this poison according to Blyth. It is responsible for the loss of a great many cattle in sections of the country where the wild cherries are abundant and also from sorghum poisoning. It is frequently used for criminal poisoning, at one time more frequently than now. It is nearly always taken by the mouth into the stomach, but occasionally the vapors produce death. It is generally used by Entomologists to kill insects.

Blyth gives the symptoms of poisoning as follows: Cold blooded animals require a larger relative dose than warm blooded animals except the birds which are slightly less sensitive but the action is essentially the same. Hydrocyanic acid acts in two ways:

1. It profoundly interferes in the ordinary metabolic changes in animals.
2. It causes a paralysis of the nerve centers.

Normal blood decomposes with great ease hydrogen peroxide into oxygen and water. If it is normal venus blood and a little hydrogen is added it becomes bright red, but if a trace of prussic acid be present it is a dark brown color.

The blood corpuscles lose their power of conveying oxygen to all parts of the system and asphyxia results. The main symptoms in animals are as follows:

The main differences between the symptoms induced in cold-blooded and warm-blooded animals, by a fatal dose of hydric cyanide, are as follows:

The respiration in frogs is at first somewhat dyspnoeic, then much slowed, and at length it ceases. The heart, at first slowed, later contracts irregularly, and at length gradually

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1 Proc. Roy. Soc. lxviii and lxxii (See Blyth "Poisons" p. 204).
2 Ib., lxxxiii. (See Blyth "Poisons" p. 204).
3 Journ. Cham. Soc. lxxxix. (See Blyth "Poisons" p. 204).

stops; but it may continue to beat for several minutes after the respiration has ceased. But all these progressive symptoms are without convulsion. Among warm-blooded animals, on the contrary, convulsions are constant, and the sequence of the symptoms dyspnoea, slowing of the pulse, giddiness, falling down, then convulsions with expulsion of the urine and faeces.

When the dose is short of a fatal one, the symptoms are as follows: Evident giddiness and distress; the tongue is protruded, the breath is taken in short, hurried gasps, there is salivation, and convulsions rapidly set in, preceded, it may be, by a cry. The convulsions pass into paralysis and insensibility. After remaining in ths state some time, the animal again wakes up, as it were, very often howls, and is again convulsed; finally, it sinks into a deep sleep, and wakes up well.

Dr. K. Winslow in his work on Veterinary Materia Medica and Therapeutics, gives the Toxicology of Hydrocyanic Acid as follows: "Three stages may be distinguished in fatal poisoning. First: a very short period elapses before the symptoms appear. There are giddiness, difficult breathing, and slow pulse in this stage. Second: the pupils dilate, vomiting may occur, and the animal utters loud cries. Spasmodic defacation, micturition and erections may be present, with convulsions and unconsciousness. Third: the last stage is characterized by collapse, spasms, general paralysis and death. The subacute form of poisoning may ensue and prove fatal, or, owing to the volatile character of the drug, complete recovery may take place within one-half or three-quarters of an hour. Occasionally dogs continue to be paralyzed for several days and get well. The minimum fatal dose recorded in man is 9/10 of a grain of pure acid, or about 50 drops of the medicinal solution. Four to five drachms of the diluted acid frequently, but not invariably, causes subacute poisoning and death, in horses, within an hour. One or two drachms of the pharmacopoeial preparation usually kills dogs within ten minutes."

Poisoning from Toxalbumins, Black Locust, Ricinus and Abrus.

In recent years much work has been done with a class of poisons, known as toxalbumins. These are of especial interest because many of the bacteria produce such poisons. Some of the fungi responsible for "forage poisoning" produce, it is thought, toxalbumins. In recent years a number of cases of horse poisoning from Black Locust bark have been reported. The poisoning from castor oil bean (*Ricinus*) and from Abrus are also of this class.

Castor Oil Seed and Abrus. One of the best known of the toxalbumins is that occurring in the castor oil seed, known as *ricin*. This albuminous substance is very poisonous, more so than strychnin and prussic acid. Ricin coagulates the blood. Blyth in his work on poisons states:

> If castor-oil seeds are eaten, a portion of the poison is destroyed by the digestive processes; a part is not thus destroyed, but is absorbed, and produces in the blood-vessels its coagulating property. Where this takes place, ulcers naturally form, because isolated small areas are deprived of their blood supply. These areas thus becoming dead, may be digested by the gastric or intestinal fluids. and thus, weeks after, death may be produced. The symptoms noted are nausea, vomiting, colic, diarrhoea, tenesmus, thirst, hot skin, frequent pulse, sweats, headache, jaundice, and death in convulsions or from exhaustion. Animals may be made immune by feeding them carefully with small doses, gradually increased.
>
> The post-mortem appearances are ulceration in the stomach and intestines. In animals the appearances of haemorrhagic gastro-enteritis with diffuse nephritis, haemorrhages in the mesentery, and so forth have been found.

A toxalbumin also occurs in the Jequirity seed (*Abrus precatorius*) which causes similar effects and symptoms. That the poisons are not the same have been shown by experiments with animals. It is known that animals may become immune by repeated doses of Jequirity against *abrin* and the principle of castor oil does not produce immunity against abrin, nor does abrin confer immunity against the ricin of the castor oil bean. The abrin when applied to the con-

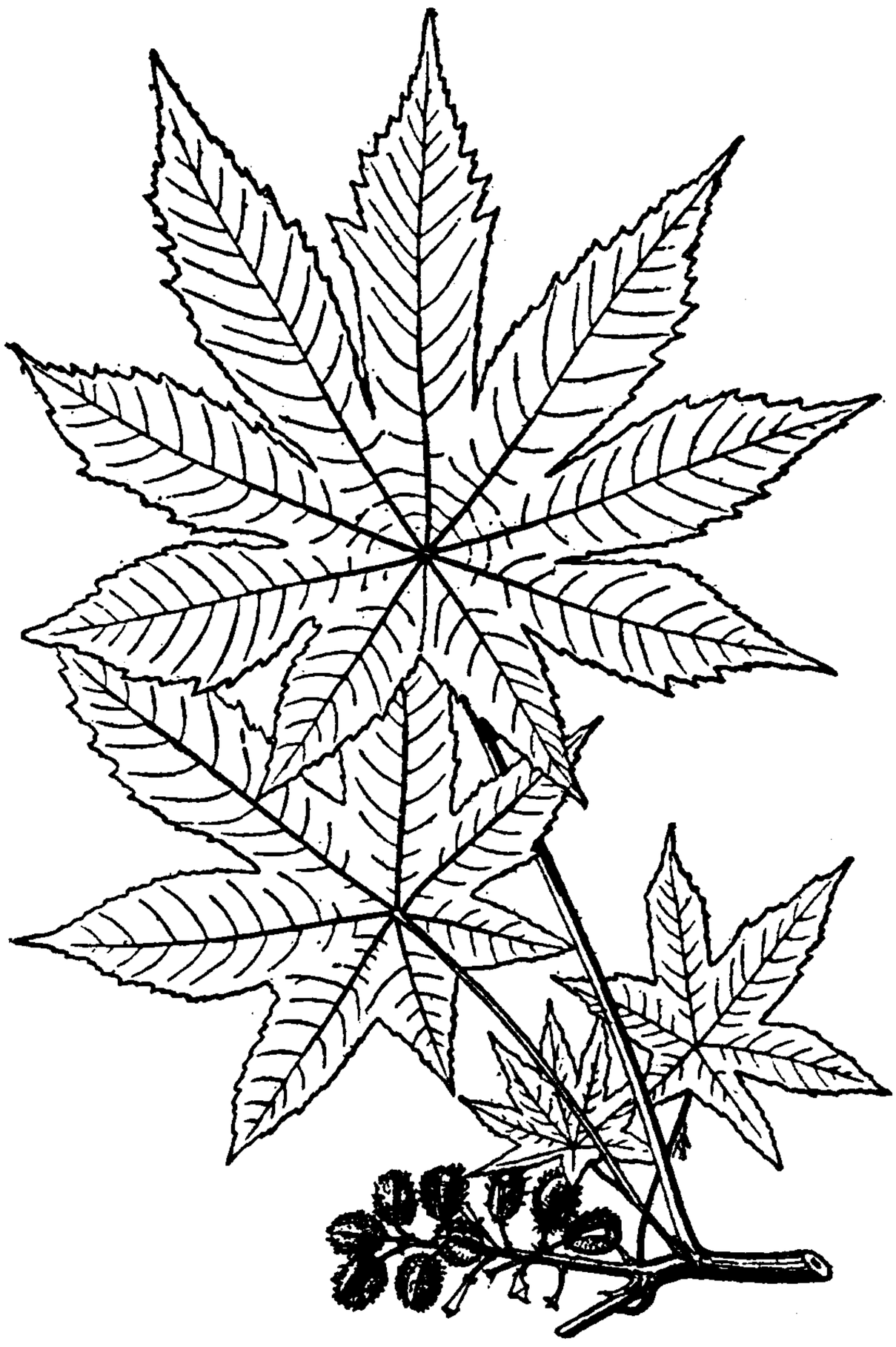

Fig. 16b. Castor oil plant (*Ricinus communis*). The seeds furnish the castor oil of commerce, and also contain an acrid poison. (Chestnut, U. S. Dept. Agr.).

junctiva causes coagulation in the vessels and a secondary inflamation. The disease is known as Jequirity opthalmia. More details in regard to the poisons of these plants are given under the plants of the families in which they occur.

Black Locust Poisoning. The Black Locust which is commonly planted as an ornamental tree has in a number of instances caused death.

Dr. Waldron in the American Veterinary Rewiew, writes thus of locust bark poisoning, referring especially to the beating of the heart of a horse that had been poisoned by the locust bark. This beating shook the horse and could be heard outside the stable.

The sound was caused by the action of the diaphragm. It was greater when the ribs were at their fullest expansion and could be heard most distinctly at a distance of ten feet. I tried to locate or rather find out what produced the sound, but in that I am as ignorant as I was then. . . . In questioning where the team was hitched at the mill, it was found that the driver had tied them to a young locust tree that had been cut down a few years before. This was a sapling of about four inches in diameter and had probably made a very rapid growth and the bark, from this reason was tender and easily peeled. They had done a good job of peeling, but as they had their bits in they were not able to swallow much. The poison obtained from this bark is, in my opinion, the cause of the trouble.

Dr. Waldron says that the symptoms otherwise are about the same as those occurring in cases of belladonna poisoning and are about as follows:

Extreme lassitude, which includes almost imperceptible pulse and which, when found, is weak and prolonged; respiration less than normal by one-third and sonorous; temperature normal; no pain, no appetite, mucous membrane congested, of a blue, rusty, or yellow color. Mucous membrane of the mouth some swollen, caused by the congestion of the capillaries; slight ptyalism, and above all, the dilation of the pupil of the eye; in fact I should judge, we have nearly the same symptoms we get in belladonna poison.

He also says that he had not known before that locust bark was poisonous and although he had searched for literature upon the subject had found but one reference, that being in the U. S. Pharmacopoeia ,which records a case reported in Jan., 1887, when 33 children were said to have been poisoned by chewing locust bark. In mild cases there were "flushed faces, dryness of the throat and mouth, and dilation of the pupils. In severe cases, were added epigastric pain, extremely intermittent heartbeats, and stupor." It is evident from the fact that there is not much literature on the subject that such poisoning does not occur often.

Dr. H. S. Murphy has kindly contributed the following case on locust bark poisoning:

Anamusis: Gray mare twelve years old, pregnant ten months, has been at light work

eously until four doses are given, also ξ I potassi iodidum is given per orem at once and the following prescription left.

Ex Strychnini sulphatis gr. IV.
Fld. ext. digitalis ξI.
Potassi iodidum II.
Aqua ξXVI.
Sig.: A table spoon full every 2 hours.

Result: After the second hypodermic was given a slight improvement noticed (lessening in force and frequency of thud, which was quite marked after fourth hypodermic. Owner reports thud gone at end of 24 hours but stupidity still present though not so marked.

Recovery uneventful and at end of gestation parturition proceeds normally, and foal lives.

Toxicity of bark proven in the following manner: Two cc of 10% tincture (bark grated) killed half grown kittens which showed above symptoms in an aggravated form.

One ounce necessary to kill a 25 pound dog.

This tincture dropped in eye acted much as atropin but no toxic symptoms developed. Simply a dilation of pupil. Grated root in lard produced slight symptoms in two dogs. P. m. on cats revealed a generalized dirty m. m. but only a very slight yellow on cartilages. Swollen liver and a few petechia on serous membranes. Blood quite dark.

The cadaver, resembled somewhat that of one dead of septicaemia.

CHAPTER IX

POISONING FROM OPIUM, SOLANACEAE AND PLANTS THAT CONTAIN SAPONINS.

The use of opium by Chinese and other races is as Flückiger and Hanbury say, "in the words of Pereira, the most important and valuable used in medicine of the whole Materia Medica; and we may add, the source by its judicious employment of more happiness by mankind." Blyth in his work on Poisons, states that in England and Wales 1505 deaths were attributed to the use of opium or its active constituents between the years 1898-1903. Of these 882 were accidental or because of negligence, 621 were suicidal. In France opium and morphin poisons are said to cause about 1 per cent of the cases of poisoning. Various patent medicines contain opium or some of its products and in the past have been the cause of frequent cases of poisoning. The use of the drug in patent medicines for children in the United States in the form of soothing syrups was once more common than now. The use of opium for infants is a common practice in India, according to Blyth who quotes from Dr. Chevers.[1]

In general the opium and morphin poisoning are as follows: The beats of the heart are at first accelerated and then diminished. Large doses introduced into the circulation diminish the pulsations without acceleration and may even cause heart paralysis. "The arterial blood pressure, at first increased is afterwards diminished. If morphin is in sufficient quantity thrown into the circulation, then tetanus at once occurs. Depression and stimulation depend on dosage. The common form occurring in 99 per cent of the cases; excitement, narcosis, and coma, bowels nearly always constipated. (2) A very sudden form in which death occurs rapidly, the person sinks into a deep sleep almost immediately. (2) An abnormal form in which there is no coma but convulsions.

Blyth in referring to opium eating says:

> The consumption of opium is a very ancient practice among Eastern nations, and the picture, drawn by novelist and traveler, of poor, dried-up, yellow mortals addicted to this vice, with their faculties torpid, their skin hanging in wrinkles on their wasted bodies, the conjunctivae tinged with bile, the bowels so inactive that there is scarcely an excretion in the course of a week, the mental faculties verging on idiocy and imbecility, is only true of a percentage of those who are addicted to the habit.

In the case of opium poisoning the stomach tube should be used to empty the stomach, and wash with warm water, then coffee may be given. Permanganate of potash is a perfect antidote and should be given when at hand.

The alkaloid codein also found in opium produces sleep but its effects are different. Large enough doses produce death and cause epilepti-form convulsions. Thebain found in opium produces symptoms that resemble those produced by strychnin, namely tetanic spasms. Apomorphin found in opium is an active emetic. Papaverin causes paralysis of respiration in guinea pigs.

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1 Jurisprudence 232 (3rd ed.).

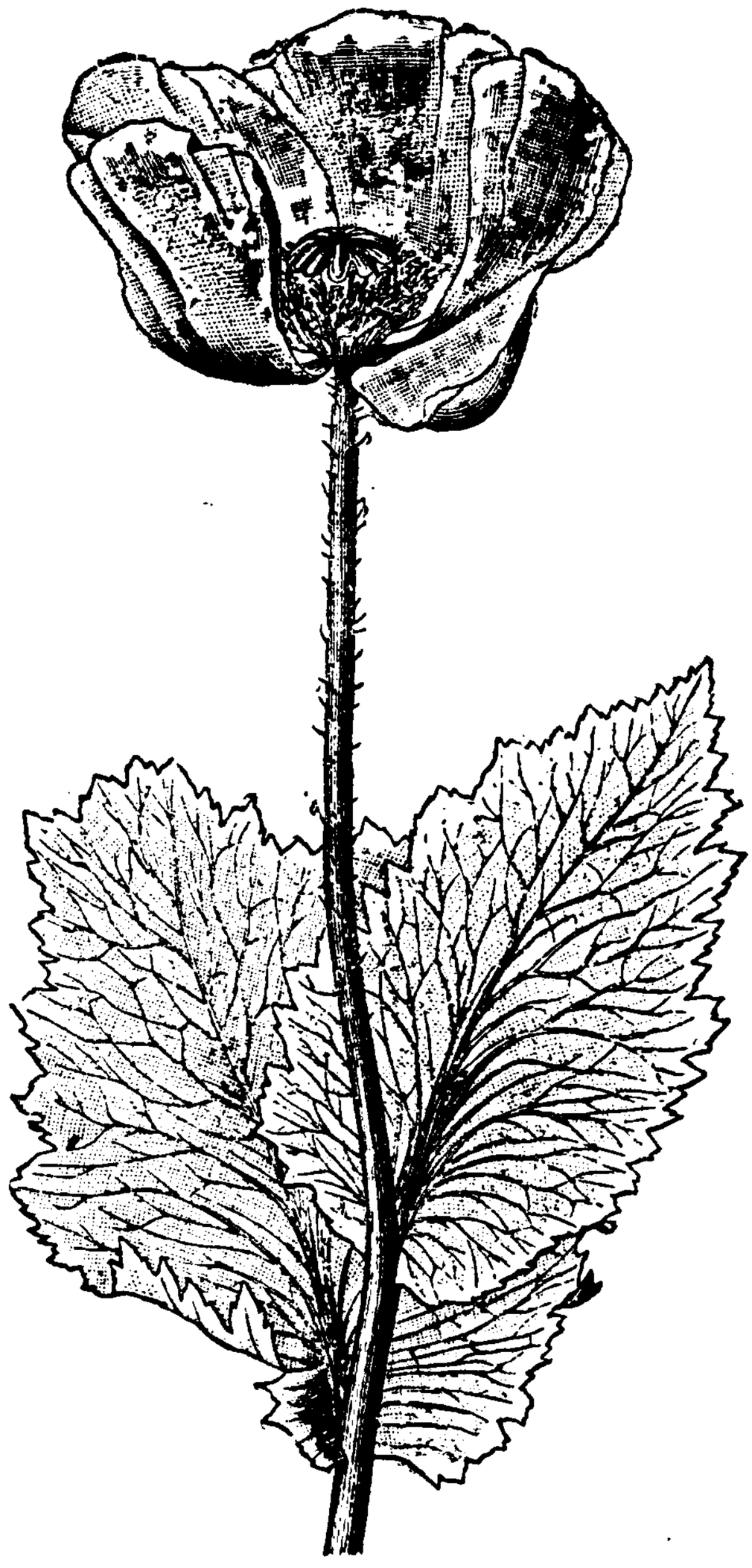

Fig. 16d. Garden Poppy (*Papaver somniferum*). A poisonous plant producing a large number of alkaloids like morphin, codein, etc., (Strasburger, Noll, Schenck and Schimper).

Poisoning from Solanaceae.

A number of plants of the Solanaceae are known to be poisonous; among them the common thorn-apple or Jimson weed (*Datura Stramonium*), the atropa (*Atropa Belladonna*) and hyoscyamin (*Hyoscyamus niger*) besides such suspected plants as the common black nightshade (*Solanum nigrum*), horse nettle (*Solanum carolinense*), bittersweet (*Solanum dulcamara*) and scopiola. The cases of poisoning from atropin are more frequent, perhaps, than statistics seem to indicate. The English death statistics for ten years, ending 1903, according to Blyth show 95 per cent of the deaths from atropin; 35 per cent were suicidal. Most of the accidental cases arise from mistakes made by the pharmacist or physician. Criminal poisoning is carried on to a less extent in Europe and America than in India. Blyth states that of the 120 cases recorded in works on Indian toxicology no less than 63 per cent were criminals, 19 per cent suicidal, and 18 per cent accidental. The most important alkaloids found are *atropin, hyoscyamin, scopalamin* and *solanin.* Solanin is poisonous and is regarded as a nitrogenised glucoside. In man the symptoms of atropin poisoning are: Dilating of the pupils, dryness of the mouth and throat; the

mucous membrane is reddened, inability to swallow, deranged vision, breath at first a little slow and then rapid; the nervous system is affected in a marked degree; the lower extremities are often partly paralyzed. There is want of coördination. "The person reels like a drunken man." In adults this takes on a hilarious pleasing form. The symptoms of poisoning from hyoscyamin are similar to those of atropin. The absence of delirium and excitement, however, makes it decidedly different. The symptoms in animals for both of these substances do not differ essentially from those given above

Fig. 16e. Nightshade (*Solanum nigrum*) From Darlington's Weeds and Useful Plants.

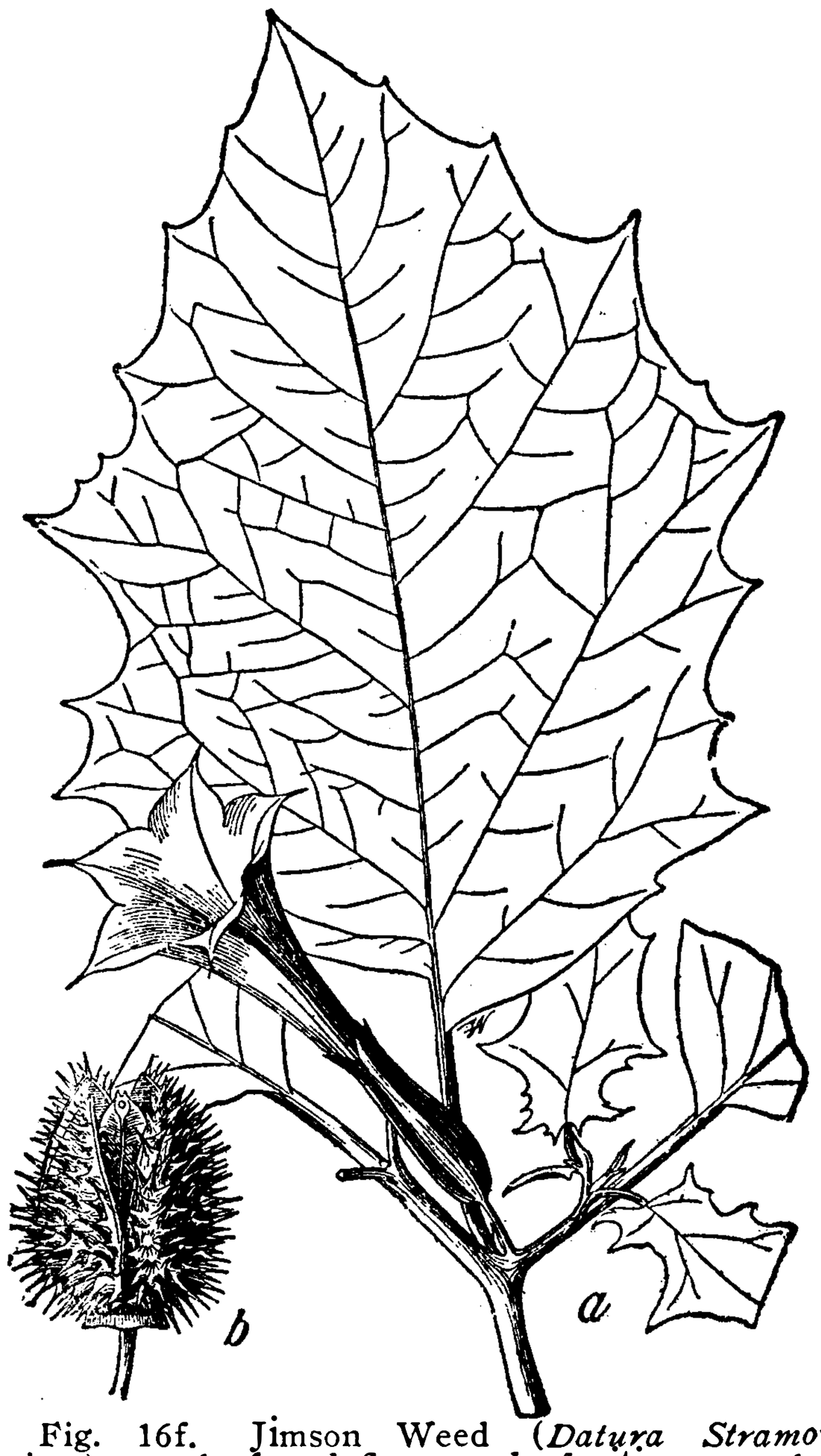

Fig. 16f. Jimson Weed (*Datura Stramonium*). *a*, leaf and flowers; *b*, fruiting capsule. (U. S. Dept. Agrl.).

Atropin may be absorbed by the skin, and enough may be absorbed, if it is broken, to cause death. Blyth quotes Ploss[1] to the effect that atropin sulfate applied as an ointment to the abraded skin was fatal. Atropin has also been absorbed from the bowel as recorded by Blyth:

A clyster containing the active principles of 5.2 grms. (80 grains) of belladonna root was administered to a woman 27 years of age, and caused death. Allowing the root to have been carefully dried, and to contain .21 per cent of alkaloid, it would seem that so little as 10.9 mgrms. (.16 grains) may even prove fatal, if left in contact with the intestinal mucous membrane. Belladonna berries and stramonium leaves and seeds are eaten occasionally by children. A remarkable series of poisoning by belladonna berries occurred in London during the autumn of 1846.

1 Zeitschr f. Chir. 1863. Blyth, Poisons; Their Effects and Detection.

Poisoning from Plants that contain Saponin.

In recent years our knowledge of the Saponins has been greatly extended; many of these studies have been made by Kobert or his students in the laboratory at Dorpat. The term saponin has been applied to a class of substances of a glucosidal nature which are poisonous and when dissolved in water form a solution which froths much like soap-suds. These substances are not all the same chemically, but have the general formula $C^nH^{2n}O^{10}$. Blyth gives the following list with their formulae:

Saponin 1.

Saponin senegin
Quillaja sapotoxin
Sapindus sapotoxin
Gypsophila sapotoxin
Agrostemma sapotoxin } $C_{17}H_{26}O_{10}$.

Saponin 2.

Asamin
Digitonin saporubrin } $C_{18}H_{28}O_{10}$.

Saponin 3.

Quillagic acid
Polygalic acid } $C_{19}H_{30}O_{10}$.
Herniaria saponin

Cyclamin
Sarsaparilla saponin } $C_{20}H_{32}O_{10}$.

Sarsa saponin..... $C_{22}H_{36}O_{10}$
Parillin ..... $C_{26}H_{44}O_{10}$
Melanthin ..... $C_{29}H_{50}O_{10}$

The suggestion is made that possibly dulcamarin $C_{22}H_{34}O_{10}$, and syringin $C_{17}H_{50}O_{10}$ may belong to the same series.

