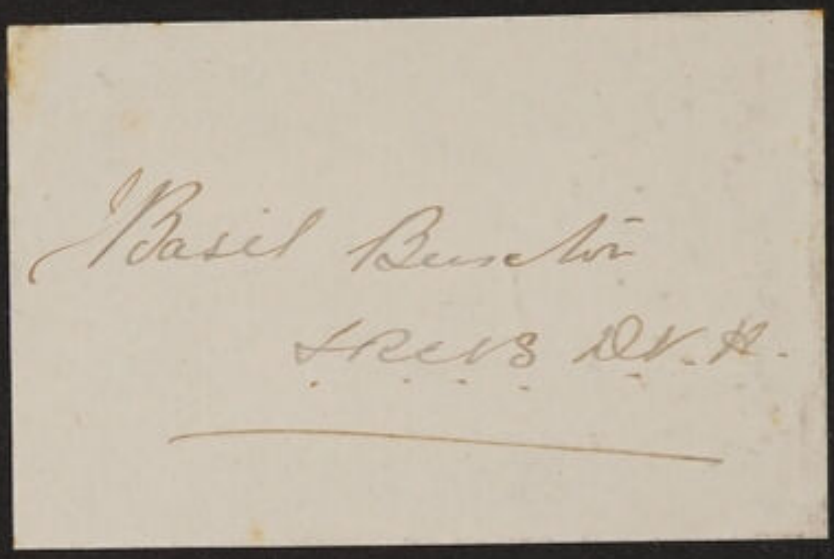
Veterinary toxicology / by G.D. Lander.



VETERINARY TOXICOLOGY

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BY

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PREFACE

Apart from Colonel Nunn's work, there is no special textbook of veterinary toxicology in English. The suggestion of the need of some such comprehensive work has reached me from several quarters, and many offers of help have encouraged me to compile the present volume. I have endeavoured to base the accounts of each poison on records published in the veterinary literature or encountered in my own practice. Failing them, I have had recourse to the standard textbooks and the experience of veterinary friends.

The list of my indebtedness is therefore heavy. I acknowledge, with cordial pleasure, Mr. Wallis Hoare's perusal of my manuscript, and the many valuable original observations of a practised clinician arising therefrom. Major-General Smith has responded freely and promptly with his advice, and has drawn my attention to recondite sources of knowledge, particularly as regards the East. He has, moreover, freely placed at my disposal the excellent plant illustrations in his work on hygiene. To Professor Woodruff I am indebted for a critical perusal of the Introduction, and for the whole of the section on treatment. Professor Macqueen's help has been invaluable. He placed his library and his unique knowledge of the literature at my disposal, and also gave me the benefit of his experience and of his acute critical faculty.

I am under obligation also to all those gentlemen who, from time to time during the last nine years, have brought

under my notice cases of poisoning, upon which the bulk of the analytical data of the text is based.

As regards the botanical parts of the work, I have had the advantage of the expert help of Mr. E. M. Holmes, F.L.S., of the Pharmaceutical Society, who perused all the plant descriptions given.

The abstraction of original papers in the veterinary literature was performed by my wife. Had it not been for this assistance, involving many hours of close and uninterrupted work, the preparation of the text would scarcely have been possible in a reasonable period of time.

Free use has been made of the textbooks, and I am particularly indebted to Finlay Dun, 'Veterinary Medicines'; Winslow, 'Veterinary Materia Medica'; Gamgee, 'Veterinarian's Vade-Mecum'; Nunn, 'Toxicology'; Cushny, 'Pharmacology'; Kaufmann, 'Traité de Matière Médicale'; Cornevin, 'Des Plantes Vénénenses'; Gadamer, 'Lehrbuch der chemischen Toxikologie'; Walsh, 'South African Poisonous Plants'; and Bentham and Hooker, 'British Flora,' upon which the botanical descriptions are based.

I hope that egregious blunders will prove to be few in number, and shall welcome *errata*, and especially notes of cases from my veterinary friends both at home and abroad.

G. D. L.

The Royal Veterinary College,
London,
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INTRODUCTION

Toxicology embraces the general study of the origin, properties, and effects upon the animal organism of poisons. In so far as toxicology is concerned with a study of the mechanism of the absorption, action, and elimination of a drug, it is a branch of the science of pharmacology.

The art of toxicology has the practical object of the diagnosis, treatment, post-mortem indications, and chemical detection of a poison.

A complete scientific and practical development of the subject manifestly demands the joint contributions of the botanist, chemist, physiologist, pathologist, and clinician, and in proportion as there is increase in exactness in our knowledge of these subjects, so do toxicology and the wider domain of pharmacology assume greater precision and efficiency.