One of the oldest of the known saponins was isolated from the Bouncing Betty, *Saponaria officinalis*, and later from the corn cockle *Agrostemma Githago* and many other plants. This saponin is a white amorphous powder, very soluble in water, is neutral and reäcts without odor; it causes sneezing when applied to the mucous membrane of the nose; tastes at first sweetish, then becomes sharp and acrid. The saponin when rubbed on the skin exerts no action because not absorbed; when injected subcutaneously into frogs it becomes quickly absorbed and acts upon the nerves and muscles. In warm blooded animals there is little or no absorption because of an aseptic abscess which forms. Intravenous injections in small amounts in the laboratory of Kobert proved fatal for cats and dogs. It acts injuriously on the striated muscle and heart muscle. The sensor and motor nerve fibers are also affected in a serious way. On the digestive tract it causes inflammation and peristalsis. The saponin substances dissolve the blood corpuscles of all animals and thus penetrate the corpuscles. It is thought that the haemolytic action of these substances is due to the liquefaction of the cell membrane.

Ransom found that the saponin may become bound to the corpuscles and the serum. That this action depends on the cholosterin, saponin so bound will not act on the red corpuscles. The saponin cholosterin mixture exerts no action on dog's blood.[1] Dr. R. F. Bacon and H. T. Marshall who made a study

[1] The toxic action of saponin. Phil. Jour. Sci. 1:1037. Dr. R. F. Bacon and H. T. Marshall.

of the saponin found in *Entada scandens* proved that it was highly toxic for rabbits and guinea pigs.

When diluted with normal salt solution to a concentrated form 1-200 and injected into the peritoneal cavity, 2-5 mgrms. of saponin to 100 grms. of animal was uniformily fatal, while a quarter of this amount killed in several cases. Where smaller quantities were used and animals living for a longer period of time localized peritotinitis was found.

"The saponin is powerfully haemolytic" 0.005 cubic cm. of ½ of 1% solution (0.025 mgrms.) completely dissolved one cubic cm. of a 5% suspension of rabbits corpuscles which were freed from serum and the half of this amount produced haemolysis of the serum of the free corpuscles of the guinea pig. "Saponin, however, loses its haemolytic power after the addition of serum." Immunity could not be produced in rabbits or guinea pigs recently treated with intraperitoneal doses of saponin or saponin serum mixture.

Blyth has studied the general action of saponin on kittens. He states that when 13 to 22 mm. (1/5 to ½ gr.) is injected underneath the skin of a kitten immediately symptoms of local pain occur, in 5 to 10 minutes the respiration is quicker and the animal falls into a lethargic condition with signs of muscular weakness; just before death breathing became rapid with all the signs of asphyxia. The appearances after death were fullness in the right side of the heart and congestion of the intestinal canal. In man the taking of saponin causes an increase of mucus secretion and nausea.

Saponin or saponin-like substances occur in the following families:

Phytolaccaceae, (*Phytolacca abyssinica*); *Caryophyllaceae,* (*Gypsophila struthium, Agrostemma Githago, Lychnis, Saponaria officinalis, S. rubra, Herniaria*); *Berberidaceae,* (*Caulophyllum thalictroides*); *Leguminosae,* (*Entada scandens, Gymnocladus dioica, Gleditschia, Enterolobium*): *Oleaceae,* (*Chionanthus virginica, Syringa vulgaris*); *Amaryllidaceae,* (*Agave*); *Liliaceae,* (*Yucca glauca?, Chlorogalum pomeridianum*); *Rosaceae,* (*Quillaja Saponaria*); *Sapindaceae,* (*Sapindus trifoliatus, S. saponaria, Pometia pinnata, Magonia*); *Hippocastanaceae,* (*Aescula Hippocastanum, A. Pavia*); *Theaceae,* (*Thea Sasanqua, T. assanica*); *Sapotaceae,* (*Omphalocarpum procerum*); *Polygalaceae,* (*Polygala Senega*).

In all more than 200 species of plants contain saponin.

See Chapter XIV, and Pt. II, for a list of others.

CHAPTER X

POISONING FROM FLOWERS, POISONING FROM HONEY, MECHANICAL INJURIES

Poisoning from Flowers. The odors from a large number of flowers are more or less injurious, especially to some individuals. The flowers of the common wild black cherry (*Prunus serotina*) when past their prime give off a cyanogentic odor that is quite objectionable to many people, causing headache. The carion-like odors, like those produced by *Stapelia*, the carion flower (*Smilax herbacea*) and *Aristolochia* are sickening to many people, causing headache and a depressing feeling. Many flowers that are ordinarily sweet scented like the tuberose (*Polyanthes tuberosa*) often give people the headache if the flowers are abundant in the room they are sitting in. The flowers of Wistaria are injurious to some people. The flowers of *Magnolia grandiflora* are "overpowering" according to some authorities.

Poisoning from Honey. The honey obtained from the flowers of some plants is said to be poisonous; for instance the honey collected by insects from the oleander was long ago recorded as being injurious, and it is said that the honey collected from the mountain laurel (*Kalmia latifolia*) appears to be poisonous under some conditions according to Chesnut. According to Chesnut the honey collected from the flowers of the snow-on-the-mountain, (*Euphorbia marginata*) is bitter and disagreeable, but does not appear to be a serious poison. Several cases of poisoning from the nectar of the flowers of *Datura metel* and *D. Wrightii* are on record and the flowers of the Brazilian magonia of the family *Sapindaceae* produces poisonous honey; also the flowers of Rhododendron are said to contain andromedotoxin. Toxic honey has also been gathered from the flowers of Black Locust (*Robinia pseud-acacia*) and Lily of the Valley (*Convallaria majalis*).

Prof. Lyman F. Kebler who has made a somewhat extended investigation with poisonous honey[1] has given an excellent bibliography with reference to the earlier literature on the subject. It has been known for centuries that the honey collected from *Ericaceae* acts as a narcotic irritant, producing giddiness, vomiting, and purging. Poisonous honey was described by Xenophon. He gives a fairly accurate description of how the soldiers of his army acted that ate honey that was poisoned. He states that they lost their senses, vomited and were affected with purging, and those who had eaten but little were intoxicated, but when they had eaten much they were like mad men. Strabo and Pliny spoke of poisonous honey, the latter writer, an early naturalist noted for his accurate observations, records poisonous honey which he called "aegolethron" (goat's death), which bees collected at Heraclea. He gives a description of the honey which is said to have had a peculiar smell and produced sneezing. It is generally supposed that this honey came from a species of *Rhododendron,* the *R. pontica.* This and allied species are the chief source of poisonous honey in Asia and Asia Minor,

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1 Poisonous Honey. Proc. Amer. Pharm. Assoc. 1896: 167-173.

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Fig. 16g. The flowers of Black Locust (*Robinia pseud-acacia*) produces a toxic nectar. (Ada Hayden).

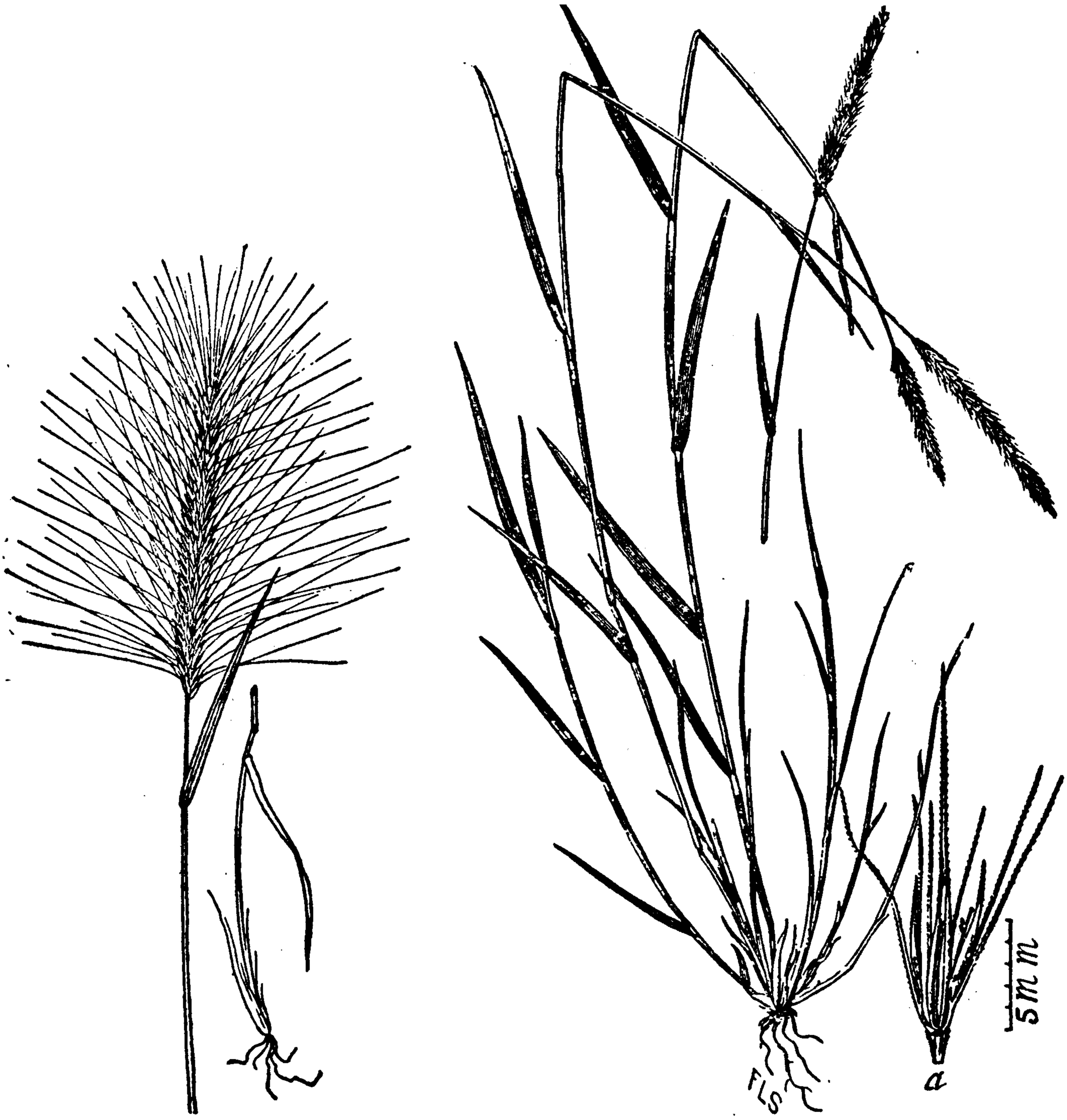
Fig. 16h. The wilted and old flowers of Wild Cherry (*Prunus*) produce a cyanogentic poisonous odor. (Ada Hayden).

Mechanical Injuries

We have a long list of plants that act injuriously in a mechanical way. Among the best known of these are Wild Barley or Squirrel-tail Grass (*Hordeum jubatum*) and the related species, which, by mechanical means, injure sheep, horses, and cattle. The awned heads, when eaten with hay or grass, break up into sections, the awns working their way into the mucous membrane, insinuating themselves around the teeth, thus causing inflammation and deep ulcerating sores, with the formation of pus. The teeth may consequently become loosened and fall out.

A Cheat or Brome Grass (*Bromus tectorum*) which is common in Utah, parts of Colorado, and westward, produces similar injuries.

The Needle Grass, common in the dry gravelly hills and sandy plains of Northern Mississippi Valley has a fruit with a sharp pointed callus, with hairs above the pointed callus projecting upwards. The sharp-pointed callus of the "seed" enters the skin, especially of sheep, where it produces an irritation which is sometimes followed by death. This has sometimes been eaten with forage, thus entering the intestinal tract, perhaps perforating it, causing death, especially when they have pierced the walls of the intestines. Several allied species as Black Oat (*Stipa avenacea*) produce similar injuries. The Western Needle Grass (*Stipa comata*), however, is less injurious than our Common Needle Grass.



(a) (b)

Fig. 16i. Wild Barley (*Hordeum jubatum*). b. Wild Barley (*H. nodosum*), cause of mechanical injuries to animals.

Similar and allied species in other parts of the world are known to be injurious in the same way. Other plants are injurious by means of their sharp-pointed fruit. Among those of the last named type are members of the Geranium family notably the Stork's bill (*Erodium cicutarium*), common in the west, which frequently gets into the wool of sheep and may produce local irritation. Mechanical injuries are also produced by the Sand Bur (*Cenchrus tribuloides*) whose spiny involucre may work into the flesh of animals and induce an irritation followed by inflammation, and the formation of pus. Hogs and cattle often receive mechanical injuries from the Cocklebur (*Xanthium canadense*) and allied species.

A recent number of Horticulture (Jan. 1, 1910) notes that rose thorns have caused the poisoning of the hands of clerks who handle the roses.

Prof. J. Davy states that in South Africa the burrs of a clover *Trifolium terrestris* var *hispidissimus* are said to injure young lambs.

The Burdock (*Lappa major*) sometimes produces no little irritation, and the same may be said of the Spanish Needle (*Bidens frondosa*). The sharp, stiff

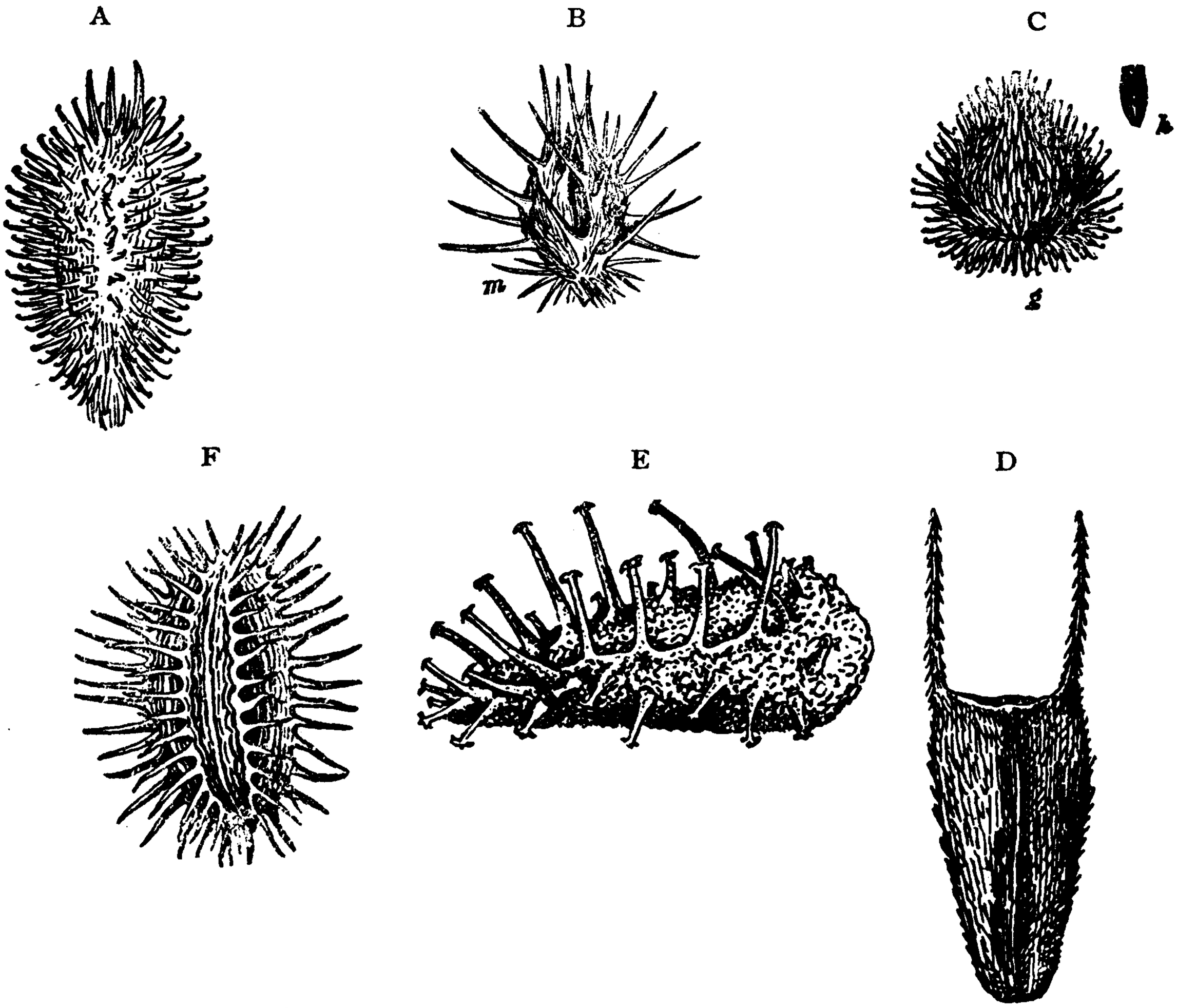


Fig. 17. A. Fruit of the Cocklebur (*Xanthium canadense*), causes mechanical injuries to hogs when eaten by them. B. Sandbur (*Cenchrus tribuloides*). C. Burdock (*Arctium major*). D. Boot Jack (*Bidens frondosa*). E. One of the Borages (*Cynoglossum*). F. Carrot (*Daucus Carota*).

branches of Greasewood (*Sarcobatus vermiculatus*) easily penetrate the skin and sometimes induce pus infection. The bristles and spines of the rose and stiff bracts of the Russian Thistle may be the cause of injury to animals and men. The small, barbed trichomes of the calyx of Crimson Clover (*Trifolium incarnatum*), according to Prof. Coville, produce phytobezoars similar to those commonly produced by hair. Dr. Trelease has described similar bezoars from the barbed trichomes and spines of cacti. Millet and barley awns are known to produce similar balls in horses as well as in cattle. Corn stalks, when eaten with an insufficient amount of water, produce impaction. This has, however, been attributed to other causes also. Bloat is known to follow the use of such forage as white and red clover, alfalfa, pigweed and many other plants consumed under the same conditions.

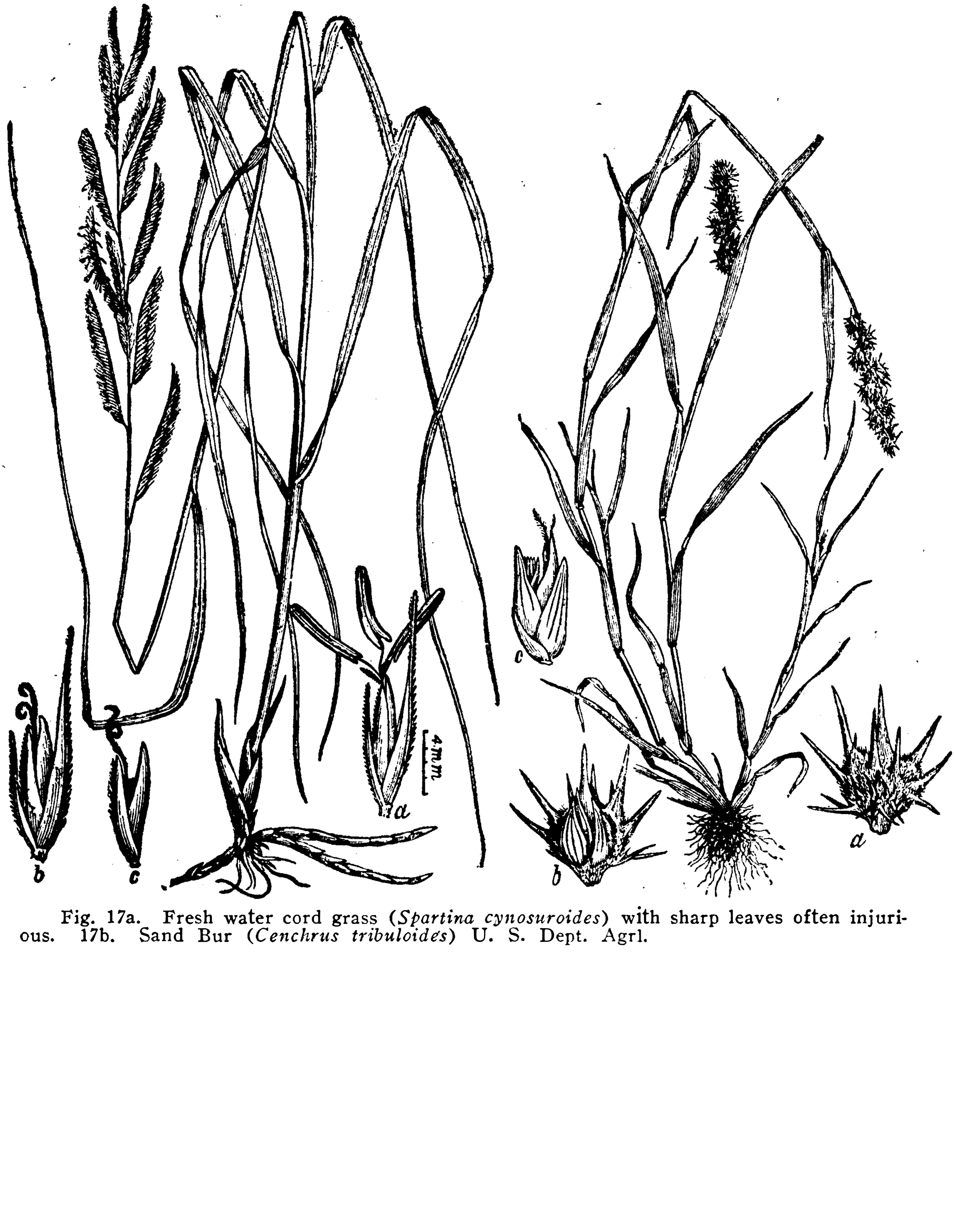


Fig. 17a. Fresh water cord grass (*Spartina cynosuroides*) with sharp leaves often injurious. 17b. Sand Bur (*Cenchrus tribuloides*) U. S. Dept. Agrl.

Fig. 18. Needle Grass (*Stipa comata*). U. S. Dept. Agrl. Fig. 18a. Needle (*Stipa spartea*) inflicting mechanical injuries. U. S. Dept. Agrl.

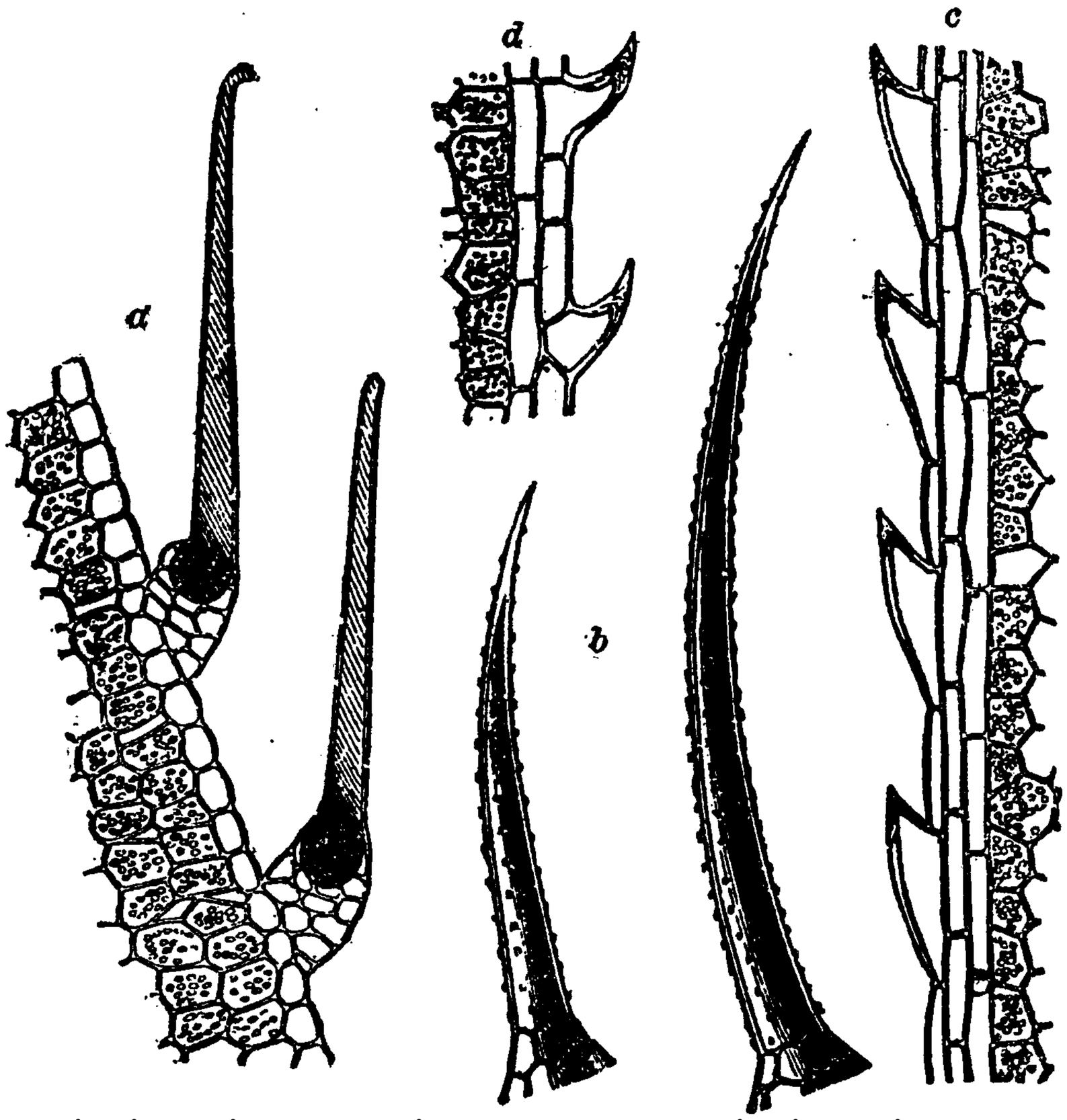


Fig. 18b. Urticating hairs and cutting leaves. *a*. Urticating hair of nettle. *b*. Bristles of bugloss. *c*. barbed margin of a leaf of sedge. *d*. barbed margin of a leaf of grass.

CHAPTER XI

CLASSIFICATION OF POISONS, SYMPTOMS AND ANTIDOTES

Blyth classifies poisons as follows:

A. POISONS CAUSING DEATH IMMEDIATELY OR IN A FEW MINUTES. Prussic acid, cyanides, oxalic acid and occasionally, strychnin.

B. IRRITANT POISONS. Symptoms mainly pain, vomiting, and purging. Savin, ergot, digitalis, colchicum, yew, laburnum, and putrid substances.

C. IRRITANT AND NARCOTIC POISONS. Symptoms of an irritant nature, with more or less cerebral indications. Oxalic acid or oxalates.

D. POISONS MORE ESPECIALLY AFFECTING THE NERVOUS SYSTEM.

1. Narcotics. Chief symptoms: insensibility which may be preceded by more or less cerebral excitement. Opium.

2. Deliriants. Delirium, for the most part, a prominent symptom: Belladonna, hyoscyamus, stramonium, and other Solanaceae, poisonous fungi, Indian hemp (*Cannabis*), darnel (*Lolium temulentum*), camphor and *Oenanthe crocata.*

3. Convulsives. Almost every poison has been known to produce convulsive effects, but the only true convulsive poisons are the alkaloids of the strychnin class.

4. Complex nervous phenomena. Aconite, digitalis, poison hemlock, Calabar bean, tobacco, *Lobelia inflata,* and curare.

Kobert's classification is as follows:

I. POISONS WHICH CAUSE COARSE ANATOMICAL CHANGES OF THE ORGANS.

A. Those which especially irritate the part to which they are applied.

1. *Acids.*
2. *Caustic alkalies.*
3. *Caustic salts,* especially those of the heavy metals.
4. Locally irritating organic substances which neither can be classified as corrosive acids nor alkalies, nor as corrosive salts; such are: *cantharadin phrynin,* and others in the animal kingdom, *croton oil* and *savin* in the vegetable kingdom; locally irritating colors, such as the *anilin dyes.*
5. Gases and vapors which cause local irritation when breathed; such as *ammonia, chlorin, iodin, bromin, and sulphur dioxid.*

B. Those which have but little effect locally, but change anatomically other parts of the body; such as lead, phosphorus, and others.

II. BLOOD POISONS.

1. Blood poisons interfering with the circulation in a purely physical manner; such as *peroxid of hydrogen, ricin, abrin.*
2. Poisons which have the property of dissolving the red corpuscles; such as the *saponins.*
3. Poisons which, with or without primary solution of the red blood corpuscles, produce in the blood methaemoglobin; such as *potassic chlorate, hydrazine, nitrobenzene, anilin, picric acid, carbon disulphid.*

Treatment. (Immediate:)
EMETIC and STOMACH TUBE.
Pilocarpin Nitrate, one-third of a grain subcutaneously, repeated if necessary.
Ammonia or Amyl Nitrite to nostrils.
TANNIN.
(Later:)
Stimulants, Castor Oil.
DEMULCENTS (swallowed and injected).
Note—Muscarin poisoning give Belladonna Tincture B. P. fifteen minims
Plants furnishing poisons that act as deliriants—Thorn Apple (*Datura Stramonium*). Black Nightshade (*Solanum nigrum*), Hemp (*Cannabis sativa*), Darnel (*Lolium temulentum*), Several Fungi as Fly Agaric (*Amanita muscaria*).

III

INEBRIANTS

Symptoms. Excitement of cerebral functions, and of the circulation; loss of power of co-ordination, and of muscular movements, with double vision; leading to profound sleep, and deep coma.
Treatment. (Immediate:)
EMETIC and STOMACH TUBE.
TANNIN.
(Later:)
Epsom Salts.
DEMULCENTS (swallowed and injected).
Plants furnishing poisons that act on the brain as inebriants: Wormwood (*Artemisia Absinthium*), Jamaica Dogwood (*Piscidia Erythrina*).

POISONS ACTING ON THE SPINAL CORD

CONVULSIVES

Symptoms. Clonic (intermittant) spasms, extending from above downwards. Opisthotonos very violent; but trismus (lock-jaw) rare. Swallowing' spasmodic. Death, usually, in less than three hours, or rapid recovery.
Treatment. (Immediate:)
EMETIC and STOMACH TUBE. TANNIN.
Chloral Hydrate five grains subcutaneously (a weak solution as it is an irritant), repeated if necessary. Chloroform inhalation. Artificial respiration.
Potassium Bromide, one drachm in water every half-hour. Morphia (?).
(Later:)
DEMULCENTS (swallowed and injected).
Castor Oil. Chloroform inhalation if convulsions return.
Plants furnishing poisons that act upon the spinal cord as convulsives: Nux-vomica Tree (*Strychnos Nux-vomica*), St. Ignatius's Bean (*Strychnos Ignatii*).

POISONS ACTING ON THE HEART

I

DEPRESSANTS

Symptoms. Vertigo; vomiting; abdominal pain; confused vision; convulsions; occasional delirium; paralysis; syncope; sometimes asphyxia.

Treatment. (Immediate:)
EMETIC and STOMACH TUBE. TANNIN.
STIMULANTS. Strychnin solution B. P., two minims subcutaneously.
Atropin, half a grain subcutaneously, repeated if necessary.
(Later:)
Stimulants. Hot fomentations.
Artificial respiration if necessary. Castor Oil.
DEMULCENTS (swallowed and injected).

Plants furnishing poisons that act on the heart as depressants: Tobacco (*Nicotiana Tabacum*), Hemlock (*Conium maculatum*), Indian Tobacco (*Lobelia inflata*).

II

ASTHENICS

Symptoms. Numbness, and tingling in the mouth; abdominal pain; vertigo; vomiting; purging; tremor; occasional delirium; paralysis; dyspnoea, ending in syncope.

Treatment. (Immediate:)
EMETIC and STOMACH TUBE. TANNIN.
STIMULANTS. Cold affusion.
Faradic current. Atropin, half a grain subcutaneously.
(Later:)
DEMULCENTS (swallowed and injected).
Continued recumbent position.
Artificial respiration if necessary. Castor Oil.

Note—In Aconite poisoning inject Digitalis Tincture B. P., twenty minims subcutaneously (12½ per cent strength).

Plants furnishing poisons that act on the heart as asthenics: Lima Bean (*Phaseolus lunatus*), Tapioca (*Jatropha manihot*), Aconite (*Aconitum Napellus*), Cohosh (*Cimicifuga racemosa*), Oleander (*Nerium Oleander*), Fox-glove (*Digitalis purpurea*), White Hellebore (*Veratrum album*), Green Hellebore (*Veratrum viride*).

VEGETABLE IRRITANTS

I

PURGATIVES

Symptoms. Abdominal pain; vomiting, and purging; cramps; strangury and tenesmus, followed by collapse, and sometimes accompanied by drowsiness, and slight nervous symptoms.

Treatment. (Immediate:)
EMETIC and STOMACH TUBE. TANNIN.
(Later:)
Opium to relieve pain.
Stimulants to counteract collapse.
DEMULCENTS (swallowed and injected).

Plants producing poisons that act as purgatives: Castor Bean (*Ricinus communis*), Green Hellebore (*Helleborus viridis*), May Apple (*Podophyllum peltatum*), Marsh Marigold (*Caltha palustris*).

II

ABORTIVES

Symptoms. Nausea; vomiting; stupor; polyuria; sometimes tenesmus. Abortion may or may not occur; coma.

Treatment. (Immediate:)

EMETIC and STOMACH TUBE. TANNIN.

Ammonia or Amyl Nitrite to nostrils.

(Later:)

Opium to relieve pain.

Stimulants to counteract collapse.

DEMULCENTS (swallowed and injected).

Plants producing poisons that act as abortives: Ergot (*Claviceps purpurea*), Herb of Grace (*Ruta graveolens*), Cotton root (*Gossypium herbaceum*), Pulsatilla (*Anemone patens*, and its variety).

III

IRRITANTS WITH NERVOUS SYMPTOMS

Symptoms. Abdominal pain; vomiting and purging; dilated pupils; headache; tetanic spasms; occasional convulsions; sometimes rapid coma.

Treatment. (Immediate:)

EMETIC and STOMACH TUBE. TANNIN.

Opium to relieve pain.

Stimulants to counteract collapse.

Bleeding if necessary.

(Later:)

Castor Oil.

DEMULCENTS (swallowed and injected).

Plants furnishing poisons that act as irritants, causing also nervous symptoms: Indian Pink (*Spigelia marilandica*), Cut-leaved Water Parsnip (*Berula erecta*), Fool's Parsley (*Aethusa Cynapium*).

IV

SIMPLE IRRITANTS

Symptoms. Burning pain in the throat and stomach; thirst; nausea; vomiting; tenesmus; purging; dysuria; dyspnoea and cough occasionally. Death through shock; convulsions; exhaustion; or starvation due to injury to throat or stomach.

Some few (i. e. the Nettles) cause smarting pain on the merest contact with the secretions of the plant; quickly followed by erythema and urticarial rash, which slowly subsides.

Treatment. (Immediate:)

EMETIC and STOMACH TUBE. TANNIN.

Opium to relieve pain.

Stimulants to counteract collapse.

Chloral and Bromides if convulsions.

(Later:)

Castor Oil.

DEMULCENTS (swallowed and injected).

Plants producing poisons that cause the above symptoms: Arrow Root

(*Arum maculatum*), Wood Anemone (*Anemone nemorosa*), Cursed Crowfoot (*Ranunculus sceleratus*), Buttercup (*Ranunculus acris*), Bouncing Betty (*Saponaria officinalis*), Kinnikinnik (*Arctostaphylos Uva-ursi*), Sundew (*Drosera rotundifolia*), Poison Ivy (*Rhus Toxicodendron*), Nettle (*Urtica dioica U. gracilis*), Wood Nettle (*Laportea canadensis*), Bull Nettle (*Jatropha stimulosa*).

V

SIMPLE IRRITANTS WHEN TAKEN IN LARGE QUANTITIES

Symptoms. Burning pain in throat and stomach. Vomiting; purging; difficulty in swallowing. Recovery usual.

Treatment. (Immediate:)

EMETIC and STOMACH TUBE. TANNIN.

Opium to relieve pain.

Stimulants to counteract collapse.

(Later:)

Castor Oil.

DEMULCENTS (swallowed and injected).

Plants affording poisons that act as simple irritants when taken in large quantities only: White Mustard (*Brassica alba*), Black Mustard (*B. nigra*), Black Pepper (*Piper nigrum*), Common Ginger (*Zingiber officinalis*), Cayenne Pepper (*Capsicum annuum*).

TABLE OF SYMPTOMS OBSERVED AFTER THE ADMINISTRATION OF POISONS, ADAPTED FROM CATTELL AFTER THE WORK OF KOBERT.

ACUTE SYMPTOMS PRESENT	WE SHOULD THINK OF
1. Death within a few seconds or minutes.	Hydrocyanic acid; potassium cyanid; carbonic acid; carbolic acid.
2. Deep coma.	Alcohol; morphin; opium.
3. Collapse.	Nicotin; colchicin.
4. Feverish rise of temperature.	Cocain; enzymes.
5. Mania; furious delirium; psychic excitement.	Chronic alcoholism; atropin; camphor; physostigmin; veratrin.
6. Mental disturbance of the most diverse kind.	Alcoholism; morphinism; cocainism; pellagra; ergotism.
7. Violent at times, tetanic convulsions.	Strychnin; toxin of tetanus; cytisin; cornutin; picrotoxin; cicutoxin; active principle of digitalis; cocain; santonin; aconitin; gelsemin; filicic acid.
8. General paralysis, for the most part ascending.	Coniin; curarin; colchicin.
9. Dilation of the pupil.	Atropin; hyoscyamin; scopolamin; cocain; ephedrin; aconitin.
10. Contraction of the pupil.	Muscarin; pilocarpin; nicotin; codein; opium; physostigmin.
11. Aneurosis.	Quinin; extract of male fern; belladonna; uraemic poisoning.
12. Diplodia and ptosis.	Sausage (botulism) and fish poisoning.
13. Conjunctivitis.	Irritating vapors; ethereal oil of mustard;

		croton oil vapor; irritating kinds of dust as roots of ipecac, quillaja bark. pepper, formalin, etc.
14.	Moist skin.	Opium; morphin; aconitin; pilocarpin; nicotin; physostigmin; lobelin.
15.	Skin conspicuously dry even in a warmed bed. Mouth and throat parched.	Atropin; belladonna; stramonium; hyoscyamus; hyoscyamin; scopalamin; sausage and fish poisoning.
16.	Urticaria or scarlatiniform erythema.	Atropin; hyoscyamin; antipyrin; quinin; balsam of copaiba; cubebene; morphin; handling of nettles; buckwheat; smartweed.
17.	Eczematous eruptions of skin.	Croton oil; curcas oil; cardol; Rhus Toxicodendron; powdered cinchona bark; carbolic acid; tar.
18.	Diffuse dermatitis with perspiration of the hands.	Anilin colors; aurantia; butter yellow.
19.	Acne pustules.	Powdered ipecac.
20.	Blisters on the skin or the mouth, containing clear serum.	Ranunculus acris; R. sceleratus, etc.
21.	Gangrenous ergotism; carbolism.	Ergot.
22.	Cyanosis.	Antifebrin; exalgin; anilin.
23.	Yellowish-brown discoloration of the conjunctiva, in combination with that of the skin.	Helvellic acid; lupinotoxin (ictrogen).
24.	Discoloration primarily of the tongue and mucous membrane of the mouth.	Carbolic acid.
25.	Salivation.	Pilocarpin; muscarin; arecolin; nicotin; cornutin; physostigmin; cytisin; saponin.
26.	Metallic cough and aphonia.	Atropin; hyoscyamin; scopolamin; sausage poisoning.
27.	Oedema of the glottis.	All caustic poisons.
28.	Oedema of the lungs.	Muscarin; morphin; pilocarpin; nitric acid vapors.
29.	Increased dullness of the liver.	Agaricus bulbosus; poley oil; alcohol.
30.	Diarrhoea with vomiting.	Digitalin; pilocarpin; nicotin; muscarin; colchicin; corrosive poisons; colocynthin; emetin; cephalin; croton oil.
31.	Vomiting without diarrhoca.	Apomorphin; lobelin; cytisin.
32.	Diarrhoea without vomiting.	Jalap; podophyllotoxin; croton oil; calomel.
33.	Pulse continuously and markedly becoming slower.	Opium; morphin; muscarin; arecolin; physostigmin; all narcotics.
34.	Pulse first slower, then irregular, finally accelerated.	Digitalin; hellebore; adonis; coronilla; cheirathin; nervin; scilla; strophanthus; convallaria; pilocarpin; nicotin; scopolamin.
35.	Pulse greatly accelerated.	Belladonna; hyoscyamin; atropin.
36.	Abortion.	Sabina; thuja, rue; mentha; pulegium; ergot; cotton root.

37.	6-12 hour period of good health between the poisoning and the appearance of the symptoms.	Most of the poisonous fungi, but especially Amanita phalloides.

TREATMENT FOR POISONING. (CHIEFLY AFTER KOBERT).

KIND OF POISON	TREATMENT.
Aconite.	Use stomach pump at once; give emetics of sulphate of zinc, or a hypodermic injection of apomorphin; patient should recline; when stomach has been evacuated, give atropin (4 drops U. S. P. solution) hypodermically or by the mouth, or 20 drops of tincture of belladonna; if there is a tendency to heart-syncope, give tincture of digitalis, in ½ drachm doses by mouth, or hypodermically in doses from 10 drops upwards; apply mustard plasters to pericardium; aid vomiting by plenty of water; if necessary apply artificial respiration.
Alcoholism, acute.	Wash out the stomach with a siphon tube; cause vomiting by emetics, cold and hot effusions alternated; strychnin hypodermically.
Aspergillosis	Treatment unsatisfactory; moulds that have entered lungs cannot be destroyed.
Atropin	Wash out the stomach with a solution of tannic acid or cause evacuation with an emetic; hypodermic injection of strychnin to stimulate respiration; administer tea or whiskey.
Carbolic acid.	A liberal dose of whiskey or alcohol as a diluent; use soft stomach tube to wash out the stomach with sodium sulfate; sodium sulfate, raw eggs, milk and saccharate of lime are antidotes.
Cicuta virosa. C. maculata cowbane.	Tannin and narcotics according to symptoms, especially chloral hydrate.
Cicutoxin.	Chloroform, chloral hydate, artificial respiration, stimulants like whiskey.
Cocain.	Evacuate the stomach; then give vegetable astringents, iodin 1 gr. and potassium iodid 10 gr. dissolved in water; digitalis and amyl nitrite are given for syncope; give stimulants like oxygen and whiskey for cyanosis.
Colchicin.	Mucilaginous potions, morphin, warm compresses on the abdomen.
Coniiu.	Wash out the stomach after giving tannic acid or some other astringent; strong coffee and whiskey; strychnin hypodermically; apply artificial respiration if necessary.
Conium.	Tannin, stimulants, especially camphor.
Cytisin.	When spasms occur, give narcotics; artificial respiration in case of paralysis of the organs of respiration.
Delphinosis.	Potassium permanganate is the antidote; in extreme depression of circulation and respiration, atropin sulfate administered hypodermically.
Dermatitis. Rhus.	Antidote is acetate of lead.

Dermatomycosis.	Cleanliness; sanitary surroundings; disinfection; preparation of bichloride of mercury.
Digitalis.	No antidote; treat symptomatically.
Equisetosis.	Administer cathartic; also nerve and heart stimulants to combat symptoms of depression; change of food.
Ergotism.	Administer purgatives and stimulants, especially camphor; tannic acid is chemical antidote and will neutralize unabsorbed portions of poison; chloral is physiological antidote; further treatment symptomatic.
Fagopyrism.	Change of food.
Forage Poisoning.	Change of food.
Formic Acid. (In stinging nettles).	Cooling compresses externally; chalk, soda, or magnesia internally.
Gelsemin.	Wash out the stomach thoroughly; give stimulants and hot applications to the epigastrium and extremities; digitalis to strengthen the heart action and atropin to increase respiration.
Helleborus niger.	Use stimulants, especially camphor.
Hydrocyanic Acid.	Wash stomach with 0.5 per cent potassium permanganate solution or with hydrogen peroxid; the latter may be given hypodermically in small doses but with great caution; artificial respiration.
Lathyrism	Treat paralyzed parts electrically; massage.
Locoism.	In advanced stage of disease, treatment of no avail; in early stage, removal to an uninfected pasture with plenty of good nourishing food besides may benefit.
Lupinosis.	Chiefly preventive; no specific antidote; to prevent further absorption of poison, administer an acid; also give purgative.
Maydism.	Change of diet; transfer to hospital.
Morphin.	Wash out stomach with siphon tube using water containing potassium permanganate in the proportion of 20 gr. of the permanganate to 1 tumbler full of water; or use in the same manner an infusion of tea or tannic acid; emetics like mustard, using one or two teaspoons of each; apomorphin 5-10 minims of a 2 percent solution; hypodermic doses of strychnin 1-20 gr. or sulphate of atropin 1-60 gr.
Muscarin.	Atropin used hypodermically; stimulants like strychnin may be given; wash out stomach.
Nicotin.	If free vomiting has not occurred, wash out stomach with warm water or tea; give stimulants like whiskey or use hypodermic injection of strychnin nitrate 1-25 gr.
Phallin.	No known antidote; undigested material should be removed from stomach and same remedies as those suggested in muscarin poisoning may be administered.
Picrotoxin.	Chloroform; chloral hydrate; artificial respiration.
Pilocarpin.	Evacuate the stomach and wash out with so-

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CHAPTER XII

THE PRODUCTION OF POISON IN PLANTS

THE USE AND ACTION OF POISONS ON PLANTS

An extraordinarily large number of poisonous substances belonging to the alkaloids, glucosides, saponins, and toxins, occur in the vegetable kingdom. In addition there is a larger number of substances not strongly poisonous which are curative in their nature. These substances so widespread in the vegetable kingdom are the products of metabolism and probably in some cases, waste products, although according to Weevers, may act as reserve food substances. Treub [1] states that the hydrocyanic acid in the *Pangium edule* is of importance in the metabolism of the plant. It occurs not only in certain parts of the fibro-vascular bundle but in the cells from the leaf and certain specialized cells of the epidermis, occurring both in a free and in an unstable combination. His conclusion is that the hydrocyanic acid is the first recognizable product of nitrogen assimilation. It is certainly true that in some cases the poisonous products formed in the plant do not undergo any further change. Undoubtedly the substances serve as a protection to the plant and it may be interesting to note that large quantities of these substances may be excreted and occur in an insoluble form in the cell sap and do no injury to the plant. Digitalin, morphin, atropin, eserin, muscarin, and veratrin, seem to exert little or no poisonous action on most plants; while strychnin may act as a strong poison.[2] There are also other alkaloids that when applied to the plant are poisonous to the plants from which they have been obtained. Morphin is said to poison the poppy, and the motile spores are speedily killed by the same substance according to Strasburger. Too little, however, is known about this subject to make any extended remarks. It is interesting, however, to observe in this connection that as in the case of man and other animals, plants can be gradually accustomed to doses which would probably prove fatal in many cases. The Blue Mould (*Penicillium glaucum*) and some species of *Aspergillus* can accommodate themselves to strong solutions of copper and formalin. There are some reasons for believing that the protoplasm of different plants is not a uniform substance but varies, and that one substance may be toxic to the plant while harmless to another and even act as a stimulant.

DISTRIBUTION OF POISONOUS SUBSTANCES IN PLANTS

The seed may contain a toxic substance and upon the germination the poisonous material may occur not only in its juvenile stage but at maturity. In some cases the seed and the juvenile form may be non-poisonous, but as the plant becomes older the poisonous substance is elaborated as in the *latex* of some plants that contain narcotic principles. In some cases the seed is poisonous and the young plants apparently do not contain a toxic material; the

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1 Ann. du Jardin Bot. de Buitenzorg 13. Pharm. Review. 14:278.

2 Pfeffer, Physiology of Plants. English Translation. 2:260; Schwarz, Wirkungen von Alkaloiden auf Pflanzen, Erlanger Dissertation. 1897.

poisonous substance apparently being broken up to serve as a nourishing material for the growth of the plants.

Cornevin [1] in his work upon poisonous plants says: In one group "the poisonous substance does not exist in the plants themselves, but, in some parts or tissue, elements are present which are not really poisonous in themselves but become so when the parts or tissues come in contact with one another. An example is seen in the glucoside *amygdalin* which in contact with *emulsin* forms *hydrocyanic* acid."

The activity of vegetable poisons may depend upon the age of the plant producing them. No definite rule can be established in regard to the age at which a plant produces its poisonous substance. Sometimes a younger plant is more actively poisonous than when older, sometimes, also, the poison is stored in certain tissues.

Poisonous principles are found in various parts of the plant, such as the root, stem, flowers, fruit, leaves, bark, tubers, seed and bulbs.

In many aerial parts of plants the poisonous substances are more ephemeral than they are in organs of the plant that serve as store houses of food.

Cornevin says:

It sometimes happens that the subterranean part only is poisonous as in *Atractylis gummifera*. This is true, also, of the common European Violet. On the other hand, when subterranean organs of certain plants come in contact with the light a poisonous substance may be produced. This is true of the tuber of potato which when green is poisonous. Poisonous substances are elaborated both in evergreen and deciduous leaves; no rule of comparison has, however, been established.

Certain variations of the plant are dependent upon its environment, certain conditions of which play an important part especially in the elaboration of poisonous substances.

These conditions are light, heat, season, climate, soil, culture and fertility.

LIGHT. It is a well knewn fact that light seriously interferes with the growth of parasitic fungi and bacteria so that pathogenic species may become quite harmless when placed in direct sunlight. The poison *atractylin* is formed only in darkness, on the other hand *solanin* is formed only in the light, as in the case of the green potato.

HEAT. Cornevin says:

The action of heat upon plants should be considered with that of light, of the seasonal variation, and of climate, and not as an isolated cause. When it is prolonged it induces dessication of the plant and as a consequence evaporation follows and destruction of the poison when it is volatile. This result is produced in some *Ranunculaceae, Chenopodiaceae*, etc.

Moist heat, that is boiling, conduces to the same result in some poisons. Three-seeded mercury (*Mercurialis annua*) becomes inoffensive when it has been submitted to the action of heat, because of the volatilization of its toxic substance *mercurialin*.

SEASONS. Seasons cause considerable variation in the poisonous material produced in plants, the amount of poison contained often varying with the advance of the season. In aconite the poison, at first contained in the leafy organs becomes concentrated little by little in the seed.

The time of the year may have an important bearing upon the amount of poisonous material found in the plant. According to Prof. Hedrick the Cowbane or Musquash root (*Cicuta vagans*) of the west is much more toxic in the fall, winter, and spring, than in the summer, and this is partially confirmed by an experiment conducted with our common Cowbane (*Cicuta maculata*). It is also well known that the mature bulbs of *Colchicum* contains a much larger amount of the toxic substances than the growing bulbs and that for medicinal

1 Des plantes vénéneuses et des empoisonnements qu'elles deterrminent, 524. Paris, 1893.

purposes the bulbs are usually collected between the decay of the foliage and the production of the flowers.

Opium is obtained from the capsules of the opium plant a few days after the petals have fallen, the seed containing comparatively little of the narcotic substances. It is well-known also that the poisonous principles of the Larkspurs are much more active in the spring than in the summer. Dr. Albert C. Crawford[1] says referring to the *Delphinium camporum*, with which he conducted some experiments and was able to kill several guinea pigs with toxic material obtained from the plant collected on April 26th and May 16th, but failed to get positive results of material collected in June.

There is no question as to the fact that Delphinium when injected subcutaneously will kill, and these experiments also establish the fact that the plant loses much of its toxicity as it approaches the flowering stage. It has been noted that *Delphinium consolida*[2] is also less active when mature.

Just after flowering, the purple larkspur turns yellow and ceases to be attractive, so that there is less danger of poisoning, although Chesnut and Wilcox report death in cattle from eating *Delphinium glaucum* in September. The great danger early in the season seems to arise from the fact that the Delphinium appears early in the spring, and the ground may again be covered with snow, so that it is the only green plant in sight, and therefore when in an especially poisonous stage it is eaten by cattle.

Botanical and other writers have frequently called attention to the fact that the greatest amount of poisoning in the west occurs in early spring. Of course, this may be because there is less green food and live stock may consume more of this plant than at other seasons. However, there seems scarcely any reason to doubt that the plant does contain a larger amount of the acrid toxic substances in the spring than in the summer, as proved by the experiments of Dr. Crawford.

The same author who investigated the Mountain Laurel[3] calls attention to the well-known fact that most of the cases of poisoning from Mountain Laurel occur in the winter. Undoubtedly the animals will eat more of the tough and leathery leaves in the winter because there is very little green for them; but may they not also have a larger amount of the toxic material? The plant is evidently also poisonous in the summer, as indicated by numerous reports of the experiments by Dr. Crawford, who conducted an experiment with material collected in the summer, in May and June, death occuring in a sheep weighing 49 lbs., that had received 90 grams of powdered dried laurel leaves.

The late lamented Dr. Greshoff has called attention to the peculiar distribution of hydrocyanic acid in plants. In referring to a species of *Hydrangea* of the *Saxifragaceae* he states that he sometimes found considerable quantities of hydrocyanic acid in some of the well-known ornamental plants like the *H. hortensia* and sometimes he did not find it. He surmises that the cyanogentic principle disappears from the leaves in the autumn and that the young leaves have much more of the HCN than the older ones. In the case of the Plane Tree (*Platanus*) he found considerable of the same acid in the young leaves but as the leaves grow older the HCN content falls off to small traces. He states further that in the ordinary plane tree of the London Streets there is so much HCN that the amount from each London Plane Tree leaf would be enough to kill a London sparrow.

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1 Bull. Bur. Plant Ind., U. S. Dept. of Agric. 111: Part II; 7.
2 Dammann, C. Gesundheitspflege. 1886:1072.
3 Bull. U. S. Dept. of Ag., Bur. Plant Industry. 121:21.

Miss Alice Henkel in a paper on American Root Drugs [1] notes the importance of collecting medicinal plants and drugs at the right time. The roots of the American Hellebore (*Veratrum viride*) should be collected in the autumn after the leaves are dead. Generally speaking the drugs contain more of the medicinal virtues after the period of cessation of growth of the plant. Dr. C. Mueller [2] calls attention to the fact that *Colchicum* is much better if the rhizome is collected at time of flowering. On this point all authorities do not agree. Cornevin who has made a study from month to month of the migration of poison in plants such as the *Laburnum* found the following conditions: On May 20th, 2 grammes of the dried leaves of *Cytisus Laburnum*, administered to some carnivorous animal, were sufficient to induce vomiting. On June 10 (at which time pods were forming) 4 grammes were required to produce the same result. July 28 (when the pods were fully formed) 12 grammes were required. September 28 (pods were beginning to dry) 20 grammes were required.

The experiments show that at all periods the leaves were poisonous but as the poison became concentrated in the pod the leaves became less toxic.

Similar experiments resulted in demonstrating that while the poison concentrates in the pod it also loses toxicity as the season advances, enormous doses taken from dried seed in October failing to produce death, while a 2-gramme dose from a June pod proved fatal.

CLIMATIC. Latitude has an influence upon the formation of poisons. There are more poisonous plants in tropical regions than in colder regions. Certain plants which are poisonous in temperate regions lose their poisonous properties when taken into colder regions. Examples of this are aconite and cherry laurel.

It is difficult to make a comparison between the total number of poisonous plants in tropical and temperate regions, but it is probable that the warmer regions will show the greater number of poisonous species.

It might be well, however, to note in this connection that the poisonous properties of *Rhododendron Chrysanthemum* and *R. catawbiense* as well as of *Aconitum* are developed in colder regions while such toxic plants as the calabar bean (*Physostigma venenosum*), *Strychnos nux-vomica*, the Upas tree (*Antiaris toxicaria*) and numerous others are inhabitants of warm climates.

It is well known that the sorghum grown in dry climates produces a greater amount of a glucoside which is capable of being converted into hydrocyanic acid than in more moist regions. Some writers even assert that in dry seasons the drought depauperates the plant and that in the nodes there are considerable quantities of potassium nitrate.[3]

Again Dr. Ludwig Bernegau notes that the amount of alkaloid in Cola Nut varies considerably, it depending upon the source of the nut.

With reference to the presence of alkaloids in different kinds of opium, it is known that the Smyrna opium is of superior quality for medicinal purposes. It is said to be superior to the opium obtained from India, which is in part attributed to climatic conditions, and probably in part to the method of collection. According to Blyth, the amount of morphin varies as follows:

Crude morphin (containing about 7-10 of pure morphin). Highest, 12.30; lowest, 6.76; mean, 9.92 per cent, which equals 12.3 per cent of the dried drug.

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1 Bull. U. S. Dept. Agrl., Bur. Plant Industry, 107. Farmers Bull. 188.

2 Pharm. Review, 14:113. Die Natur. 44:550.

3 An English writer, Pease, attributed the death of animals to this substance rather than to the hydrocyanic acid. Pharm. Review, 15:208.

The nicotin of tobacco depends largely on climatic and soil conditions, The Havana, Porto Rico, Sumatra, Connecticut Seed Leaf and Wisconsin have qualities all peculiarly their own.

Blyth records the following percentages of nicotin in various tobaccos as given by Cox.[1]

Variety examined	Nicotin per cent
1. Syrian leaves (a),	.612
2. Syrian leaves (b),	1.093
3. Gold Flake (Virginia),	2.501
10. "Navy-Cut" (Light colored),	3.640
15. "Best Shag" (b),	5.000
17. Algerian tobacco (a),	8.813

According to Professor Garner[2] the nicotin contents vary as follows:

Nicotin soluble in petroleum ether in domestic filler tobacco 2.20 percent; Imported Cuban Santa Clara tobacco 1.33 percent.

That climate plays an important part in connection with chemical products is illustrated in the essential oils. The most important of these such as bergamot, cassia, cinnamon, jasmine, fennel, lavender, orange, rosmary, attar of roses, and many of the others are imported into the United States. These are produced in localities that are more favorable for their production than in many parts of this country. However, some of the essential oils like those from peppermint, sweet birch, and sassafras are produced extensively in the United States. The time of collecting and many other factors as well as the matter of labor enter into the production of these oils. The variation in composition has been indicated by Dr. Edward Kremers and his students in various publications.[3]

It is well known that the locality for attar of roses which supplies most of the commerce of the world is a small district of country on the southern side of the Balkan Mountains. The best localities according to those best informed on the subject are those occupying southern or southeastern slopes. The flowers attain perfection in April or May and are gathered before sunrise. It is concluded from some experiments carried on in some of the northern countries in Europe, that a cool northern climate is not conducive to the production of highly odorous oils.[4]

It is interesting to note that the oil of orange flowers comes chiefly from the southern part of France, and that the flowers of sweet orange afford about one-half the amount of oil that those of the bitter orange do. Lavendar oil, made from *Lavandula vera,* is very variable in quantity, depending upon its source, although not grown to any great extent in England. Flückiger and Hanbury state that the Mitcham oil grown in the Surrey, is of a superior and high quality. The above is used here to illustrate the fact that the chemical products of plants vary depending upon the different climatic conditions.

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1 Pharm. Journ., Jan. 20, 1894. Blyth, Poisons; Their Effects and Detection. Ed. 2.274.
2 Bull. U. S. Dept. Agr., Bur. Plant Ind. 141; Pt. I, 12.
3 Kremers and Schreiner. The Quantitative Estimation of Carvone in Volatile Oils. Pharm. Review. 14:76.
Edward Kremers and Florence M. Gage. Notes on Two Oils Containing Pulegeone. Pharm. Review. 16:412.
Kremers and Sievers. Oils from Milfoil. Pharm. Review. 25:215.
The Volatile Oils, by E. Gildemeister & Fr. Hoffman. English Translation, E. Kremers.
4 Flückiger & Hanbury, 262.

Dr. Rodney H. True in an article in the Cyclopedia of American Agriculture,[1] says:

"The sources of our crude drugs and condiments are very widely separated, depending in large part on climatic conditions. Common drug plants belonging to the temperate zone, such as digitalis, burdock and caraway, are in very large part produced in northern and central Europe, frequently in more or less localized regions. Caraway comes chiefly from Holland, in small quantities from Norway, east Prussia and southern Germany. Fennel is cultivated in Saxony, Galicia, Macedonia and Italy. Digitalis leaves and belladonna reach the market of northern Germany, Austria, Belgium, Holland and England. Peppermint oil is produced chiefly in Japan and the United States. Other plants demanding tropical conditions are obtained from regions in which their culture has been undertaken. Cinchona bark, from which quinine is obtained, came formerly from the slopes of the Andes. Cultivation of this plant in India, Java, and other parts of the Orient has succeeded in so far as to cause the practical disappearance of the wild barks of South America from the market. Ipecacuanha, likewise a native of northern South America, is apparently repeating this history. Black and white pepper are chiefly produced in southeastern Asia, coming on the market through Singapore and Penang. Cloves are in large part supplied by Zanzibar, where the crop constitutes one of the royal monopolies. Some products are derived from still more localized regions, as buchu leaves from the vicinity of Cape Town, South Africa, and aloes from South Africa, the island of Socotra in the Red Sea, and the Barbadoes islands. Some are cultivated, as may be seen in numerous cases cited above, and some are wild products. Camphor until recently has been derived from an essentially wild tree growing in Japan, China and Formosa. The great depletion of the natural forests has led the Japanese government to make extensive plantings. Several African sorts of the red peppers of the market are collected by natives from the wild plants and brought long distances to market."

CULTURE. Cultivation often affects the amount of poison contained in a plant. A wild vetch of Europe (*Laythrus*) is extremely bitter, but through cultivation the poisonous material has been largely eliminated and the vetch has become a useful cultivated forage plant. It is stated also that the aconite (*Aconitum Napellus*) when cultivated loses some of its toxic properties and that this loss of toxic action occurs in a few generations. Prof. S. M. Tracy informs me that the cultivated forms of the Cassava are not injurious to stock but the wild form, as is well known, contains toxic materials. The wild forms of the lima bean (*Phaseolus lunatus*) contains much more HCN than the cultivated forms.

Dr. Rodney H. True in speaking of the physiological action of the betel nut states that its physiological action[2] depends on several factors. The green nuts produce temporary dizziness. The poisonous variety according to Indian authorities is one that is reverted to its former wild condition, while the common or ordinary betel nut which has been cultivated for hundreds of years is a mild narcotic stimulant bringing about a feeling of general comfort, good humor and exhilaration.

It is also well known that when certain toxic bacteria as *Streptococcus pyogenes* are cultivated they lose some of their pathogenic properties.

We must not, however, conclude that because a plant is cultivated it loses its poisonous properties, because there are certain cultivated ornamental plants that are as poisonous in their cultivaed form as when grown wild. For example the tobacco which has been cultivated for several hundred years contains as much *nicotin* in its cultivated as in its primitive form.

SOIL. It is believed also that the soil plays an important part in connection with the amount of poison produced in the plant. There can be no question that the soil plays an influence upon the quality of the fruit as well as upon

1 Cyclopedia of American Agriculture. 2:458.
2 Pharm. Review, 14:130.

the quality of the grain. Why should it not also influence the poisonous constituents of the plant? Cornevin mentions that soil affects the color of *Digitalis*, it being much paler when grown in calcareous than when grown in granitic soil.

RELATED SPECIES AND TOXIC SUBSTANCES. In some cases the same toxic substance is widely distributed in different families of plants. On the other hand closely related plants frequently have entirely different amounts of toxic substances. The bitter and sweet almond illustrate this in a very marked degree, the bitter almond having considerable amounts of the glucoside amygdalin, while the sweet almond is entirely harmless.

Frank Rabak[1] has made an investigation of the amount of the chemical substances found in the kernels of the fresh apricot and plum, in which it appears that the amount of hydrocyanic acid by per cent contained in these plants is as follows:-

	Per Cent
Peach	2.20
Apricot	2.40
Apricot	2.05
Prune	1.75
Bitter Almond	4.80

According to Dohme and Engelhardt[2] the thin green or young bark of *Prunus serotina* is richer in hydrocyanic acid, than the dark brown or older bark.

It may be interesting to note that certain species of Cacti are used for stock food,[3] and others contain powerful drugs. For instance, the Mescal Bean, which is derived from several species of *Anhalonium* which contain peliotin, is a narcotic of considerable potency.[4]

According to Dr. Peinemann[5] the alkaloidal-content of *Datura* varies as follows; the seeds, 0.541 percent.; root, 0.315; leaves, 0.41 percent. of *atropin*, The *Datura alba* is richer in alkaloids than the native species, *D. Stramonium*.

Dunstan and Henry[6] are authorities for the statement that two forms of Mandrake, the American (*Podophyllum peltatum*) and the Indian (*P. emodi*), produce the same principle podophyllotoxin $C_{15}H_{14}O_6$ a strong purgative and also the so-called podophyllin which consists of a resin. The podophyllotoxin occurs in the Indian plant from 9-12% and in the American plant from 4-5%.

DISTRIBUTION OF CHEMICAL SUBSTANCES. The same chemical substance is frequently found in plants that are not closely related. For instance, Edward Kremers and many other pharmaceutical chemists have found the essential oils in widely separated families, and Greshoff states that a leaf of Five-finger (*Potentilla davurica* of China) which is closely related to our shrubby Cinquefoil (*P. fruticosa*) produces a powerful odor of roses. Geranoil occurs, for instance, in the young foliage of willow and many other plants.

The substance berberin is found in a large number of plants like the Barberry, the Mandrake, Twin Leaf (*Jeffersonia diphylla*), Meadow Rue (*Thalictrum flavum*), *Toddalea aculeata, Hydrastis canadensis, Argemone mexicana*, etc.

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1 Bull. U. S. Dept. Agr., Bur. Plant Industry. 133.
2 Pharm. Review. 14:13.
3 Griffiths and R. F. Hare, The Tuna as Food for Man. Bull. U. S. Dept. Agrl. Bur. Plant Ind. 116.
4 Pharm. Review. 14:153.
5 Pharm. Review. 14:233.
6 Proc. Chem. Soc. 189:42-44.

ceae), contains HCN, and Greshoff says must henceforth be counted among the poisonous plants. Of the Grass Family (*Gramineae*) quite a number of species such as the *Stipa lessingiana,* are now known to contain HCN. Some of them like our *Stipa robusta,* have been known to be toxic for a long time, but such genera as the Quaking Grass, Briza (*Catabrosa*), Wild rye (*Elymus*), Manna Grass (*Glyceria*), Salt Grass *Holcus lanatus* all contain HCN.

CUMARIN: The substance *cumarin* also occurs in many different plants which we may list as follows:

Talauma (Magnoliaceae); *Phoenix* (Palmae); *Dipteryx, Toluifera, Melilotus* (Leguminosae); *Prunus* (Rosaceae); *Ceratopetalum* (Saxifragaceae); *Asperula, Basanacantha, Borreria, Diodia, Galium, Mitracarpum, Spermacoce* (Rubiaceae); *Ageratum, Chrysanthemum, Eupatorium, Humea, Trilisa, Liatris* (Compositae); *Alyxia* (Apocynaceae); *Hemidesmus* (Asclepiadaceae); *Rhinacanthus* (Acanthaceae), *Aceras, Angraecum, Orchis* (Orchidaceae); *Hierochloe, Anthoxanthum* (Gramineae); *Adiantum, Cheilanthes, Polypodium* (Filices); *Lactarius, Russula* (Fungi).

CYTISIN. Greshoff reports cytisin as occuring in the following plants of the Pulse family: *Cytisus Adami; C. alpinus; C. Alschingeri; C. biflorus; C. hirsutus; C. Laburnum,* seed 1.8 per cent; *C. nigricans; C. polytrichus; C. proliferus; C. Weldeni; C. Attleanus; C. candicans; C. formosissimus; C. monspessulanus; C. Ruthenicus; C. scoparius; Ulex europaeus,* seed 1 per cent; *U. Jussiae; Genista ephedroides; G. monosperma,* seed 1.9 per cent; *G. florida; G. germanica; G. spicata; G. tinctoria; G. ramosissima; Sophora secundiflora,* seed 3.5 per cent; *S. tomentosa; S. flavescens; S. sericea; Baptisia perfoliata; B. tinctoria; B. alba; B. australis; B. leucantha; B. versicolor; Euchresta Horsfieldii, Anagyris foetida,* Rauwerda reported it in 28 species of *Lotus, L. suaveolens; Thermopsis Caroliniana;* and *Th. montana.*

In some instances apparently the same alkaloidal substance was first reported in some plants, but later investigations have shown that these substances are not identical. For instance Schlotterbeck who investigated the alkaloids found in the poppy family, especially *Argemonia Mexicana,* reported that this plant does not contain morphin but protopin and berberin.

CHAPTER XIII

ALGAE IN WATER SUPPLIES

Algae have long been known to be injurious to water supplies and numerous papers in the United States and Europe have discussed some of the problems arising therefrom. Algae are generally small, microscopic plants found in water and belong to the great class known as Thallophyta. These frequently cause the pollution of water supplies and are therefore of first importance in this connection. Some algae contain a green coloring matter called chlorophyll and hence they are able to make their own food out of water and carbon dioxid. Many of the algae, however, are not green, some being blue, others red, and still others brown. The Sea Mosses are algae also, and occasionally attain great size and length. Some algae are supposed to be injurious to stock, Mr. George Francis in Australia having attributed a disease of horses, sheep, dogs, and pigs to some blue-green algae. The organism causing the trouble in this case was referred to *Nodularia spumigera*, which floats on the water and, being wafted to the lea shores, forms scums from two to six inches thick. He says that, when animals drink the water, it acts as a rapid poison and causes death. It first produces stupor and unconscioueness, the animal falling and remaining quiet, as if asleep, unless touched, when convulsions come on, with head and neck drawn back by rigid spasms which subside before death. This, in sheep, takes place in from 1-6, or 8 hours; in horses, in 8-24 hours; in dogs, in 4-5 hours; and in pigs, in 3-4 hours. A post mortem was made on a sheep that had 30 ounces of fresh scum administered by mouth, death occurring in 15 hours. The post mortem, 6 hours later, showed that the stomach had none of the green scum left, all having been absorbed; the abdominal cavity contained 2 pints of yellowish material; the heart was flaccid with effusion of serum around it; lungs, liver, kidneys, and brain normal; dura mater enlarged; blood of ventricles and arteries black. It was thought that poisoning was due to the decomposition of the algae which smelled like putrid urine, previous to this it had an odor like butyric acid. Account was not taken of the products of decomposition by bacteria. The trouble may have been caused by the poisonous products of bacteria rather than these algae. This seems not have occurred to Dr. Francis.

Many of the algae, especially the following, *Anabaena, Clathrocystis* and bacteria-like *Beggiatoa*, produce what is known as the working of the lakes and pig pen odors. Dr. Farlow has described these from Massachusetts and Dr. Trelease has described them for the lakes about Madison, Wisconsin. The greenish-yellow scum occurs on the surface of the lakes, especially noticeable during hot weather of the summer. Dr. Trelease says:

> When a lot of it is present, it appears as fine granules suspended in the water, scarcely visible to the naked eye except as they reflect the light, when they call to mind the dancing motes in a beam of sunlight. Under the influence of a gentle, but continuous breeze, these particles are collected into fleshy masses and are driven ashore, so that they accumulate along the margins of the lake, forming a slimy scum, which quickly putrefies, giving off a very disagreeable odor. During this change, its color changes to a decided

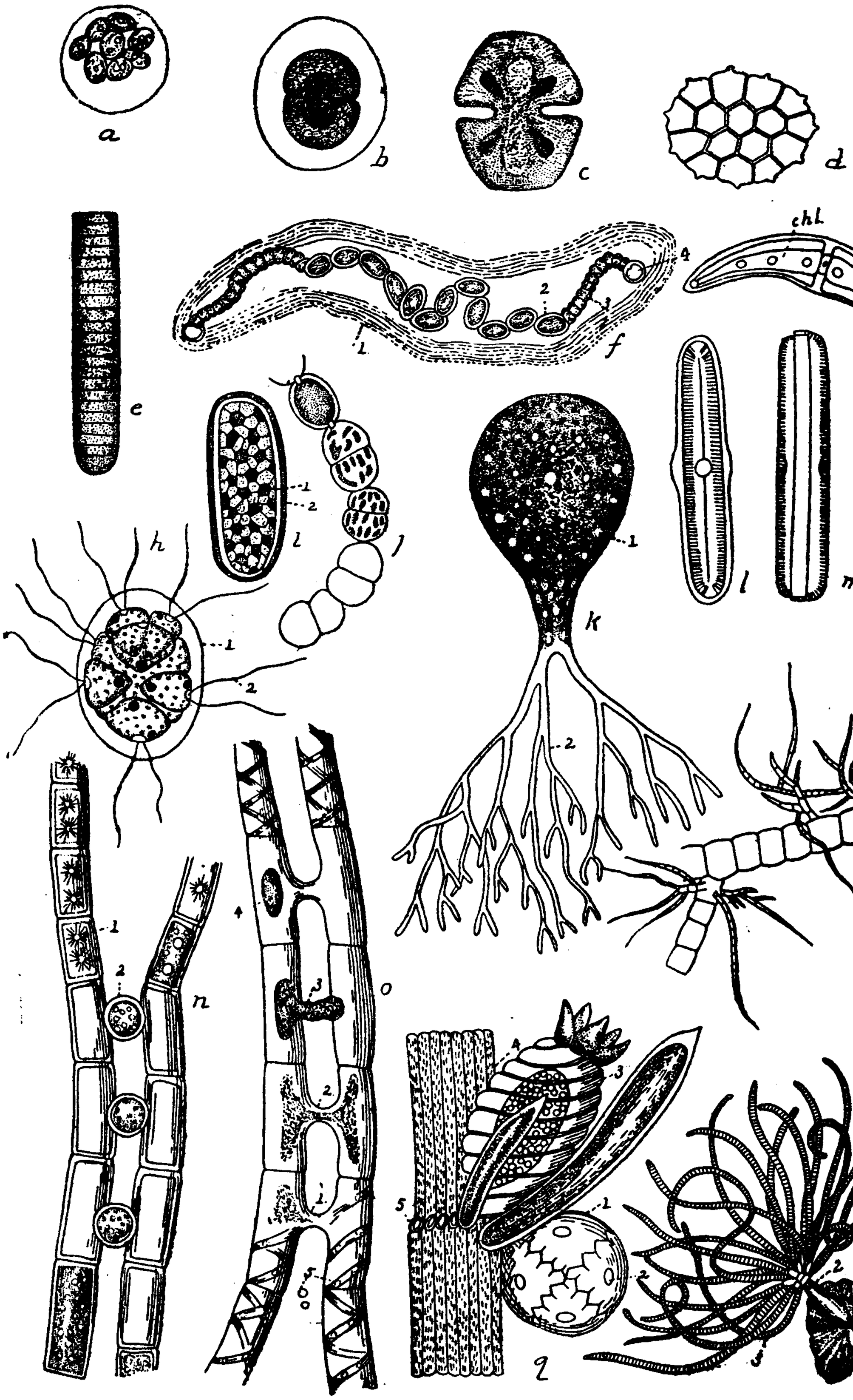

Fig. 19. Algae found in water supplies. *a.* Protococcus. *b, c. Cosmarium*, one of the desmids. *d. Pediastrum. e. Oscillatoria. f. Nostoc paludosum.* g. *Closterium, chr*—chromatophore. *h. Pandorina*; 1, gelatinous envelope; 2, cilia. *i, j.* (*Anabaena flos-aquae*). *k. Botrydium granulatum*; 2, *rhizoids. l* and *m.* Diatoms. *n. Zygnema*; 1, chromatophore; 2, zygospores. *o. Spirogyra*; 1, 2, 3, 4, different stages in the formation of the zygospores. *p. Draparnaldia*; 2, thallus. *q. Stoneworts* (Chara); 1, antheridium; 3, archegonium; 5, nodal cells. *r.* Portion of antheridium with sperm cells.

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The majority of organisms which have been recorded are of microscopic size. At first sight it might appear that these organisms would be insignificent in proportion to their mass, and that more attention should have been devoted to the larger plants and animals. Very few cases have been observed, however, in which really serious trouble in water supplies can be referred directly to the growth of large plants or animals; whereas, in many instances, waters with very objectionable qualities contain nothing to which these qualities can be attributed except microscopic organisms. Further, although the organisms here dealt with are of such small dimensions, the immense numbers in which they occur more than counterbalance their small size; and I am therefore, of the opinion that, of those organisms which cause objectionable qualities in water, the microscopic ones are the more important.

HOW GROWTH OF ALGAE IN WATER MAY BE PREVENTED

The growth of algae may be prevented by covering the reservoir; since light is essential for their growth, they will not develop in a closed reservoir, and we hear of little complaint under such conditions. Large reservoirs for public water supplies are not generally covered. Often reservoirs are much troubled by algal contamination; in some cases it becomes necessary to remove these by a laborious method. The removal of organic matter by keeping the source of the water supply in as pure condition as possible will no doubt do something toward keeping algae out, but nearly all water contains sufficient organic matter for the growth of algae, especially water coming from water sheds.

THE USE OF ALGICIDES

Within recent years much work has been done in preventing the growth of algae and bacteria by copper sulphate solutions. Moore and Kellerman in a recent paper arrive at the following conclusions:

The disagreeable odors and tastes so often present in drinking water are due almost exclusively to algae, although the economic importance of studying these plants has not been recognized until recent years. These algal forms are widely distributed, and reservoirs in many states have been rendered unfit for use by their presence. It has been found that copper sulphate in a dilution so great as to be colorless, tasteless, and harmless to man is sufficiently toxic to the algae to detroy or prevent their appearance. At ordinary temperatures one part of copper sulphate to 100,000 parts of water destroys typhoid and cholera germs in from three to four hours. The ease with which the sulphate can then be eliminated from the water seems to offer a practical method of sterilizing large bodies of water when this becomes necessary. The cost of material for exterminating algae will not exceed fifty to sixty cents per million gallons and will usually be less. The destruction of pathogenic bacteria requires an expenditure of from $5.00 to $8.00 per million gallons, not including the cost of labor.

It has been found that Spirogyra will die in water containing one part of copper to one billion parts of water. Some of the algae like Anabaena are destroyed in dilutions from one to five million, although Moore and Kellerman found that one species of Spirogyra requires a greater strength of solution. There is a wide-spread opinion that metallic copper and copper salts are injurious, which is certainly true when the same are taken in larger quantities. According to Tschirsch, .05 to .2 of copper sulphate causes vomiting and diarrhoea. In the paper cited, Moore and Kellerman write as follows:

It is evident that there is still a considerable difference of opinion among eminent authorities as to the exact amount of copper which may be injurious, but as a very conservative limit we may accept 0.02 gram as the amount that may with safety be absorbed daily. According to Merck's Index, the National Dispensatory, and the United States Dispensatory, the dose of copper sulphate for tonic and astringent purposes is one-fourth grain, or 0.016 gram; as an emetic, a dose of five grains, or 0.33 gram. Thus it is seen that even if the maximum concentration of copper sulphate necessary to destroy algae in reservoirs were maintained indefinitely, the absorption from daily use would be very far below an amount that could produce the least unpleasant effect. Taking a dilution of one

to one million, which in all cases would be sufficient to prevent the growth of a polluting algal form, it would be necessary to drink something over twenty quarts of water a day before an amount which is universally recognized as harmless would be introduced into the system, while more than fifty quarts would have to be consumed before there would be danger of producing an unpleasant or undesirable effect. As will be seen from the preceding tables the use of copper sulphate at this maximum strength of one to one million would need to be resorted to only in extreme cases, and for a very short length of time, for, the reservoir once entirely free from the organisms, a very much weaker solution would be sufficient should any further application be necessary.

The Report of the Massachusetts State Board of Health for 1905 stated as follows:

The objectionable tastes and odors of pond and reservoir waters, which are often attributed to decaying fish and other causes, are, in practically all cases which have been brought to the attention of the Board, caused by the presence of organisms, in some cases of kinds which attach themselves to the sides and bottom of the reservoir, but in the majority of cases of those kinds which live in suspension in the water. Early in 1903, Dr. George T. Moore formerly Algologist of the United States Department of Agriculture, brought to the attention of the Board the results of investigations which he had made, indicating that the microscopic organisms which are the chief cause of objectionable tastes and odors in the waters of ponds and reservoirs could be destroyed by applying sulphate of copper or blue vitrol to the water in very small quantities; and information was also submitted tending to show that bacteria were also destroyed in water brought in contact with metallic copper.

The results of some of the experiments and investigations so far as obtained tend to support the conclusions which had been reached when the matter was first brought to the attention of the Board as to the practicability of the copper treatment for the removal of growths of organisms and bacteria, but the results of other experiments conflict with some of these conclusions. Further study and experiment are necessary before the probable results of the use of copper in preventing objectionable conditions resulting from growths of organisms, or the probable effect of the use of this substance in public water supplies upon the public health can be conclusively determined.

Dr. Moore states that:

Spirogyra is often the cause of considerable trouble in a mechanical way, and on account of its method of forming resting spores is usually able to withstand the most unfavorable conditions to which it may be subjected in a pond or reservoir. In at least one instance this alga has been the cause of the loss of thousands of dollars by the damage it produced in smothering out the young water-cress plants in the artificial beds constructed for the winter propagation of this vegetable. When the cress is cut for market, it necessarily leaves the plants in a weakened condition, and if the Spirogyra gets a start it will form a thick, heavy mat over the water, which is sufficient to prevent the growth, if not entirely to kill, the cress plants.

CHAPTER XIV

A CATALOGUE OF THE MORE IMPORTANT POISONOUS PLANTS OF THE UNITED STATES AND CANADA

For the purpose of facilitating a study of the poisonous plants of this country a partial catalogue is given here. It is based in part on a catalogue of the poisonous plants of Iowa,* issued by the writer and Estelle D. Fogel (Buchanan). To further facilitate the study of the poisonous plants the following papers on the subject should be mentioned:

Chesnut,[1] Chesnut & Wilcox,[2] Wilcox,[3] Nelson,[4] O'Gara,[5] Kennedy,[6] Peters, Slade & Avery,[7] Glover,[8] Crawford,[9] Heald & Peters,[10] White,[11] Johnson,[12] Bessey,[13] Rusby,[14] Guttenberg,[15], Pammel,[16] Millspaugh,[17] Coville,[18] Peck,[19] J. U. & C. G. Lloyd,[20] Stebler & Schroeter,[21] Schaffner,[22] R. Schimpfky,[23] Winslow,[24] Farlow,[25] Maiden,[26] Atkinson,[27] Greshoff,[28] Wilson,[29] Cornevin,[30] Halsted,[31] Kobert,[32] Crawford,[33] and Marsh.[34]

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*Proceedings of the Iowa Academy of Science. 14:147-172. Contributions, Department of Botany, Iowa State College. 37:147-176.

1 Principal Poisonous Plants of the United States, Div. Bot. U. S. Dept. Agr. 20-60, f34. Thirty Poisonous Plants of the United State, U. S. Dept. Agr. Farmers Bull. 86:32, f24. Rep. Bu. An. Ind. 15:387-420, f69.

2 The Stock Poisoning plants in Montana, Bull. Div. Bot. U. S. Dept. Agr. 26:150, pl. 36.

3 Rep. Bu. An. Ind. 17:91-121, p. 32.

4 Feeding Wild Plants to Sheep, Rep. Bu. An. Ind. 15:421-425.

5 Rep. Nebr. Agr. Expt. Sta. 16:14-84, f. 13.

6 Bull. Nev. Agr. Expt. Sta. 51-57, pl. 26.

7 Poisoning of Cattle by Common Sorghum and Kafir Corn, Neb. Agr. Expt. Sta. 77:16.

8 Bull. Col. Agr. Expt. Sta. 113:24, pl. 8.

9 The Poisonous Action of Johnson Grass, Bull. Bur. Pl. Ind. 90, part 4.

10 Ergot and Ergotism, Press Bull. Nebr. Agr. Expt. Sta. 24, f. 3.

11 Dermatitis Venenata, An Account of the Action of External Irritants upon the Skin, 216:1887.

12 A Manual of the Medical Botany of N. Amer. p. 292, pl. 9, f. 160.

13 Bull. Dept. Bot. 1884. Proc. Soc. Prom. Agr. Sci. 23:35-41.

14 Poisonous Plants of the Vicinity of New York City, 19.

15 Poisonous Plants which Grow in and around Erie, 21:25.

16 Bull. Ia. Agr. Expt. Sta. 70:423-448. Poisoning from Cowbane, Bull. Ia. Agr. Expt. Sta. 28:215-225, f. 5.

17 Medicinal Plants, 1:99, pl. 99. 2:100-180, pl. 180.

18 Poisonous Mushrooms, Cir. Div. Bot. 13:24.

19 Edible and Poisonous Fungi of New York, Ann. Rep. State Bot. of State of N. Y. 1896: 105-248, pl. 43. Rep. State N. Y. Mus. Nat. Hist. 48.

20 Drugs and Medicines of N. Amer., Ranunculaceae 1:304, pl. 24, f. 105.

21 Beitrage zur Kenntniss der Matten und Weiden der Schweiz. Landw. Jahrb. d. Schweiz 5:141-225, pl. 20, f. 4.

22 Poisonous and other Injurious Plants of Ohio, Ohio Naturalist 4:16, 32, 69.

23 Deutschlands Wichtigste Giftgewächse in Wort und Bild nebst eine Abhandlung ueber Pflanzengifte, pl. 27.

24 Veterinary Materia Medica and Therapeutics, 775.

25 Some Edible and Poisonous Fungi. Bull. U. S. Dept. Agr. Div. of Veg. Path. and Physiology. 15:453-470.

26 Plants Reported to be Poisonous to Stock in Australia. N. S. W. Dept. of Agr. Misc. Pub. 477.

27 Mushrooms; Edible, Poisonous, etc. 322. Ithaca, N. Y. (2nd Ed.).

28 Phytochemical Investigations at Kew. No. 10, 1909. Monog. de plantis venenatis et sapientibus, etc. Batavia, 1900.

29 Oleander Poisoning of Live Stock. Bull. Ariz. Agr. Expt. Sta. 59:381-397.

30 Des plantes vénéneuses et des empoisonnements qu'elles determinent. 524. Paris, 1893.

31 The Poisonous Plants of New Jersey. Bull. N. J. Agr. Expt. Sta. 135:28.

32 Lehrbuch der Intoxikationem. Stuttgart, 1902. Practical Toxicology for Physicians and Students. Trans. and ed. by L. H. Friedburg. 201. N. Y., 1897.

33 Laboratory work on loco-weed Investigation. Bull. U. S. Dept. Agr. Bur. Pl. Ind. 121:39-48.

34 The Loco-weed Disease. Far. Bull. U. S. Dept. Agr. 380:16.

produce very disagreeable odors when decomposition occurs, and this plant is no exception to the rule. A few of these algae may be mentioned.

Anabaena flos-aquae Breb.

Anabaena stagnalis, Kg. Both of these are found floating on the water. In their decomposition they produce pig-pen odors.

EUPHYCEAE—Algae

CHLOROPHYCEAE.

Volvocaceae.

Pandorina. Common in stagnant pools, especially in barnyards. The water is repulsive. Cattle will not drink it unless driven to do so. May be injurious. Volvox may be placed in the same category.

EUMYCETES---Fungi

PHYCOMYCETES. BLACK MOULDS AND DOWNY MILDEWS.

Mucoraceae.

The species of this family are common; among them are: Mucor mucedo L., found on horse manure.

Mucor corymbifer F. Cohn, and Mucor rhizopodiformis F. Cohn. Both species are pathogenic.

Mucor stolonifer Ehrb.

Common in the United States. Not pathogenic. Mouldy vegetables, etc.

Mucor racemosus A. Fres.

Is widely distributed in North America. Mouldy vegetables, etc.

BASIDIOMYCETES.

Ustilaginaceae.

Ustilago Zeae (Beckm.) Ung. Corn smut is supposed to be poisonous to cattle, but the evidence is not very conclusive.

Ustilago avenae (Pers.) Jens. The common loose smut of oats is supposed to be injurious in large quantities, the same may be said of other smuts occurring upon cereals. Among these are barley smut *Ustilago hordei, U. nuda, U. tritici.*

Ustilago neglecta Niessl.

Pigeon Grass smut is thought by many farmers in Iowa to cause poisoning, especially abortion. Prof. Power, formerly of the University of Wisconsin, found present in this smut a small quantity of ergotin.

Ustilago utriculosa Tul. Smartweed Smut.

This common smut is said to produce irritation and frequently is the cause of trouble at husking time. This fungus is widely distributed in North America.

Tilletiaceae.

Tilletia foetens (B. & C.) Trel.

Stinking smut of wheat is not common in this state, but when it occurs in flour it causes bad odors. The spores give to the flour a dark color and make it unsalable.

Melampsoraceae.

Coleosporium solidaginis (Schw.) Thum.

Parasitic on golden rod and some other plants of the order. Common in

the state. A number of horses in Black River Falls, Wisconsin, a few years ago, became diseased, it is thought, by means of this rust. It may produce a form of Mycotic stomatitis.

Pucciniaceae.

Puccinia graminis Pers.

Wheat and oat rust, especially the uredo stage, produces inflammation of the mucus membrane of the mouth and nose. The dust coming from the straw when the grain is threshed often causes serious disturbances. Other rusts might be mentioned in this connection, like *Puccinia coronata,* Cda., the uredospores of which have an effect similar to that of the common grass rust. The above rusts are widely distributed in North America. *Uromyces trifolii,* Clover Rust, is widely distributed on red clover and, according to several authorities, is responsible for mycotic stomatitis.

Polyporaceae.

Boletus felleus Bull.

It has a bitter taste and is poisonous.

Agaricaceae.

Lepiota morgani Pk.

This fungus is very common in the fall in meadows, pastures, and lawns. The cap is from 5 to 11 inches across. The gills or radiating plates beneath the cap are brown when mature. The lower part of the stipe is somewhat enlarged but no cup occurs. This when eaten by some people is known to produce poisoning.

Amanita muscaria L. Fly agaric.

Used to poison flies in Asia. Poisonous to persons.

Amanita phalloides Fr. Amanita.

This species is very poisonous and no one should eat so-called mushrooms unless he is familiar with them.

Amanita spreta Pk.

This species is said to be poisonous in Eastern North America.

Amanita rubescens Fr.

This species is said to be edible, but authorities recommend that it should be used with great care. The same is true of *A. solitaria.* Both species are found in E. North America.

Amanita verna Bull.

This species is closely related to A. phalloides, but it is pure white in color; it is deadly poisonous. *A. virosa* Fr. belongs to the same category. *A. frostiana Peck* is also poisonous.

Clitocybe illudens Schw. Jack-my-Lantern.

This species is common in Eastern North America and is phosphorescent. While it is not dangerously poisonous, it is not edible.

Phallaceae.

Ithyphallus impudicus (L.) Fries.

The common stinkhorn is probably poisonous. Its disagreeable odor, however, would seem to render it distasteful to animals.

Lycoperdaceae.

Lycoperdon Bovista L.

The giant puffball is edible in fresh condition, when the flesh is white, but

in the mature form in considered poisonous; the same may be said of other puffballs.

ASCOMYCETES

Aspergillaceae.

Aspergillus glaucus Link.

This fungus is supposed to produce staggers. Frequently found in mouldy hay and gives rise to digestive disorders. Mycotic stomatitis.

Aspergillus niger van Tieghem.

This mould also occurs in mouldy hay and other mouldy substances and like the preceding species is injurious.

Aspergillus fumigatus Fr.

Commonly found on decaying substances, especially mouldy hay. It is pathogenic. Found in the ear of man as a parasite.

Aspergillus flavus Link.

Also pathogenic.

Hypocreaceae.

Claviceps purpurea Fr.

Found on many different grasses, especially wild rye, cultivated rye, timothy and quack grass. Produces a disease known as ergotism. The fungus is very poisonous, causes dry gangrene and abortion.

FUNGI IMPERFECTI

Many of the imperfect fungi undoubtedly produce Mycotic stomatitis.

Oidium albicans Robin.

Commonly found in the mouth of sucking animals of different kinds, especially calves. Blastomycosis produced by *Oidium* or *Saccharomyces.*

Fusarium equinum Norg. Umatilla horse mange.

Dr. Mohler and others have demonstrated that the fungus is parasitic and produces this mange.

Fusarium roseum is commonly found on corn and other mouldy grain. It is probably concerned in forage poisoning. Dr. Burrill and Prof. Barrett report several forms of Fusarium on corn in Illinois.

Diplodia zeae Lev.

This disease is widely distributed on the sheaths and blades of corn in the United States. It is reported as common in Illinois by Burrill and Barrett and in Nebraska by Heald and Wilcox. It is also common in the state of Iowa. Dr. Erwin F. Smith suggests some connection between pellagra and this mould.

Sporotrichum Furfur Rob. Favus.

Sporotrichum tonsurans. Barber's Itch.

Polythrincium trifolii Kunze. *Helminthosporium gramineum* and other fungi may be responsible for mycotic stomatitis. The rape fungus, *Polydesmus exitiosus* Kühn, has long been associated with this disease.

EMBRYOPHYTA

PTERIDOPHYTA

Polypodiaceae. Ferns.

Pteris aquilina L.

The common brake is found in eastern states and across the continent;

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Probably poisonous, like the preceding.
Juniperus sabina L. Swedish juniper.
Poisonous, like the preceding species. Cultivated.
Juniperus horizontalis Moench. American Savin.
Poisonous, Canada to Minn., in swamps.
Juniperus occidentalis Hook and vars. Red cedars.
Common from the western slope of the Rocky Mountains westward.
Sequoia sempervirens Endl.
Redwood leaves are said to be poisonous.

ANGIOSPERMAE

Typhaceae.

Typha latifolia L. Common Cat-tail.
Said to be poisonous to cattle.

MONOCOTYLEDONEAE.

Alismaceae.

Alisma Plantago-aquatica L. Water plantain.
Recorded as being poisonous. Swamps.
Sagittaria latifolia Willd. Large arrow head.
The milky juice is somewhat bitter. The plant is edible, when cooked. The root stocks of several species of the genus are eaten by the Indians and in China. Swamps.

Gramineae.

Zea Mays Gärtn. Numerous cases of poisoning have been recorded from the consuming of corn. This, however, is probably due to the production of a poison from the growth of mould or bacteria. The feeding of large amounts of dry fodder produces impaction of the stomach.
Andropogon sorghum Brot. Sorghum.
Second growth sorghum has frequently been reported as poisonous to live stock; this is due to the formation of HCN in the wilted leaves. *Calamagrostis, Milium* and some other grasses contain the same substance.
Setaria italica, Kunth. Millet.
Both the German Millet and the Hungarian Grass are poisonous to horses, acting especially on the kidneys. The poisoning is probably due to a glucoside.
Stipa spartea Trin. Needle Grass, or Porcupine Grass.
The sharp pointed callus often inflicts serious injuries; the fruits work their way under the cuticle into the flesh of the animals, and in some instances they have even penetrated the intestines.
Stipa comata Trin. Western needle grass.
Somewhat injurious, like the preceding. West of Missouri River.
Stipa robusta Vasey. Sleepy grass.
This grass has been suspected of producing stupor in horses. Rocky Mountains. Some species produce HCN.
Avena sativa L. Common oats.
The chaff of this grass sometimes produces balls in the stomachs of horses, known as phytobezoars.
Avena fatua L. Wild oats.
Common in a few counties in northern Iowa. Common in N. West., Cali-

fornia, Rocky Mts. Sometimes causes mechanical injuries on account of the pointed callus of the fruit.

Lolium temulentum L. Darnel.

The grain of this grass is injurious when ground in with flour. It produces stupor and symptoms resembling drunkenness. The poisoning is due to the fungus found in the seed. Principle loliin, a glucoside.

Hordeum vulgare L.

The chaff and awns of barley are often injurious, especially when coming in contact with the mucous membrane, not only in man, but in lower animals.

Hordeum jubatum, L. Squirrel-tail. Wild Barley.

Common throughout the west. This grass produces mechanical injuries in animals that feed on hay containing it, the awns working their way in between the teeth and maxillae, where they cause inflammation and the formation of pus. *G. gussonianum, H. caespitosum, H. secalinum*, etc., produce similar injuries.

Sitanion Elymoides Raf. Squirrel tail.

Common Rocky Mts. to Pacific Coast. Troublesome like Wild Barley.

Agropyron repens Beauv. Quack grass.

Widely distributed in northern Iowa. Produces a slight irritation of the mucus membrane. Contains triticin.

Araceae.

Arisaema triphyllum (L.) Schott. Jack-in-the-pulpit.

It is widely distributed in the north. The corm is known to be very acrid and poisonous, but when boiled or roasted the poisonous substance is expelled.

Arisaema Dracontium (L) Schott. Dragon's head.

Widely distributed, especially in eastern and central Iowa and northern states. The corm is somewhat acrid and is used to destroy insects; it is said to be a good vermifuge. The action of the plant in fresh condition is somewhat similar to that of ammonia.

Symplocarpus foetidus (L.) Raf. Skunk Cabbage.

Local only in a very few places. Said to be poisonous, causes vomiting, and temporary blindness. The juice is acrid and the plant has a very disagreeable odor.

Liliaceae.

Zygadenus elegans Ph. Swamp Camas.

Common in northern Iowa, Minnesota and westward. In the western states it is regarded as poisonous to cattle and sheep, occasionally causing death. It is not as poisonous as some other species of the genus.

Zygadenus venenosus Wats. Death Camas. Common in the Rocky Mountains and westward. The bulb is especially poisonous.

Melanthium virginicum L. Bunch-flower.

Common on low grounds in eastern Iowa and South and East. The root stocks are regarded as poisonous, but reports have come to us of the poisonous effect on horses, of the leaves and stems, when occurring in hay.

Veratrum woodii Robb. False Hellebore.

Southeastern Iowa to Mo. Poisonous like the eastern white Hellebore and the western California Hellebore. Probably contains jervin, cevadina and cevadin.

Veratrum viride Ait. Common swamp hellebore.

Eastern North America; swampy places. Seeds are poisonous, also herbage under some conditions.

Veratrum californicum Durand. California hellebore.

Rocky Mountains to Pacific Coast.

Amianthium Muscaetoxicum (Walt.)Gray. Fly poison. Crow poison.

Common along the Atlantic Coast and Long Island southward.

Erythronium purpurascens S. Wat. Dog-tooth Violet.

California. Contains saponin. Said to be poisonous.

Leucocrinum montanum Nutt. Leucocrinum.

Northwestern United States. Thought to be poisonous to sheep.

Nothoscordum bivalve (L.) Britton. Crow poison.

Southern United States.

Aletris farinosa L. Colic root.

Said to be poisonous. Sandy soil. E. N. Am.

Medeola virginiana L. Indian cucumber root.

From New Brunswick to Minn., and Florida. Said to be poisonous.

Chamaelirium luteum (L.) Gray. Blazing Star.

Said to be poisonous. Mass. to Ark. and Fla. low grounds.

Chlorogalum pomeridianum Kunth. Soap plant.

Pacific Coast. Contains saponin.

Allium canadense L. Wild onion.

Widely distributed; common in low pastures, N. U. S. Milk is flavored where cattle feed on the plant. The *A. unifolium* of California is poisonous.

Allium tricoccum Ait. Wild Leek.

Eastern and northern States. Taints milk like preceeding.

Lilium superbum L. Turk's-cap lily.

According to Schaffner, this species produces dermatitis. The bulbs produce mental exhaustion and headache.

Asparagus officinalis L. Asparagus.

According to Dr. White, in his Dermatitis Venenata, persons who constantly work with asparagus may have the skin somewhat blistered.

Convallaria majalis L. Lily-of-the-valley.

All parts of this plant are very poisonous to man and domestic animals. Contains two glucosides, convallamarin $C_{43}H_{44}O_{24}$, and convallarin $C_{34}H_{31}O_{11}$.

Trillium grandiflorum, Salisb. Large flowered Trillium.

This is used as an emetic and contains a principle which has been called trillin, found in a few other species of the genus.

Trillium erectum L. Erect Wake-robin.

The root stock of this species is somewhat poisonous.

Smilax rotundifolia L. Round-leaved Greenbrier.

Widely distributed in the northern states. Dr. Schaffner reports a case of poisoning from eating the young leaves of these plants. The spines are injurious in a mechanical way; they cause inflammation and pus formation.

Haemodoraceae.

Lacnanthes tinctoria (Walt) Ell. Red root.

Mass. to Florida. It is said that white hogs are subject to poisoning from this plant.

Amaryllidaceae.

Narcissus poeticus L. Narcissus.

Urticaceae.

Maclura pomifera (Raf.) Schneider. Osage orange.

Cultivated especially in the southern part of Iowa to Nebraska and Southward. The leaves and fruit are more or less poisonous. The thorns upon the plant produce serious injuries giving rise to inflammation.

Humulus Lupulus L. Common Hop.

Hop pickers often have an inflammation of the hands. The plant is a sedative and contains cholin, lupulic acid and oil of humulus.

Cannabis sativa L. Hemp.

Naturalized in many parts of the North. The narcotic effect of the resin of the plant is well known and in India an intoxicating drink is made from the juice of the leaves. Contains the substances cannabin, and oxycannabin.

Urtica dioica L. Stinging nettle.

The urticating properties of our common nettle are known to all who have had any experience in collecting the plants; there is at first a reddening, followed by a swelling, intense burning, and a small amount of itching. It is said to contain formic acid. The Western Nettle *U. holosericea* of Utah has similar properties.

Urtica gracilis L. Slender nettle.

Injurious like the preceding but more widely distributed.

Laportea canadensis Gaud. Wood nettle.

This plant is even more widely distributed than the preceding nettles. It is found in deep woods. It produces an irritation of the skin like that caused by common nettle.

Loranthaceae.

Poisoning has been reported from the European mistletoe *Viscum album* and there are a few references in America regarding poisoning by the false mistletoe *Phoradendron flavescens.* Possibly the Arceuthobiums may also be injurious.

Aristolochiceae.

Artistolochia Clematitis L.

Atlantic States from New York to Md. Some of the European species of Aristolochia are poisonous like the A. Clematitis which is naturalized along the Atlantic Coast.

Polygonaceae.

Rumex acetosella L. Sheep sorrel.

The plant is widely distributed in the U. S. and is becoming more common. Said to be poisonous to horses and sheep. Contains oxalic acid.

Rumex crispus L. Sour dock.

The plant is an astringent and is looked on with suspicion as are some of the other species of the genus.

Fagopyrum esculentum Moench. Buckwheat.

A dermatitis produced by the eating of buckwheat cakes is well known to most people and occasionally where screenings of this material are fed in quantities to hogs a similar rash is produced. Buckwheat straw is also considered poisonous. The plant contains the glucoside indican.

Fagopyrum tataricum Gaertn.

Poisonous like the preceding.

Polygonum acre H B K. Smartweed.

The acrid properties of many of the species of Polygonaceae are well known. This species is widely distributed in the north. Contains probably polygonic acid.

Polygonum hydropiper L. Smartweed.

Poisonous like preceding.

Chenopodiaceae.

Chenopodium anthelminticum L. Worm-seed.

Occasionally reported in the state. Cases of poisoning from the oil of the seeds have been reported in medical literature. Contains the volatile oil of worm-seed. This is a narcotic-acrid poison,

Chenopodium ambrosioides L. Mexican Tea.

This species is occasionally reported with properties like the preceding.

Kochia scoparia (L.) Schrad. Summer cypress.

This plant contains saponin and, according to Greshoff, the extracts from the seeds of another species froth in a solution up to 1 to 700. These plants must be regarded as slightly toxic in their effect. We may add here that saponin has also been found in *Eurotia ceratoides*. *Eurotia lanata* is used as a forage plant in the West; it is commonly called winter fat.

Chenopodium mexicana Moq. Mexican Lamb's Quarters.

This plant contains saponin and is known to be poisonous.

Amaranthaceae.

Amaranthus retroflexus L. Green pigweed.

Common everywhere in eastern north America, also in the great basin. O'Gara reports bloat from it in Nebraska.

Amaranthus spinosus L. Thorny pigweed.

The species are injurious.

Greshoff states that the leaves of *A. hypochondriacus* give an extract which froths strongly and contains saponin. The saponin is only slightly toxic.

Atriplex Nuttallii S. Wats. Salt bush.

The leaves of the salt bush contain saponin and Greshoff also found the same substance in *A. halimus, A. hortensis* and *A. laciniata*. He states that the haemolysis of the seeds of some of the species is moderately great. In China a skin disease known as Atriplicismus is caused by a species of Atriplex.

Beta vulgaris. Mangolds and Sugar Beets.

The feeding of mangolds and sugar beets to sheep causes renal calculi.

Sarcobatus vermiculatus Hooker. Grease wood.

Frequently produces mechanical injuries. According to Chestnut one man lost over 1000 sheep, probably due to bloat, caused by this plant.

Phytolaccaceae.

Phytolacca decandra L. Pokeweed.

The roots and seeds contain a very poisonous substance. The young shoots are eaten as greens; probably the poisonous principle is dissipated on boiling the plant. Found from southern Iowa Eastward and Southward. Contains phytolaccin.

Caryophyllaceae.

Stellaria media L. Chick-weed.

This has been reported as poisonous, although the seeds are eaten by birds.

Agrostemma githago L. Corn cockle or cockle.

Generally found in wheat fields. Screenings are often sold as stock food

and several cases of poisoning from food that contained screenings of cockle have been reported. When cockle is in flour, it is poisonous. Several cases of poisoning from flour containing cockle are on record. Cockle is said to be especially poisonous to poultry. Contains the substances saponin, sapogenin and the alkaloid agrostemmin.

Silene antirrhina L. Sleepy catchfly.

Very widely distributed in the north. Said to be poisonous.

Silene noctiflora L.

Widely distributed in the north. Said to be poisonous.

Widely distributed in northern states. Clover fields. Native to Europe, probably also poisonous.

Saponaria officinalis L. Bouncing Betty.

This plant is said to be somewhat poisonous. Naturalized in the East. Ry Mts. and Pacific Coast.

Vaccaria vulgaris Host. Cow cockle.

Common only in grain fields, seeds said to be poisonous, like corn cockle.

Nymphaeaceae.

Nelumbo lutea Pers. The American Nelumbo.

The root stock is used by the Indians for food. According to Schaffner it is said to be used to destroy cockroaches. Roasting dispels the poisonous principle.

Ranunculaceae.

Hydrastis canadensis L. Orange Root. Golden Seal.

In northeastern Iowa and eastward. Contains the alkaloids hydrastin, berberine, and xanthopuccine. Hydrastis causes severe ulceration and catarrhal inflammation.

Caltha palustris L. Marsh marigold.

The leaves of the marsh marigold are eaten, but the poisonous principle is dissipated on boiling. Plant found on low grounds, especially in northern states. The related species with whitish flower *R. leptosepala* of the Ry Mts. must be regarded with suspicion.

Actaea alba Mill. White baneberry.

More or less poisonous, but generally not eaten by live stock. Found in woods more or less widely distributed in northern states.

Actaea rubra Willd. Red baneberry.

Widely distributed in the state, but never abundant. Berries poisonous.

Delphinium consolida L. Field Larkspur.

Naturalized from Europe. Poisonous and fatal to cattle, frequently cultivated as an ornamental plant. It contains several poisonous alkaloids. The alkaloids, delphinin, delphisin, delphinoidin and staphisagrin occur in *D. Staphisagria* and may be looked for in some of our native larkspurs.

Delphinium carolinianum Walt. Carolina Larkspur.

Native to prairies, especially gravelly knolls. Reported as fatal to cattle.

Delphinium exaltatum Ait. Tall Larkspur.

Frequently cultivated, native to Europe.

Delphinium tricorne Michx.

Produces fascicled tuberous roots. Common in southern Iowa. Very poisonous to cattle.

Delphinium hesperium Gray. Larkspur.

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same genus. The herbage is said to be acrid and caustic. The juice of some species of the genus causes blisters, or even ulcers. The fresh leaves of the *C. erecta* are used as a vesicant in Europe, especially by beggars, hence sometimes called beggar's weed.

Clematis Fremonti Wat. Clematis.

Common from Missouri to Kansas.

Clematis Pitcheri Torr. & Gray.

From Southern Indiana to Texas.

Clematis ligusticifolia Nutt.

Very abundant in the Rocky Mountains west to the Pacific Coast along streams at a lower altitude. Greshoff reports saponin in a large number of species, notably *C. Pitcheri, C. recta,* and others; also hydrocyanic acid in *C. Fremonti.*

Fig. 19a. Common Crowfoot (*Ranunculus acris*). An acrid poison. (U. S. Dept. Agr.).

Ranunculus acris L. Tall buttercup.

Poisonous, causes inflammation when it comes in contact with mucous membrane. Rarely found in Iowa, but sometimes naturalized; common eastward.

Ranunculus septentrionalis Poir. Creeping Buttercup.

Widely distributed in low grounds. Acrid like the preceding species.

Ranunculus fascicularis Muhl. Tufted buttercup.

Found in eastern Iowa, as far west as the Iowa River; common north and east. Probably poisonous like the preceding species.

Ranunculus bulbosus L. Bulbous Crowfoot.

Common eastward, naturalized from Europe.

Ranunculus abortivus L. Crowfoot.

Common weed in many parts of the north, the leaves are quite acrid and have a sharp, peppery taste.

Ranunculus sceleratus L. Cursed crowfoot.

A very poisonous species, especially to cattle, since it grows in marshes along with other herbage and is often eaten with other forage plants. Contains anemonol and anenmonic acid.

Fig. 19b. Common Poppy (*Papaver somniferum*). Narcotic. (Lois Pammel).

Anonaceae. Custard Apple Family.

Asimina triloba Dunal. Papaw.

It occurs in eastern Iowa as far north as Clinton and Dubuque. Common Southward. The pawpaw is commonly eaten, but a case of poisoning is reported.

Trollius laxus Salisb. Spreading globe flower.

From western Connecticut to Michigan and the Rocky Mountains. Several species contain saponin and *T. Europaeus* is considered poisonous in Europe. It contains saponin.

Thalictrum revolutum DC. Meadow Rue.

Common in low moist meadows in Western North America. All of the species of meadow rue are more or less acrid. The *T. polycarpum* of California is poisonous to man according to Chesnut.

Magnoliaceae. Magnolia Family.

Magnolia grandiflora L. Magnolia.

The flowers of this plant are said to be injurious.

Illicium floridanum Ellis. Anise tree.

Native to Florida. The leaves are poisonous to stock.

Berberidaceae. Barberry Family.

Berberis repens. Lindl. Trailing Mahonia.

Cultivated. Poisonous. According to Schaffner, the berries are injurious to birds. Probably contain the alkaloids berberin and oxyacanthin which occur in the common barberry.

Berberis Aquifolium Pursh. Oregon grape.

This plant also causes poisoning. Cultivated. In the Cascade Mts., Oregon, Washington.

Podophyllum peltatum L. May Apple or Mandrake.

This plant is widely distributed east of the Missouri divide. The roots and leaves are drastic and are known to be poisonous. It is said also that when the leaves are eaten by cows, they produce injurious milk. The roots, according to Dr. White, are irritating to the eye, nose, mouth and skin, and contain picropodophyllin, podophyllotoxin. The podophyllin is a resinous mixture.

Caulophyllum thalictroides Mich. Blue Cohosh.

Widely distributed from Iowa eastward and northward. Said to be poisonous.

Menispermaceae.

Menispermum canadense L. Canadian Moonseed.

This plant is widely distributed in woods in Iowa and the north. Contains menispermin, and menispin. A case is reported of the death of three boys from eating the berries in mistake for grapes. The *Cocculus indicus* is a well-known remedy for the destruction of pediculi and is known to be poisonous. It contains picrotoxin, cocculin and an alkaloid menispermin.

Calycanthaceae. Allspice Family.

Calycanthus floridus L.

Cultivated in Southern Iowa and in the southern states. The aromatic properties of the flowers resemble those of strawberries. This makes it a very desirable cultivated ornamental plant. It contains an active principle calycanthin. Chesnut records it as poisonous.

The other species of Calycanthus may also be regarded as poisonous.

Lauraceae. Laurel Family.

Sassafras officinale Nees. Sassafras.

According to Schaffner the berries of this plant are reported to be poisonous. This species is native to southeastern Iowa, southward and eastward.

Umbellularia Californica (Arnott) Nutt. California Laurel.

California. Leaves smart. Used to drive fleas away.

Papaveraceae. Poppy Family.

Papaver somniferum L. Opium Poppy.

Opium is obtained from the common garden poppy. This species is widely cultivated in the state, and is frequently spontaneous. The more common alkaloids found in the poppy plant are morphin, codein and narcotin. The seeds of poppy are sometimes used to spread on top of cookies and bread.

Papaver rhoeas L. Corn Poppy.

mentioned. Dr. Rushy refers to its irritating properties when taken in excess especially because of its action upon the urinary organs. One case referred to by Dr. Johnson was extreme and serious.

Sisymbrium officinale Scop. Hedge Mustard.

This plant is widely distributed in this state, as a weed, and produces more trouble than mustard.

Sisymbrium altissimum L. Tumbling Mustard.

This weed is common in grain fields in the north and may thus find its way into wheat screenings which should be fed with caution. It probably produces sinapism, as do the other plants of the order. It is also found in Iowa.

Brassica arvensis (L.) Ktze. Charlock.

This weed produces sinapism. After the application of the powdered material, there is a sense of burning. The volatile oil of mustard is a powerful irritant, and caustic, and should be used with caution.

Thlaspi arvense (Tourn.) L. Penny Cress.

This weed is said to be injurious to animals in Canada and is more or less avoided by them in pasture. It is widely distributed in Northern United States, but is more abundant in Canada.

Capparidaceae. Caper Family.

Cleome serrulata Pursh. Stinking Clover.

This plant is more or less pungent and acrid; it is widely distributed from Missouri River westward across the continent, especially along railways, etc. The flowers are showy and purple.

Cleome lutea Hook. Western Cleome.

This plant is a western species with yellow flowers and has similar properties to the one named above.

Polanisia graveolens Raf.

Fetid annual, with glandular hairs and common in sandy soils in the eastern States. The *P. trachysperma* T. & G. is similar to the preceding. This plant is found from Iowa to Kansas.

Sarraceniaceae.

Sarracenia purpurea L. Side-Saddle Flower.

Probably not native to Iowa, although it is found distributed with Drosera in sphagnum bogs. It contains the substance sarracenin. The root produces diuresis, gastric excitation, and an increased, irregular action of the heart. It produces papular eruptions changing to vesicular with depression as in smallpox. The plant was formerly used medicinally by the Indians.

Droseraceae. Sundew Family.

Drosera rotundifolia L. Sundew.

Sundew is said to be poisonous to cattle. In bogs in the northern states.

Greshoff reported hydrocyanic acid in this species and in *D. intermedia.* Several other species in Europe were reported as harmful and toxic to cattle.

Crassulaceae. Orpine Family.

Sedum acre L. Stone Crop.

Produces inflammation when applied to the skin of many persons. The juice is acrid and biting.

Other species of this genus are known to be acrid. Several species with yellow flowers are common in rocky soils in the Rocky Mountains.

Saxifragaceae.

According to Greshoff several species of the genus Saxifraga of Europe contain hydrocyanic acid. The order also contains the currant and gooseberry which are well known food plants.

Hydrangea arborescens L. Wild Hydrangea.

Common in the Southern States and woods from Southern Iowa eastward to New York. According to Greshoff this species contains saponin; hydrocyanic acid occurs in the well known ornamental plant *H. Hortensia.*

Jamesia americana T. & G. Jamesia.

This plant is widely distributed from New Mexico to Montana. The leaves of this Rocky Mountain shrub contain hydrocyanic acid.

Philadelphus grandiflorus Willd. and P. coronarius L.

Mock Orange or Syringa. Native in the mountains from Virginia to Florida. The well known cultivated syringa contains saponin; according to Greshoff the Rocky Mountain *P. microphyllus* also contains saponin.

Deutzia. Several species of Deutzia, as *D. staminea,* contain saponin.

Chrysosplenium oppositifolium L.

This plant contains some saponin and the *C. tetrandum* Fries., is known to be poisonous to sheep.

Rosaceae. Rose Family.

Fragaria vesca L. European Strawberry.

Found on sand-stone and limestone rocks. In some people it produces irritation of the stomach.

Fragaria virginiana Mill. Wild Virginia Strawberry.

Similar to the preceding.

Fragaria chiloensis Duchesne. Cultivated Strawberry.

Like the preceding. There are people who can not eat strawberries or pick them without being irritated.

Rosa arkansana Porter. Arkansas Rose.

Widely distributed in the west, especially in prairie regions. Not poisonous, but the bristles and prickles often enter the skin and produce serious inflammation. Other species produce mechanical injuries.

Rosa rubiginosa L. Sweetbriar.

A frequent escape in pastures. The recurved spines and prickles are injurious like those of the preceding species.

Pyrus Aucuparia Meyer. Mountain Ash.

The berries are poisonous to man, but not to birds. However, they are readily disseminated by birds.

Pyrus communis L. Pear.

Dr. Schaffner states that horses are reported to have been killed by eating rotten pears.

Nuttallia cerasiformis T. & G. Oso Berry.

It is found in moist places and on north slopes in western U. S., and contains amygdalin. It is poisonous.

Gillenia stipulata (Muhl) Trel. Indian Physic.

From New York to Kansas. This species and *G. trifoliata,* known as Bowman's Root, are said to contain a poisonous glucoside.

Filipendula ulmaria (L.) Maxim Queen of the meadow.

It is cultivated and occasionally an escape; said to be poisonous.

Sanguisorba canadensis L. Burnet.

From Labrador to N. Mich. and Alleghany Mts. Said to be poisonous.

Fig. 19c. Coffee Bean (*Gymnocladus dioica (L) Koch.*
The sweet juice in pod is poisonous. (Ada Hayden).

Pyrus malus L. Apple.

The seeds are poisonous and contain the glucoside which is changed into hydrocyanic acid.

Crataegus mollis L. Haw.

Cases of poisoning are reported by persons eating the fruit of *Crataegus mollis,* the injurious effects being probably due more to the inedible seeds than to the fruit.

Prunus americana Marsh. Wild Plum.

The shoots and seeds contain the principle amygdalin, which is converted into hydrocyanic acid. The fruit, it should be said, is entirely harmless.

Prunus pumila L. Sand Cherry.

This cherry is not widely distributed in the state, but is found along the Missouri river and in sandy soil eastward. The fruit is slightly acid and somewhat astringent, but is not poisonous, except the wilted leaves and the seeds, The astringent qualities in our wild fruit are undesirable. The same is true of P. Besseyi of Nebraska, the Dakotas and the Rocky Mountains.

Prunus pennsylvanica L. Wild Red Cherry.

Common, especially in the eastern part of Iowa and northern states. The leaves are poisonous, as well as the seeds. The fruit is edible.

Prunus virginiana L. Choke Cherry.

The leaves and seeds are poisonous. The fruit is so astringent it often produces very unpleasant conditions when eaten in any considerable quantity. Choke Cherry is widely distributed in the north. The leaves in the wilted condition contain hydrocyanic acid.

Prunus demissa (Nutt) Walp. Choke Cherry.

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Crotalaria sagittalis L. Rattle-box.

This is found in the western part of the state in the Missouri River Bottoms and produces a disease known as the Missouri Bottom disease, called crotalism, by Dr. Stalker. The seeds contain an unnamed alkaloid found by Dr. Power. The plant is not only poisonous in the meadow, but also in hay.

Cytisus scoparius (L.) Link. Scotch Broom.

Found along the Pacific and Atlantic oceans. It is common in Washington and Oregon; also in Massachusetts and Virginia. The flowers are yellow. The plant contains cytisin which also occurs in several other genera of this family like *Ulex, Thermopsis* and *Baptisia.*

Lupinus albus L. Lupine.

The European lupine is occasionally cultivated and in Europe it produces a disease known as lupinosis. Our native species, *L. perennis,* is also regarded as poisonous. Contains lupinin; lupinidin; lupinin.

Lupinus leucophyllus Dougl. Western Lupine.

It is said to be poisonous in Montana, although this is disputed by some. Other suspected species of the blue lupines are *L. argenteus* and *L. argophyllus.*

Trifolium incarnatum L. Italian or Crimson Clover.

According to Coville, it produces "hair" balls. This plant is cultivated as a cover crop in the south and east.

Trifolium repens (L.) White clover.

This plant and several other species such as *T. pratense,* at times, produce bloat.

Melilotus alba Desv. White Sweet Clover.

Widely distributed in the U. S. as a weed. The honey bees collect considcrable quantities of honey from the Sweet Clover blossoms. It has been looked upon with suspicion. Dr. Schaffner states that the seeds impart a foul odor to flour.

Melilotus officinalis Willd. Yellow Sweet Clover.

This is also widely distributed in the state and is objectionable like the preceding.

Medicago sativa L. Alfalfa.

Alfalfa may cause bloat.

Psoralea tenuiflora Pursh. Slender Psoralea.

Common from Illinois to Texas. Perennial herb sprinkled with little glandular dots. The Silvery Psoralea, *P. argophylla,* was reported from Iowa as poisonous. The tuberous roots of *P. esculenta* Pursh. were eaten by the pioneers and Indians.

Tephrosia virginiana Pers. Goat's Rue.

It grows only in sandy soil from Wisconsin to Iowa, east and south. It was used by Indians as a fish poison. Several other species in South America and Mexico have been used in a similar way. One is called *T. toxicaria,* and is a well known fish poison. Other N. Amer. species probably also poisonous.

Sesbania vesicaria Muhl.

An annual vine of the Southern States, Carolinas and Westward. The seeds are said to be poisonous.

Robinia neo-mexicana A. Gray.

Common in New Mexico and frequently cultivated as a hedge plant in Southern Colorado. The leaves somewhat resemble those of the black locust.

Robinia pseudacacia L. Locust-tree. Black locust.

This plant is frequently cultivated in the north and west, and in numerous places is an escape from cultivation. Native to N. Y. and Alleghany Mts. The roots, leaves and bark are very poisonous to man. Contains robinin.

Robinia viscosa Vent. Clammy locust.

Cultivated as an ornamental plant; the roots are somewhat poisonous.

Astragalus mollissimus Torr.

A loco-weed from Neb. to New Mexico and Wyoming.

Astragalus hornii A. Gray.

Poisonous in Arizona and adjacent regions.

Oxytropis lamberti Pursh. Stemless Loco Weed.

Found in the western part of Iowa, along the Missouri River and its tributaries, very abundantly. One of the conspicuous loco or crazy weeds of the west.

Coronilla varia L. Coronilla.

An escape from cultivation in the West. It has long been regarded as a poisonous plant in Europe.

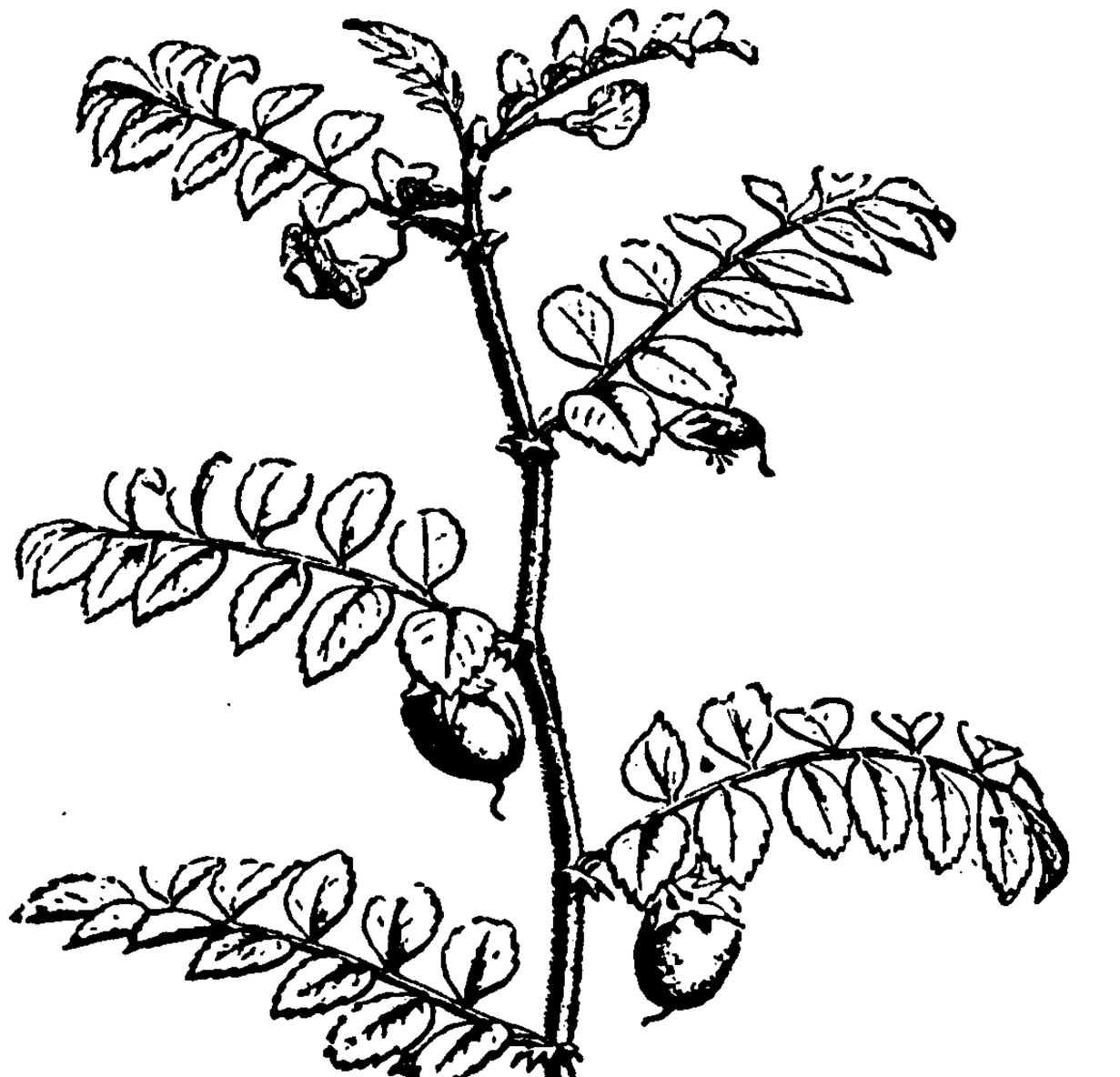


Fig. 19d. Chick Pea (*Cicer arietinum*) said to be poisonous.

Phaseolus lunatus Linn. Lima bean.

Investigations carried on in Europe seem to indicate that the lima bean leaves in the wilted condition contain hydrocyanic acid. According to Guignard practically all varieties, whether wild or cultivated, were found to contain a principle which when acted upon by an enzyme yields hydrocyanic acid. Prolonged boiling, however, extracts the greater part of it, but it is not destroyed, consequently this water should not be used, as it contains the substance which is converted into hydrocyanic acid.

Phaseolus multiflorus Willd. Scarlet Runner.

Commonly cultivated in gardens. The root is poisonous.

Vicia sativa L. Common Vetch.

A frequent weed in grain fields. The seeds of this are said to be injurious to pigs. It is not injurious to cows. Contains vicin.

Prosopis juliflora DC. Mesquit Tree.

The seeds of this tree contain a small amount of saponin, probably it is not very strongly poisonous as it is used as food for cattle in Texas.

Cicer arietinum L. Chick Pea. Idaho Pea.

Commonly cultivated in Southwestern U. S. Said to be poisonous in Europe.

Geraniaceae. Geranium Family.

Erodium cicutarium (L.) L'Her. and E. moschatum (L.) L'Her. Storksbill.

Both of these plants are widely distributed, especially on the Pacific Coast. The former is becoming abundant in Utah. The pointed callus of the seed sometimes inflicts mechanical injuries.

Oxalidaceae. Wood Sorrel Family.

Oxalis violacea L. Wood Sorrel.

Dr. Schaffner reports a case of a boy who was poisoned from eating a considerable quantity of the leaves. The leaves are frequently eaten as a salad.

Tropaeolaceae. Nasturtium Family.

Tropaeolum majus. L. Nasturtium.

This plant is commonly cultivated and has more or less acrid properties.

Linaceae. Flax Family.

Linum usitatissimum L. Flax.

Said to produce death to cattle, probably due to the formation of HCN in the wilted leaves. People working with the fiber of the plant often have a form of dermatitis. Flax-seed, when fed in considerable quantities to live stock, especially hogs, produces death. *Linum catharticum* contains a bitter principle *linin,* and *linamarin.*

Linum rigidum Pursh. Large-flowered Yellow Flax.

This plant is reported as poisonous to sheep in some parts of the country. Found westward.

Zygophyllaceae.

Tribulus terrestris (L.) Caltrop.

Caltrop is found chiefly from Nebraska to Kansas and occasionally eastward. *T. maximus,* sometimes called soap-brush, is found in the west. The prickly fruit is more or less injurious; it presumably contains saponin. This plant should be looked upon with suspicion as one exotic species is regarded as poisonous.

Rutaceae. Rue Family.

Ruta graveolens L.

This plant is more or less acrid and produces blisters. Several species of the family are regarded as poisonous.

Zanthoxylum scandens and Z. *alatum* are used as fish poisons in India. It is not known whether our native prickly ashes are poisonous or not, but they may be looked upon with suspicion.

westward to Texas. *E. maculata* is common everywhere east of the Rockies.

The *E. Cyparissias* was recently sent to me from Logan Iowa, where it is said to have killed lambs.

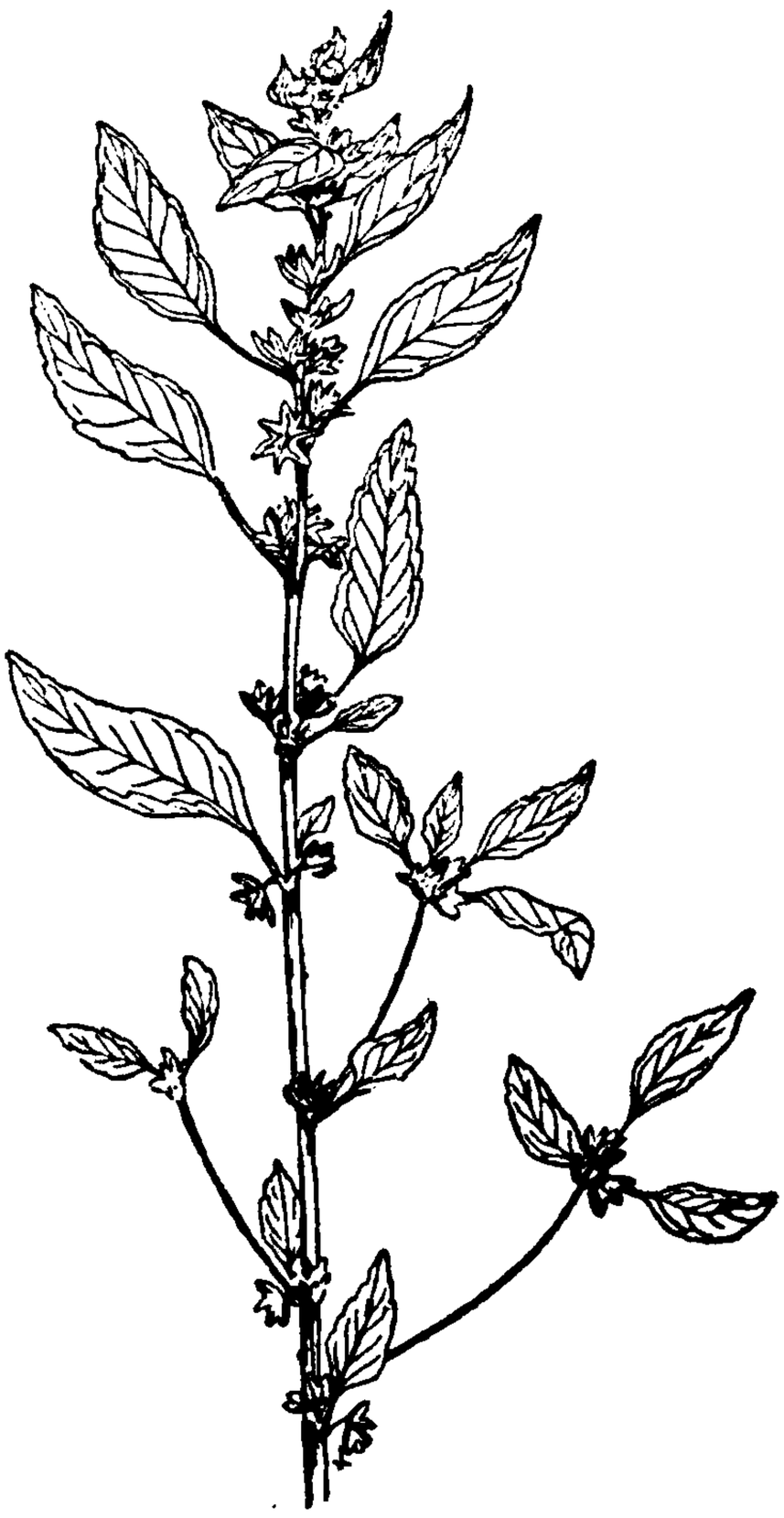


Fig. 19e. Flowering Spurge (*Euphorbia corollata*). (Lois Pammel). Fig. 19f. Three Seeded Mercury (*Acalypha virginica*). Both plants poisonous.

Acalypha virginica L. Three Seeded Mercury.

Common in fields and open places from Canada and Minnesota to the Gulf. The leaves of this plant turn purple in the Autumn.

Jatropha stimulosa Michx. Spurge or Bull Nettle.

This plant has stinging bristles and is common in sandy soil from Texas to Missouri, Virginia and Florida.

Tragia nepetaefolia Cav. Nettle Spurge.

With stinging hairs, common in the south. Other species in the Rocky Mountains and south also have stinging hairs.

Buxaceae.

Buxus sempervirens L. Box.

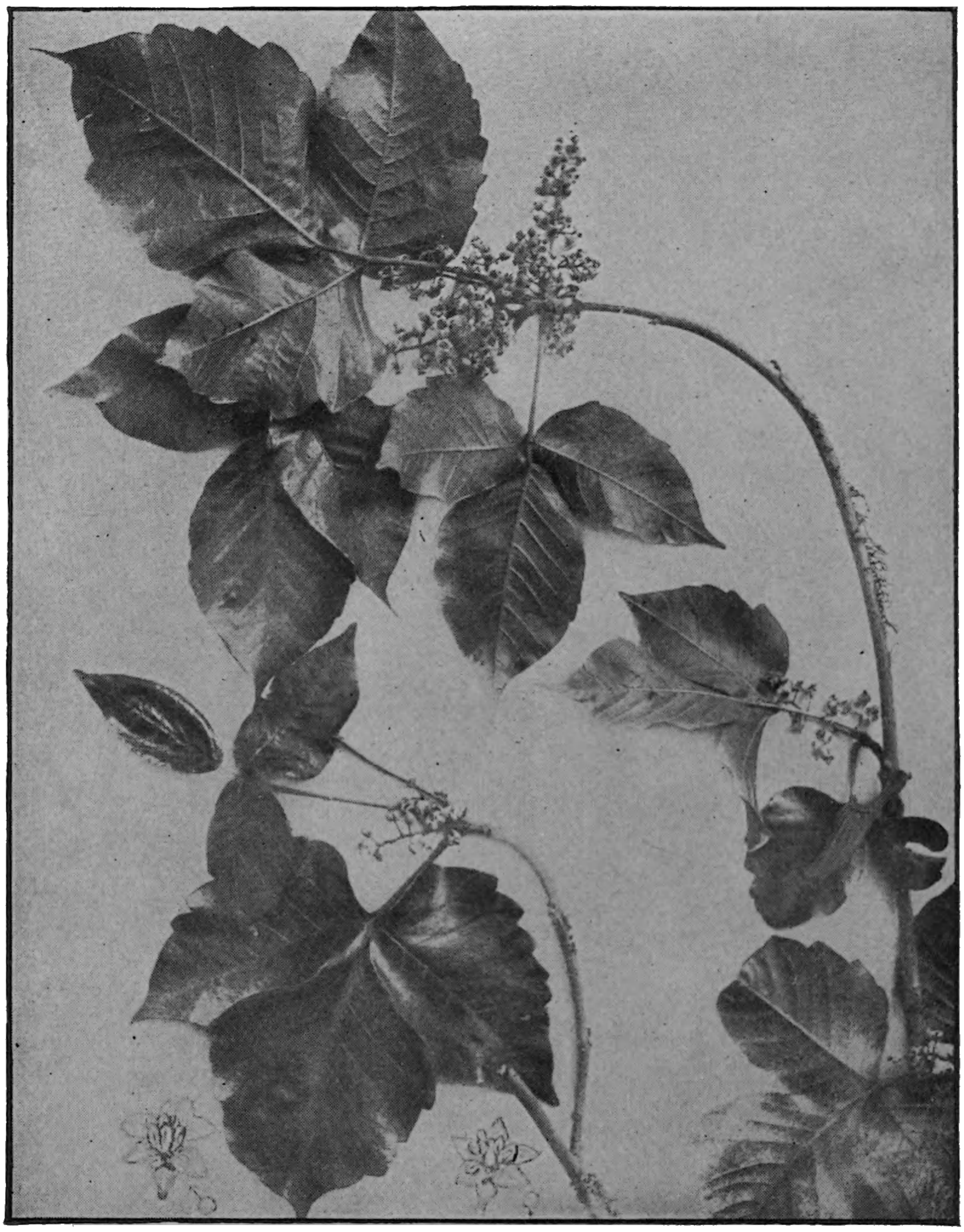
Cultivated in the eastern states for hedges. It is a well known poisonous plant.

Anacardiaceae.

Rhus toxicodendron L. Poison Ivy.

The leaves and stems are poisonous to many people. The form of dermatitis produced, and the seriousness of the case varies according to the susceptibility of the individual. The plant is widely distributed in the state.

Rhus vernix L. Sumac.



Poison Ivy. (After Halsted)

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Frequently cultivated and used as a hedge plant. The ripe fruit is said to be poisonous. It contains the glucosides rhamnin, rhamnetin, and rhamnocathartin.

Rhamnus lanceolata Pursh. Buckthorn.

Native to southern Iowa, east and south. Has the same properties as the preceding species.

Rhamnus Frangula L.

This plant is considered as poisonous. The *R. Frangula* of Europe is commonly cultivated. The tropical Colletia is poisonous.

Karwinskia Humboldtiana Zucc. Cajotillo.

Southwestern U. S. Dr. Mitchell of the 3rd U. S. Cavalry says that it is poisonous to goats. The berries have long been regarded as very poisonous.

Ceanothus velutinus Dougl. Ceanothus.

This species with shiny leaves and small white flowers occurs abundantly in the Rocky Mountains from New Mexico to British America, to Oregon and Washington. According to Greshoff, it contains saponin in considerable quantities. The same authority states that several other species examined by him contained the same substance, among them were *C. azureus* Desf., and *C. thyrsiflorus* Eschw. He also found the same substance but in less quantity in the seeds of the New Jersey tea *C. americanus L.* and *C. ovatus Desf.*, two shrubby plants of Eastern North America, the latter being also common on the east slope of the Rockies.

Vitaceae. Grape Family.

Psedera quinquefolia (L.) Greene. Virginia Creeper.

Common from New England to the Rocky Mountains, especially east of the Missouri River. This plant is regarded as poisonous by some. The leaves and fruit abound in raphides. In this connection it may be of interest to state that the fruit of *Cissus nivea* of the old world produces poisonous fruits, and that the *C. pruriens* with a pleasant, acid, sweet taste produces a painful, burning sensation later. The same is true of the southern Mustang Grape (*V. candicans*).

Malvaceae.

Abutilon Theophrasti (Medic) Gaertn. Velvet-Leaf. Indian Mallow. Butter Print.

Widely naturalized in the state. The plant gives off a very disagreeable odor, and is suspected of being poisonous.

Gossypium herbaceum L. Common Cotton.

The root of cotton is well-known as an abortive. Feeding excessive amounts of cotton seed meal frequently produces death in animals, particularly in hogs.

Dr. Crawford states that this is due to meta and pyrophosphoric acid in cotton seed meal.

Sida urens L. The Stinging Sida.

Occurs in tropical America and produces mechanical injuries because of the hairs with which it is covered. This is also true of the *S. paniculata* L. Probably other species of Sida of the southern states may produce similar mechanical injuries.

Hypericaceae.

Hypericum maculatum Walt. Spotted St. John's-Wort.

All the species are suspected of being poisonous to horses. Vesicant.

Hypericum perforatum L. Common St. John's-Wort.
Naturalized in Eastern U. S. A well known vesicant.
Hypericum Ascyron L. Great St. John's-Wort.
Most widely distributed species in the state, in woods or borders of woods.

Violaceae.

Viola odorata L. Sweet Violet.
Commonly cultivated in greenhouses. Said to be somewhat poisonous. Underground parts of the plant are emetic.

Viola cucullata Ait. Common Blue Violet.

The most widely distributed species in the eastern states; the roots, like the preceding, are emetic.

Loasaceae.

Mentzelia ornata Torr. & Gray. Mentzelia.

The backwardly-barbed trichomes sometimes produce mechanical injuries. Found in Northwestern Iowa on the Big Sioux near Sioux City, and west and southwest.

Datiscaceae.

Datisca glomerata (Presl) Benth & Hook.
California fish poison.

Cactaceae.

Opuntia Rafinesquii Engelm. Cactus.

Found in sandy soil in the state. The barbed trichomes cause mechanical injuries.

Other spiny species of Opuntia, Mammillaria and Cereus, largely developed in southwestern United States, produce similar mechanical injuries.

Anhalonium Lewinii Henn. Mescal Bean.

This cactus contains a narcotic substance which has long been used by the Indians of Mexico and adjacent regions to produce narcosis.

Cereus grandiflorus Mill. Night-blooming Cereus.

It has been regarded with suspicion where the plant is cultivated and native. Frequently cultivated in the United States.

Thymelaeaceae. Mezereum Family.

Daphne Mezereum L. Spurge Laurel.

This is a well-known poisonous plant of Europe and is occasionally cultivated. It contains the glucosides daphnin and daphnetin.

Other cultivated species in North America are known to be poisonous and several of the related genera native to Asia and Australia are poisonous.

Dirca palustris L. Leather-Wood, Moose-Wood.

Found in the North to Iowa. The bark is acrid, the berries narcotic and poisonous.

Elaeagnaceae. Oleaster Family.

Hippophae rhamnoides Linn.

A native of Europe but cultivated in the United States and said to be narcotic.

Araliaceae. Ginseng Family.

Fatsia horrida (Smith) B. & H.

From Isle Royal, Lake Superior region, to the Rocky Mountains and Alaska, and California. It causes mechanical injuries.

Umbelliferae. Parsley Family.

Conium maculatum L. Poison Hemlock.

Introduced here and there in Iowa and eastward, common in Utah. The plant contains the alkaloids coniin, conydrin, methylconiin, and a bitter principle cicutoxin. A very poisonous plant both to man and lower animals.

Petroselinum hortense Hoffm. Parsley.

Some people are suspicious of parsley. Dr, Schaffner states that the seeds are injurious to birds. He reports a case of poisoning of several parrots from eating the leaves of this plant. Cultivated.

Apium graveolens L. Celery.

Several cases are known where persons who have handled celery have had a form of dermatitis. Some persons cannot eat celery because a rash forms.

Cicuta maculata L. Water Hemlock. Cowbane.

The roots of this plant are very poisonous. The plant is widely distributed in the north, especially in low grounds. The European cowbane, *C. virosa,* contains the alkaloid coniin, a substance which probably also occurs in our plant. The poisonous principle is cicutoxin.

Cicuta bulbifera L. Bulb-bearing Hemlock.

The roots of this, and the whole plant are supposed to be very poisonous. In swamps and northern states.

Cicuta vagans Greene. Cowbane.

Occurs in Washington, Oregon and California. Occasionally said to poison cattle drinking water in which they have trampled roots of this plant, thus expressing the extract. Very poisonous.

Cicuta Bolanderi A. Gray.

Found in marshy regions in California.

Cicuta occidentalis Dougl. Cowbane.

Found in the Rocky Mountains and considered poisonous.

Sium cicutaefolium Gmelin. Water Parsnip.

Common in many parts of the north in low grounds. Said to be poisonous.

Aethusa Cynapium L. Fool's Parsley.

A poisonous herb native to Europe, with a disagreeable odor. Possibly occurs in a few places in the state. Contains the alkaloid cynapin and another coniin-like alkaloid.

Angelica atropurpurea L. Purple-stemmed Angelica.

Found in low grounds in North and Northeastern Iowa. Possibly poisonous. Cattle do not relish it.

Oxypolis rigidior (L.) Coult. & Rose. Cowbane.

Aquatic herb with white flowers, leaves simple pinnate with 3-9 linear-lanceolate leaflets.

Pastinaca sativa L. Parsnip.

Persons are often poisoned by handling the plant, which causes inflammation and vesication. Mr. F. C. Stewart, in a letter to the writer, states that in one case, the eyes became swollen, vesication occurred from poisoning caused by the flowers.

Heracleum lanatum Michx. Cow Parsnip.

Supposedly poisonous, although the leaves of the fresh plant are eaten by the Indians. This species is widely distributed in Iowa, especially in rich woods. Contains the bitter principle heraclin.

With white flowers in axillary or spiked racemes; shrub. From Virginia to Georgia in the mountains; an allied species *L. racemosa* (L.) Gray, occurs in moist thickets from Massachusetts to Louisiana, near the coast. Both species are said to be fatal to stock, the latter especially to calves.

Lyonia mariana (L.) D. Don. Stagger-bush.

From the Atlantic Coast to Tennessee and Arkansas. Said to produce intoxication, hence the name.

Primulaceae. Primrose Family.

Anagallis arvensis L. Poor Man's or Shepherd's Weather Glass. Pimpernel.

Possibly growing in the state. Known to be poisonous. Contains glucoside cyclamin.

Primula obconica Hance.

Poisonous to the touch; very much like poison ivy. This plant is commonly cultivated in greenhouses.

Primula Parryi A. Gray. Parry's Primrose.

Common in higher altitudes in the Rocky Mountains, especially near brooks or springs. The root has the odor of musk, and is said to be poisonous.

Cyclamen Europaeum L. Cyclamen.

This beautiful cultivated plant has long been regarded as poisonous in Europe. The *C. persiclum* Mill, is also regarded as poisonous.

Plumbaginaceae. Plumbago Family.

Limonium carolinianum (Walt.) Britton.

It is not known whether this plant is poisonous or not but a related plant, Statice pectinata Ait, of the Cape Verde Islands is poisonous.

Sapotaceae. Sapodilla Family.

Achras Sapota L.

A tropical fruit cultivated in Florida. It is said to be injurious and according to Greshoff contains saponin. Lucuma forms a well-known genus of West Indian fruit trees. In *L. Bonplandia*, H. B. K., Altamirans demonstrated amygdalin as early as 1876, and in another species a cyanogenetic glucoside was suspected, but the experimental proof was wanting. Greshoff reports hydrocyanic acid in the *L. mammosa.*

Ebenaceae. Ebony Family.

Diospyrus virginiana L. Persimmon.

Common in the Southern States as far north as southern Iowa. It is used as an anthelmintic, but it is not positively known whether any part of this plant is poisonous; several exotic species, however, blister the skin and one species in Madagascar is said to be very poisonous.

Oleaceae. Olive Family.

Ligustrum vulgare L. Privet.

The privet is frequently cultivated in the U. S. The leaves and fruit of the plant are said to be poisonous. Prof. Chesnut says that accidents have been occasioned in children, both by fruit and the leaves. It contains the bitter principle syringopicrin.

Forsythia suspensa Vahl. Forsythia.

Commonly cultivated in northern states. The root is slightly poisonous. The *F. viridissima* Lindl. is very bitter. Greshoff has found saponin in the seeds of the former species.

Mountain Laurel (After Halsted)

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Nerium Oleander L. Common Oleander.

Cultivated. The leaves are deadly poisonous to stock. Contains conessine, and neriin, which has the properties of digitalin. Nerianthin bears a resemblance to digitalin.

Dr. Wilson of the Arizona Experiment Station has recently demonstrated the very poisonous nature of this plant in Arizona.

Asclepiadaceae. Milkweed Family.

Asclepias tuberosa L. Pluerisy-Root.

Widely distributed in Iowa, especially on gravelly knolls and prairies. The leaves are more or less poisonous to stock. However, honey bees collect considerable honey from this plant.

Asclepias incarnata L. Swamp Milkweed.

Poisonous probably like the preceding. The root is emetic and cathartic.

Asclepias syriaca L. Milkweed.

Poisonous. Contains the glucoside asclepione, an amorphous bitter substance.

Asclepias speciosa Torr. Showy Milkweed.

Poisonous. This species is found in Western and Northwestern Iowa to Utah.

Asclepias campestris, Decne. Milkweed.

Commonly cultivated in gardens southward. It is said to be poisonous.

Asclepias eriocarpa Benth. Milkweed.

Common in California and adjacent regions. According to Chesnut sheepmen in California very much fear this weed. It has broad mullein-like leaves.

Asclepias mexicana Cav. Narrow Leaved Milkweed.

Native to California, Oregon and Nevada. According to Chesnut sheep and calves are not infrequently poisoned by eating this plant and cows have been poisoned by eating hay contaminated with it.

Convolvulaceae. Convolulus Family.

Ipomoea pandurata Meyer. Wild Potato Vine. Man of the Earth.

The large root is poisonous. Contains the gulcoside impomoein.

Convolvulus sepium L. Hedge Bindweed.

The plant produces a somewhat disagreeable odor. Dr. Schaffner states that it is supposedly poisonous to swine. Jalap contains several glucosides which also probably occur in our Morning Glory. One is convolvulin.

Cuscuta epithymum Murr. Clover and Alfalfa Dodder.

Dr. J. Q. Taylor of Lisbon, Ohio, in writing to Prof. A. D. Selby stated that dodder produced a bowel trouble in horses, and Dr. Jenkins of New Haven writes that clover hay containing a great deal of dodder produced scours. He added that the hay had moulded badly, although the injury could not be definitely traced to dodder. Some exotic dodders are poisonous. The dodder must, therefore, be looked upon with suspicion.

Polemoniaceae. Polemonium Family.

Gilia aggregata Spreng. Cypress plant.

Common in the Ry. Mts. to Neb. This species according to Greshoff contains a considerable amount of saponin and is very poisonous. He lists several other species which contain this substance.

Hydrophyllaceae. Waterleaf Family.

Phacelia circinata Jacq. Rough Phacelia.

This plant is common in the Rocky Mountains. It produces rough bristles and causes considerable irritation and inflammation; other species act in a similar way.

Boraginaceae. Borage Family.

Heliotropium Europaeum L. Wild Heliotrope.

This plant contains a poisonous alkaloid and as well as the *H. indicum* L. has long been regarded as poisonous.

Cynoglossum officinale L. Hound's Tongue.

Poison acts much like Curare.

Cynoglossum virginicum L. Wild Comfrey.

Supposed to be poisonous.

Lappula officinalis Lehm. Stickweed.

The fruit of this plant gets into the wool of sheep and sometimes produces mechanical injuries.

Echium vulgare L. Viper's Bugloss.

Occasionally spontaneous, probably in the Eastern part of the state. Contains a poisonous alkaloid.

Verbenaceae. Verbena Family.

Callicarpa americana L. Mexican Mulberry.

Common in the southern states. Several Asiatic species of the genus used as fish poisons. No report of poisoning from the American species is recorded.

Labiatae. Mint Family.

Scutellaria galericulata L. Marsh Skullcap.

Common in wet places in the North. Suspected of producing poisoning.

Stachys arvensis L. Corn or Field Woundwort.

Waste places, especially along the Atlantic Coast. Suspected.

Galeopsis tetrahit L. Dead Nettle.

Common hemp nettle with purplish flowers and stiff calyx bristles. Common Eastward and in the Northwest in British Columbia and Washington. Causes irritation.

Nepeta hederacea (L.) Trevisan. Ground Ivy.

Widely naturalized in the state. It is said to be poisonous to horses.

Hedeoma pulegioides Pers. Pennyroyal.

Common, especially in clay soils in Eastern Iowa. The oil is known to cause poisoning.

Leonurus Cardiaca L. Common Motherwort.

Known to produce mechanical injuries and dermatitis. Widely distributed in the U. S., naturalized in Europe.

Solanaceae. Ninghshadt Family.

Nicandra Physalodes (L.) Pers. Apple of Peru.

Cultivated here and there in Iowa. Said to be poisonous. Used as a fly poison in parts of the United States.

Solanum nigrum L. Black Nightshade.

The leaves and other parts of the plants are reputed to be poisonous to calves, sheep, goats and swine, and the green berries are known to be poisonous to man. The fruit of a form of this species is cultivated as an esculent.

Fig. 19i. Black Nightshade (*Solanum nigrumy*). Contains a poisonous alkaloid. (Chesnut, U. S. Dept. Agr.).

The writer has not only eaten berries of this, but has seen others eat berries of this and the common Black Nightshade without injurious results. Contains the alkaloid solanin, with a hot, bitter taste.

Solanum triflorum Nutt. Nightshade.

Common from Nebraska to Rocky Mountains to Alberta, south to Texas. Poisonous.

Solanum tuberosum L. Potato.

At certain times the tubers of the potato are poisonous, especially when green. The writer knows of an instance where the eating of potatoes acted as a poison. The substances produced in the young shoots of the potatoes are solanin and solanadin.

Solanum carolinense L. Horse-nettle.

Southern United States North to Minnesota and East to New York. Fruit with disagreeable odor. Said to be poisonous. Narcotic.

Solanum Dulcamara L. Bittersweet.

The berries are poisonous, as are also the leaves. Cattle are known to have been poisoned by it. The bitter substance contained in it is known as dulcamarin.

Lycopersicum esculentum Mill. Tomato.

Jimson Weed. (After Halsted)

The green parts of the plant contain saponin and solanin.

Physalis heterophylla Nees. Ground Cherry.

From New Brunswick southward and westward. Suspected plant as is *P. virginiana* Mill.

Cestrum cauliflorum Jacq. Cestrum.

West Indies. The *C. nocturnum* L., frequently cultivated in greenhouses. Several species are poisonous.

Atropa Belladonna L. Belladonna, Deadly Nightshade.

Occasionally cultivated and possibly also an escape. Very poisonous.

Nicotiana Tabacum L. Tobacco.

Cultivated. Narcotic and poisonous, and produces the alkaloid nicotin, a very poisonous substance.

Nicotiana alatum Link & Otto. Flowering Tobacco.

Poisonous like the preceding.

Nicotiana glauca R. Grab. Smooth Tobacco.

An escape from cultivation in California. According to Dr. G. Burtt Davy poisonous.

Hyoscyamus niger L. Black Henbane.

Probably occasionally found in Iowa, Utah, Mont., Atlantic States. Known to be poisonous to stock and also to hogs. Universally recognized as a poisonous plant in Europe and this country. Probably one of the most deadly poisonous plants in the United States. Seeds are poisonous to chickens. Contains the alkaloid hyoscyamin.

Datura Stramonium L. Jimson-weed.

Naturalized in various parts of the U. S. All parts of the plant are narcotic and poisonous, especially the seed. Several cases of poisoning in children are reported in Iowa. The plant produces a very disagreeable odor, and the hay containing the plant is poisonous to cattle. It contains the alkaloid, atropin, and hyoscyamin.

Datura Tatula L. Purple Jimson-weed.

Poisonous like the preceding, and the following species. Naturalized in U. S.

Datura Wrightii, DC. Wright's Datura.

Frequently cultivated as an ornamental plant and known to be poisonous. The nectar from the flowers which is produced in great abundance is known to produce poisoning in children in this country.

Datura alba Nees. Thorn Apple.

Cultivated. Plant said to have been much used in India for criminal purposes. Very toxic.

Capsicum annuum L. Red or Cayenne Peppers.

Well known remedy used as a stimulating plaster externally; if the pepper is applied long enough it produces vesicles. Red pepper is often injurious when taken in too large doses internally. The active poison is capsicol with a strong odor and burning taste.

Capsicum frutescens L. Shrubby Pepper.

Southwestern United States, said to be poisonous. The fruit has a sharp pungent taste.

Lycium halimifolium Mill. Matrimony Vine.

Cultivated and commonly naturalized. Somewhat spiny thorns. Said to be poisonous.

Scrophulariaceae. Figwort Family.

Verbascum Thapsus L. Moth Mullein.

Naturalized on the Atlantic coast and common in Utah and the west. Said to be poisonous.

Linaria vulgaris Hill. Butter-and-eggs,, Toad Flax.

A weed, especially northward. Suspected of being poisonous.

Scrophularia marilandica Gray. Simpson Honey Plant.

Widely distributed in E. U. S., pastures and woods. Not eaten by stock. According to Millspaugh, the physiological effect of this plant is bleeding of the gums, colic, and sleepiness. Contains a crystalline bitter substance, scrophularin.

Digitalis purpurea L. Purple Foxglove.

This plant is widely cultivated in the U. S., and naturalized on the Pacific Coast.

Is poisonous to man and live stock, especially horses. It contains the glucosides digitalin, which dilates the pupil; digitoxin, and digitonin.

Gerardia tenuifolia Vahl. Slender Gerardia.

Said to be poisonous to sheep and calves. Probably other species are likewise poisonous, like *G. grandiflora* and *G. purpurea.*

Pedicularis lanceolata Mx. Lousewort.

Widely distributed in low grounds and swamps. Said to be poisonous.

Pedicularis canadensis L. Lousewort.

Widely distributed in the state in gravelly soils and on knolls. Said to be poisonous. Sheep, however, eat large quantities of the P. groenlandica without apparent injuries.

Pedicularis groenlandica Retz. Mountain Lousewort.

Common at higher altitudes in swamps. Suspected of being poisonous.

Melampyrum lineare Lam. Cow Wheat.

Common in open woods eastern states to Tennessee. The European species, *M. silvaticum,* is regarded as poisonous in Europe. Our American plant has not, however, been reported.

Gratiola sp. Hedge Hyssop.

The European *G. officinalis* is said to be poisonous to cattle. Several species common in eastern North America.

Veronica virginica L.. Culver's Root.

Common in swamps in eastern North America especially northward. Contains saponin.

Chelone glabra L. Balmony.

In swamps northern United States. Contains an alkaloid. Suspicious.

Bignoniaceae. Bignonia Family.

Catalpa speciosa Warder. Hardy Catalpa.

Widely cultivated in U. S. Odor coming from the fragrant flowers is poisonous and Dr. White in his Dermatitis Venenata states that the flowers are irritating to many persons. Dr. Millspaugh, on the other hand, states that it is said to be dangerous to inhale the odor of the flowers for a long time, which, however, is probably not generally true. The allied Caroba contains the bitter principle carobin.

Catalpa bignonioides Walt. Common Catalpa.

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Campanulaceae. Bluebell Family.

Lobelia cardinalis L. Cardinal-flower.

This is listed as one of the poisonous plants by Dr. Schaffner. Cardinal-flower is very abundant in the swamps along river courses in Eastern Iowa, and N. Miss. Valley.

Lobelia siphilitica L. Blue Lobelia.

Also listed as a poisonous plant by Dr. Schaffner.

Lobelia spicata Lam. Pale Spiked Lobelia.

Everywhere on prairies of the northern states. Probably poisonous like the preceding.

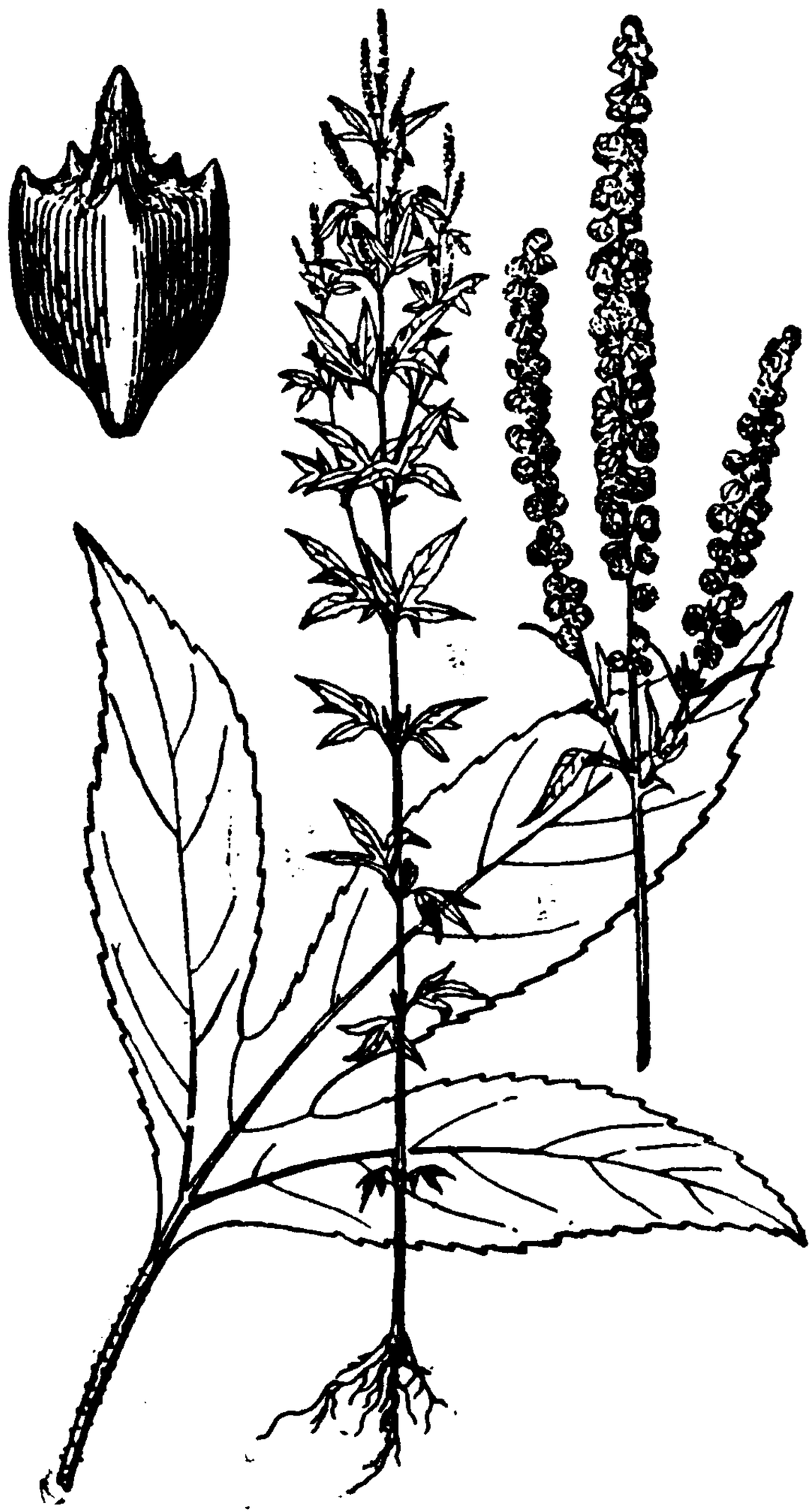

Fig. 19j. Greater Ragweed (*Ambrosia trifida*). Said to cause hay fever. (Dewey U. S. Dept. Agrl.).

Lobelia inflata L. Indian Tobacco.

Widely distributed, occurring in woods. The leaves of this plant were used by the Indians. The plant has long been used in medicine. L. inflata is very poisonous and is used for its action upon the pneumogastric nerve; the toxic doses produce exhaustion and dilation of the pupils. Death is usually preceded by insensibility and convulsions. Contains the acrid lobeliin.

Lobelia Kalmii L. Kalm's Lobelia.

Found in swamps from Nfd. to N. J. west to northern Iowa, Minnesota, and Man. Probably poisonous like the preceding.

Compositae. Composite Family.

Lactuca Scariola L. Prickly Lettuce.

Common across the continent, also the var. *integra.* Said to be poisonous.

Cichorium Intybus L. Chicory.

It has become widely naturalized in the north and west. When fed in large quantities to dairy cattle it imparts a bitter flavor to the milk and butter. It contains the bitter glucoside chicorin.

Iva xanthiifolia Nutt. Marsh Elder. Half-breed Weed.

Western Wisconsin, Red River Valley, south and west to the Rocky Mountains. Said to cause hay fever.

Ambrosia artemisiaefolia L. Common Ragweed.

The pollen of this plant is suspected of causing hay-fever.

Fig. 19k. Spiny Clotbur (*Xanthium spinosum*). Injurious in a mechanical way. Fig. 19l. Prickly Lettuce (*Lactuca Scariola*). Said to be poisonous. (Bentham).

Ambrosia trifida L. Great Ragweed.

The pollen of this species is said to produce an irritating action upon the mucous membrane.

Xanthium spinosum L. Clotbur.

Maine to Texas. It causes mechanical injuries.

Xanthium canadense Mill. Cocklebur.

Young seedlings of this plant are poisonous to horses. Several cases of poisoning to hogs have been reported in this state.

Xanthium strumarium L. Cocklebur.

Poisonous like the preceding. This species is not common in the state. Contains the glucoside xanthostrumarin.

Eupatorium perfoliatum L. Boneset.

Commonly found in low grounds and marshes. It is an emetic when given in large doses.

Eupatorium urticaefolium Reichard. White Snake-root.

Widely distributed in woods in this state. It is said to produce a disease known as milk fever. No reports of this kind of poisoning have come to us in Iowa. The E. cannabinum contains the alkaloid eupatorin and the glucoside eupatorin.

Trilisa odoratissima (Walt.) Cass.

It is said to be injurious and has the odor of sweet clover.

Grindelia squarrosa (Pursh) Dunal. Tar Weed.

From Wisconsin, Iowa, westward and northward. Is said to be injurious. The *G. lanceolata* Walt., occurs from Tennessee to Texas. It has large heads and resinous viscid leaves.

Xanthisma texana D. C.

It is said to be poisonous and contains saponin. Southward.

Erigeron canadensis L. Horse Weed.

It is a widely distributed troublesome weed in the north. The physiological action of the drug obtained from this plant is to produce smarting of the eyes, soreness of the throat, and prostration.

Erigeron ramosus (Walt.) B. S. P. White Weed.

Common in meadows westward. The *E. annuus* of the same distribution is found also in meadows. Both are regarded with suspicion.

Baccharis halimifolia L. Salt Groundsel.

The European *B. cordifolia* is said to be poisonous. It occurs in salt marshes along the Atlantic sea coast.

Solidago canadensis L. Golden-rod.

It is widely distributed in the U. S., and is one of the most common of our golden-rods. The golden-rods are generally regarded as harmless plants, but in a few cases they are suspected of being poisonous. A disease of horses near Black River Falls, Wisconsin, was attributed to a golden-rod. Chesnut thinks the disease due to a rust on the plant. As a general thing stock does not relish the golden-rod.

Madia glomerata Hooker. Tarweed.

A glandular viscid, heavily scented herb common from Saskatchewan to Colorado, Utah, Oregon and Washington. Probably poisonous or at any rate it is avoided by cattle.

Hemizonia macradenia DC. Tarweed.

Common on the Pacific Coast from San Francisco southward. A strongly and unpleasantly scented herb avoided by stock. Many of the species of the genus occur in California and most of them are strongly scented.

Aster Parryi Gray. Woody Aster.

Common in the Rocky Mountains, Utah and Wyoming. Thousands of sheep in Wyoming where this plant occurs suffer with the disease which has been attributed to "grub in the head." Healthy sheep often die within a very short time after the first symptoms appear. This plant is affected with a fungus, *Puccinia xylorrhizae*, and Prof. Aven Nelson suggests in his account of this disease, "The chances are rather better that the suggested poisonous qualities are due to the fungus. Some other parasitic fungi have been proven poisonous

Common in the south and said to be fatal to horses and mules. It imparts its bitter flavor to milk.

Helenium Hoopesii A. Gray. Mountain Sneezeweed.

Common in the Rocky, the Uintah and Wasatch Mountains. Said to be injurious to sheep.

Achillea millefolium L. Yarrow.

It is used as a forage plant, but causes an irritating sensation of the membranes and much pain in the gastric and abdominal regions. It contains the glucoside achillein, an amorphous bitter substance, and the alkaloid moschatin.

Anthemis Cotula L. Mayweed.

Has a very disagreeable odor and causes blistering of the skin. The plant is carefully avoided by stock.

Anthemis arvensis L. Corn chamomile.

Occasionally escaped from cultivation. Seeds of this and other species contain HCN.

Dysodia chrysanthemoides Lag. Fetid Marigold.

Common in the west, Dak., Ia., Neb. to Mo., Tex. The leaf bracts and other parts of the plant are provided with large pellucid glands which produce the characteristic odor of the plant.

Tanacetum vulgare L. Common Tansy.

Introduced into many parts of the north. Many serious and a few fatal cases of poisoning are recorded by the use of tansy oil. The symptoms of poisoning are varied, convulsions, violent spasms, dilation of the pupils, frequent and feeble pulse. Eleven drachms of the oil in a girl produced death in three and one-half hours. The effect on animals is salivation, vomiting, dilation of the pupils, muscular twitchings, followed by chronic spasms, death appears to be caused by paralysis of the heart and lungs.

Artemisia biennis Willd. Biennial Wormwood.

Probably poisonous.

Artemisia tridentata Nutt.

Sage brush used as forage by sheep.

Artemisia Absinthium L. Common Wormwood.

Occasionally cultivated. The volatile oil of the plant is a violent, narcotic poison, and contains the glucoside absinthiin, the alkaloid abrotin, the bitter principle santonin.

Other species probably also poisonous.

Arnica cordifolia Hook. Arnica.

This species with yellow flowers is common in the Rocky Mountains. The bruised leaves give off the odor of arnica. The European *A. montana* is suspected.

Senecio Jacobaea L. Staggerwort.

Occasionally found in the east and causes the Pictou disease. The Squaw Weed (*S. aureus*) is common in the north. The *S. plattensis*, common in western Iowa to Montana and east to Ontario, has been associated by Dr. Day with the Missouri Bottom disease. The species are numerous in the Rocky Mountains and may be responsible for some diseases. It is to be noted that the *S. guadalensis* of Mexico is fatal to stock. The exotic *S. toluccanus* contains an alkaloid with tetanus like action.

Fig. 190. Absinthium (*Artemisia absinthium*). Plant, leaves, and flowering branch. (Faguet).

Arctium Lappa L. Burdock.

Produces itching. Contains the alkaloid lappine. Common weed in the U. S.

Cirsium lanceolatum (L.) Hill. Bull Thistle.

Commonly naturalized in the north from the Atlantic to the Pacific. It acts injuriously in a mechanical way. Other species like *C. Nelsoni*, Canada Thistle (*C. arvense*), *C. scariosum*, etc., all act in a similar way. Some species contain HCN.

Centaurea solstitialis L. Knapp Weed.

Common in alfalfa meadows westward. Acts injuriously in a mechanical way. Some species, according to Greshoff, contains HCN.

Fig. 19p. Sneezeweed (*Helenium tenuifolium*). Poisonous. Fig. 19q. Bull Thistle. (*Cirsium lanceolatum*). Mechanical injuries.

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and ptomains react in a similar manner. The volatile alkaloids, represented by nicotin and coniin, possess disagreeable odors suggesting in part that of ammonia. The solid alkaloids; represented by strychnin, morphin, are odorless, but have a bitter taste, often very characteristically bitter.

Physiologically, the alkaloid is quite generally a very active intoxicant, acting directly on the nervous tissues and producing results all the way from the atonic to the strongly tonic effects. The extreme effects are seen in the muscular excitation of strychnin and the depressing action of morphin, or, in the heart stimulation of atropin, and the depressing effects of cocain.

OCCURENCE OF THE ALKALOIDS.

The alkaloids are peculiarly a plant product and probably may be regarded as a protective agency to preserve a given species of plants. They are deposited in various parts of the plant but commonly in the seeds. The seeds containing, as they do, the vital parts of annual plants, (and also perennials) are protected from destruction by micro-organisms and by animals using them for food, by the intoxicating property, just referred to. A similar case is that of the glucoside amygdalin, as found in the seeds of several of the Rosaceae. The enzyme present in the seed, under proper conditions of temperature and moisture, decomposes it into glucose benzaldehyde and hydrogen cyanid or prussic acid, this latter compound acting as the intoxicant.

The alkaloids are not widely distributed in the plant world, although they are found in several orders or families of plants. Three families are especially characterized by the presence of alkaloids, namely, the poppy family or Papaveraceae, night shade family, Solanaceae, and the Rubiaceae.

The basic property of the alkaloids suggests the probability of their occurrence in combination with acidic compounds more or less characteristic of the plants in which they are found. In some cases the so-called alkaloid appears to be similar to glucoside, i. e. it can be hydrolyzed. For example, cocain can be hydrolyzed into ecgonin, $C_9H_{15}NO_3$, benzoic acid, $C_6H_5CO_2H$ and methyl alcohol. Others are real glucosides like digitalin and solanin. It is undoubtedly true that the latter two should not be classed with true alkaloids, but with the glucosides, and are like caffien theobromin in this sense, i. e., that they are substances that have been classed with the alkaloids on superficial grounds, such as bitter taste, but really have no close chemical relation with them. Caffein and theobromin are now known to belong with the purin compounds. The purin compounds are basic, and hence their classification with the alkaloids.

The acids with which the alkaloids are often united are somewhat common in plants, or in a few cases they are characteristically found in combination with the given alkaloid, e. g., meconic acid in combination with morphin in opium, or aconitic acid united with aconitin. Other acids form compounds with the alkaloids in the various plants in which they are found. Among these acids are tannic, citric, malic and quinic. The combinations are easily broken up by strong bases, like potassium or sodium hydroxid, and in this way they may be separated from the acids. Since the true alkaloids are generally soluble in water while alkali salts of the acids are not soluble, the alkaloids may be separated by treatment with potassium or sodium hydroxid and filtering out the insoluble alkaloid. They are then further purified by the formation of the soluble salt and re-precipitating the alkaloid by an alkali. The alkaloid is then dissolved by the appropriate solvent and crystallized. Again it is interesting

to note that an alkaloid rarely exists alone in a given plant, but is accompanied by several others. For example, aconitin, as extracted from the roots of the aconite plant, Aconitum napellus, contains nine alkaloids; in the extract from poppies, called opium, upwards of seventeen alkaloids have been separated and studied; in the so-called chinchona or Peruvian bark extracts, some thirty-three distinct alkaloids have been isolated; strychnin is accompanied by brucin in Strychnos ignatii, or Saint Ignatius bean.

CLASSIFICATION OF THE ALKALOIDS.

Owing to the basic character of the alkaloids, and the fact that they always contain nitrogen, it was suggested that they were connectel with ammonia in some manner, and if so, that they might be readily broken down by distilling them with potassium hydroxid or caustic potash. They were regarded as derived or at least connected with ammonia. Hoffman, who added much to our knowledge of the amin compounds, considered that they were of the ammonia type and were tertiary amins. In attempting to find some reaction characteristic of the amins, Gerhardt and others, heated the alkaloids with caustic potash, but were unsuccessful in obtaining any results that showed that their basic character was due to this structural cause, although some of them do possess some properties resembling the amins. Others more closely resemble the ammonium compounds. Products obtained by heating some of the alkaloids with potassium hydroxid and distilling the volatile products were found to be the same as were obtained from the destructive distillation of bones. Later, 1834, a study of bone oil by Runge led to the separation of a pure compound which was shown to have the formula C_5H_5N, known as pyridin. This was later shown to be a cyclic (hetero-cyclic) compound, like benzene, one group "CH" being substituted by nitrogen, i. e., trivalent nitrogen. A number of alkaloids have been shown to be constructed on this nucleus by substituting various hydrocarbon groups and are known as the pyridin alkaloids.

Among the pyridin alkaloids and derivatives from them are nicotin, coniin, atropin, cocain. The study of the alkaloids by noting the action of potassium hydroxid proved to be a fruitful one. In 1842, Gerhardt obtained a compound from the destructive distillation of quinine that was a new substance. This was named quinolin because of its orign from this alkaloid. It was later shown to be structurally composed of a benzene and a pyridin nucleus joined by two atoms of carbon in common; the further complexity is due to substituting in this nucleus. Most of the alkaloids are esters and are consequently quite readily separated into the two parts of such compounds namely the acid and basic parts. A study of these constituent parts gives the complete facts as to the structure of the original compound. The esters may be decomposed by acids, alkalis, and water.

The nucleus quinolin has been found in quinin, cinchonin, cinchonidin, strychnin, and brucin, and hence these compounds are known as the quinolin alkaloids.

THE ISO-QUINOLIN GROUP.

This group of alkaloids have a nucleus isomeric with quinolin known as the quinolin group. Like the quinolin group, iso-quinolin has the empirical formula, C_9H_7N. The difference between this base and quinolin, so far as structural constitution is concerned, appears in the position of the atom of nitrogen. Not

only is the fundamental nucleus different in the two classes of alkaloids, but groups that enter into these nuclei are different, thus leading to a large number of possible compounds. To this group of alkaloids belong especially the alkaloids of opium. For example, morphin, thebain, narcotin, narcein, papaverin, and codein. There are also hydrastin, hydrastium and berberin.

THE PURIN GROUP OF ALKALOIDS.

These compounds are not properly included with the alkaloids but they belong with the purin compounds or xanthin bases. The nucleus (purin) or atomic framework, shown by Fischer to characterize the xanthin bases, such as uric acid, the xanthin derivatives, guanin and adenin, is found in caffein, theobromin, theo-phyllin, compounds that are yet classed with the alkaloids. The structural and mutual relationship of these to each other and to the xanthin bases has been determined synthetically and hence their classification is not a doubtful question. The alkaloids are methyl xanthins. Caffein is a tri-methyl xanthin and theo-bromin and theo-phyllin are di-methyl xanthins, the latter being an isomer of theo-bromin. The alkaloid of tea is sometimes called thein although its identity with the caffein of coffee has been recognized for a long time.

There are several alkaloids whose structural relations have not been determined. Among these are pilocarpin, colchicin, and physostigmin. For the description of these alkaloids see statement in this text under their appropriate heads.

PHYSIOLOGICAL ACTION OF ALKALOIDS AS DETERMINED BY THEIR STRUCTURAL COMPOSITION.

It is within comparatively recent time that pure alkaloids have been prepared and consequently that their physiological effects could be determined. Some of these like quinin were prepared in a fairly pure condition during the first half of the last century but most of them belong to a later period. Attention has been called to the fact that a single alkaloid is rarely produced by the plant and hence in the usual extracts from the plant there are several alkaloids with varying physiological effects. These constitutents vary in amount according to a variety of conditions under which the plant may produce them. Acordingly the extracts of such plants will vary in the proportion of the alkaloid present. For example, in cinchona bark the amount of quinin may vary from 2 to 13 percent. The physiological effect of the cinchona extracts will be markedly different in such extremes of composition, i. e. in reference to the alkaloid quinin.

In 1869, Crum, Brown, and Frazer called attention to the relation of the structure of organic compounds to their physiological effects. They were studying the comparative action of strychnin and brucin and although their knowledge of the structure of these two compounds was not complete yet they were able to trace a relation in this case. They found that the presence of methyl or ethyl groups strongly affected the active properties of these compounds. For example, brucin is regarded as the dimethoxy-deriative of strychnin. The discovery that most alkaloids are built about three nuclei, namely pyridin, quinolin, and iso-quinolin added new zest to the study of the physiological effects of certain organic groups when substituted in organic compounds.

Quinolin is a strong antipyretic and antiseptic but produces other results

Fourth group, alkaloids aconitin, atisin, and Japaconitin of the aconite group, obtained from several species of Aconitum, as *Aconitum Napellus, A. heterophyllum,* etc.

Fifth group, the mydriatic group of alkaloids. The alkaloids of this group are atropin, obtained from *Atropa, Belladonna,* and *Datura;* hyoscyamin obtained from *Datura, Hyoscyamus, Scopolia, carniolica,* and *Duboisia* (Hyoscin pseudohyoscyamin being also obtained from these plants); scopolamin from some of the same plants as the preceding; solanin which is, however, regarded as a nitrogenized glucoside, obtained from various species of Solanum; solanidin with stronger basic properties than solanin, obtained from plants of the same family; cytisin obtained from Laburnum (*Cytisus Laburnum*) of the family *Leguminosae,* found also in quite a number of other plants of the same family.

Sixth group, the Veratrum alkaloids containing the alkaloids jervin, pseudojervin cevadin, etc., obtained from various species of *Veratrum* as *V. album, V. viride,* etc.

Seventh group physostigmin, the most important alkaloid of the group derived from the Calabar Bean (*Physostigma venenosum*), and calabarin.

Eighth group, containing pilocarpin, obtained from the leaves of jaborandi (*Pilocarphus pennatifolius*) and four other alkaloids, jaborin, pilocarpin, isopilocarpin, pilocarpin. Jaborandi belongs to the family *Rutaceae.*

Ninth group, taxin, obtained from the yew tree (*Taxus baccata*).

Tenth group, the curare alkaloids which are obtained from the Curare plants (*Strychnos toxifera* and *S. Castelnaei* of the family *Loganiaceae.* The alkaloids are tubo-curarin, curin, etc. Protocurin obtained from the latter species is a slightly toxic substance.

Eleventh group, colchicin alkaloid; this alkaloid is obtained from the seeds and roots of the common meadow-saffron or Colchicum (*Colchicum autumnale*).

Twelfth group, muscarin from the *Amanita Muscaria* or Fly agaric.

GLUCOSIDES.

The glucosides widely distributed in plants are compounds of glucose and organic acids and are certainly of great importance in connection with the poisonous principles found in plants. They have been grouped by Blyth into: A first group the *digitalis group,* consisting of digitalin, digitonin, and digitogenin, all found in the common fox glove (*Digitalis purpurea*); (2) second group of glucosides acting on the heart and containing antiarin obtainel from *Antiaris toxicaria,* the upas tree; the helleborin and helleboretin found in *Helleborus niger, H. viridis,, H. foetidus* and euonymin, a resinous substance found in Wahoo (*Euonymus atropurpureus*) which is a powerful heart poison; the third group containing thevetin, obtained from *Thevetia neriifolia;* Strophantin from *Strophanthus hispidus* of the Dogbane Family belong to this group of heart poisons, but it is not a glucoside and only partly crystallizable; scillain from squill; adonidin from the root of *Adonis vernalis* of the Crowfoot Family; oleandrin from the Oleander; neriin also from the Oleander, sometimes called the Oleander digitalin and the poison of the Madagascar Ordeal plant (*Tanghinia venenifera*). The fourth group contains the digitalin-like apocynin from the common Dogbane, and other Apocynums; erythrophlein, convallamarin, a glucoside from the Lily-of-the-valley; coronillin from *Coronilla*; and cheiranthin from *Cheranthus.* These behave like the Digitalins.

GLUCO-ALKALOIDS.

The gluco-alkaloids represent a class of compounds intermediate in connection between the alkaloids and glucosides. The achillein found in Yarrow (*Achillea millefolium*) and solanin in various species of Solanum should be mentioned. The latter substance has, however, been referred to in connection with the alkaloids.

SAPONINS.

The Saponins have been treated fully in another connection. They are poisonous and when dissolved in water form solutions which froth. Of these mention may be made of saponin and senegin.

OTHER VEGETABLE POISONS.

A third division of poisonous substances includes those which cannot be readily classified and under this head is santonin, a lactone found in the heads of *Artemisia sp.* A second division of this group is mezereon obtained from *Daphne Mezercum.* A third group is ergot of rye, containing ecbolin, secalintoxin, and other substances referred to at length in another connection.

PICROTOXIN, CICUTOXIN AND TOXINS.

Picrotoxin is the active principle of the Indian Berry (*Cocculus indicus* or *Menispermum cocculus*), which contains the active principle picrotoxinin, picrotin and menispermin.

Tutin, a non-nitrogenous glucoside, is obtained from *Coriaria sarmentosa* and other species. Another poison belonging to the picrotoxin class has been isolated from the Japanese *Illicium anisatum*, a member of the Magnolia Family. The plant is sometimes called the Japanese Star Anise. To this group may also be added cicutoxin obtained from the Cowbane, the oil of savin obtained from the common savin (*Juniperus sabina*); croton oil expressed from the seed of *Croton tiglium*; the toxalbumins of castor oil seed and of Abrus; ictrogen from various species of lupines (*Lupinus luteus, L. hirsutus*, etc.); the toxic substances in the cotton seeds; toxic substances in various species of Lathyrus; the toxic substances in Arum; in the black bryony (*Tamus communis*); the toxabumin of the black locust; and the poisonous substances of the male shield fern.

ANIMAL AND PLANT TOXINS.

Another group of poisonous substances is included under the head of ptomaines and animal toxins. The word ptomaine is used in a rather indefinite way, and is open to objection, but the classification given by Blyth as animal toxin is also objectionable. Many of these toxins are the products of bacteria, some of these poisons are, however, the products of higher plants, (as toxin of the black locust, *Abrus*), etc. The groups given by Blyth are the Amines, under which head we have methylamin, found in the cultures of the Comma bacillus, and the trimethylamin, non-toxic, found in a variety of putrefying substances.

AMINS.

The *Amins* are basic and originate from ammonia; they include the *diamins*, belonging to the amin series, which are formed in putrefactive substances. Of these we have neuridin in putrefying substances; cadaverin, found

in cultures of Spirillum and putrid animal matter; mydalein, guanidin. The cholin group includes neuridin, betain, and muscarin. The muscarin has been referred to elsewhere. The neurin is intensely poisonous, and atropin is an antidote to neurin. Tetanin produces tetanus. Tetanotoxin from tetanus produces tremor paralysis and violent convulsions. Mydatoxin contained in putrid horse flesh is poisonous in large doses, causing lachrymation, diarrhoea, and convulsions. Tyrotoxicon, isolated by Vaughan from milk, is toxic. The suso-toxin isolated from hog cholera is said to be quite toxic.

ORGANIC ACIDS.

The last group of poisonous substances includes the organic acids, the most important of which is oxalic acid. This is widely distributed both in the free state and in combination with lime soda and potash in the vegetable kingdom. It occurs in some species of the geranium, spinach, *Phytolacca decandra,* pie plant, *Rumex Acetosa* and in *Atropa Belladonna,* in connection with potash. In Russian Thistle and *Salicornia* it occurs in combination with sodium. In clover, apple twigs, begonia, and many other plants it occurs in the form of so-called compound aggregate crystals, or rosettes of calcium oxalate, in the onion and some other plants of simple crystals. In aroids and Virginia Creeper it appears in the form of needle shaped crystals, known as raphides, which are formed during the metabolism of the plant, the oxalic acid being set free and uniting with the lime in the plant to form calcium oxalate.

Oxalic acid is commonly used by dyers and calico-printers and also by curriers and harness makers for cleaning leather, to remove iron stains, to bleach straw, etc.

Several cases of poisoning have been attributed to the use of plants (like the sheep sorrel, oxalis, etc.), that contain large amounts of oxalates.

Quite a number of cases of poisoning from this acid are reported, especially in Europe. The smallest dose of oxalic acid known to have destroyed life, according to Dr. Taylor, is 60 grains. Oxalic acid acts upon the central nervous system. There is temporary loss of voice, burning in the throat, burning in the stomach, vomiting, especially bloody matter, pulse weak, locally it acts on the mucous tissues.