The nefarious and often lucrative practice of malicious poisoning is as old as man, and a moment's reflection on the state of knowledge of the exact sciences among the ancients and during the Middle Ages—indeed, until the nineteenth century—will satisfy us that poisoning must have been most extensively practised, and enjoyed remarkable immunity from detection and punishment.

The ancients, besides being aware of many poisonous herbs, were acquainted with certain mineral and other poisons. Thus, arsenic was known to the Greeks, and the Egyptian priests knew how to prepare (and use) hydrocyanic acid from the peach kernel. The Dark Ages were in

these, as in most matters, far less well informed. Fiction and romance have invested the malpractices of medieval Italy—of such expert poisoners as the Borgias—with an air of subtlety, suggesting that recondite poisons of extreme violence, now no longer known, were used. There seems, however, little doubt that arsenious oxide was their chief agent. True subtlety and an empirical prevision of profound pathological fact are found rather in the East and among the gipsies, in the use by those peoples of vegetable toxines—*e.g.*, abrine—and dangerous moulds and fungi as poisonous agents.

The foundations of chemical toxicology were laid in the earlier decades of the nineteenth century by the distinguished French physiologist Orfila. He first showed that many poisons (notably arsenic) could be separated and identified in the ingesta and tissues of a poisoned subject, and he also did much towards elucidating the manner of absorption and distribution of a poison in the system.

Since the time of Orfila knowledge has extended, the active principles of very many poisonous plants have been separated and characterised, chemical methods of separation of poisons from plants and from tissues have gained in accuracy, and characteristic tests for many important poisons have reached an extraordinary pitch of delicacy. In illustration it may be remarked that arsenic can be detected in such minute quantities as the five-hundredth of a milligramme—*i.e.*, the three-thousandth of a grain—and recently it has been shown that hydrocyanic acid can be recognised in the same small proportion. Such examples render it abundantly clear that with ordinary care it is impossible to fail to detect these poisons; indeed, the difficulty is often felt in the laboratory that some of our tests are almost too delicate to be diagnostic of poisoning. It very often indeed happens that a substance is found which, on inquiry, proves to have been given as an ordinary and legitimate medicinal dose.

DEFINITION OF A POISON.

A poison may be defined as a substance which, when introduced into the body in relatively small amount, acts deleteriously, and may cause death.

It is at once evident that a hard and fast definition, here as in most things, is almost impossible. Restrictions—many based on scientific grounds, but most on those of common sense—at once suggest themselves. The forcible introduction of a bullet, or a knife-blade, or the mechanical lesions of powdered glass, cause death. Though not scientifically called a poison, the latter substance would be held equivalent in the eyes of the law. The great majority of the poisons are also drugs, and, as Cushny well expresses it, 'some bodies may, in fact, be remedies, foods, or poisons, according to the quantity ingested and the mode of application.'

The following important restrictions and limitations to the foregoing definition are accordingly suggested:

1. A poison in sublethal quantities exercises a specific effect on the organism, interfering with the normal action of some one or more particular group of cells.

This distinction appears necessary, for it is desirable to withdraw from the category of poisons, properly so understood, certain (possibly all) foods. It is distressing to hear that, on the strength of the bad results of injudicious feeding, or over-feeding, certain foodstuffs are given the grave designation of "poisonous." Yet we know that bad dieting, and especially the irrational use of a new food, may earn it an evil, but often undeserved, reputation.

2. A poison differs from a bacterial toxine in that a poison, as here understood, never gives rise on long continual sublethal dosage to the formation of an anti-body in the blood serum. This distinction seems quite sharp, although of course the bacterial toxines—*e.g.*, of diphtheria or tetanus—may be correctly said to be poisonous. Moreover, the phytotoxines (ricine, crotine, abrine) and zootoxines

(snake venoms), though originated by the living cell, and giving rise to anti-bodies, like the toxines of infective diseases, are not of bacterial origin, and are thus classed as poisons.

In practice, by common consent, certain inorganic and organic compounds, and certain plants and plant products, irrespective of any possible therapeutic action, or dietetic value, are held to be poisonous. In most, but unfortunately by no means all, cases of plants, the essential cause or active principle has been separated in a more or less pure form, capable of recognition.

GENERAL CHEMISTRY OF POISONS.

An attempt may be made to give an outline of the various classes of substances falling under the above heading, and responsible for poisoning. But it must clearly be understood that such an outline can have no pretensions to completeness.

1. Inorganic or Mineral Poisons.

(*a*) Certain *elements*—*e.g.*, phosphorus, sulphur.

(*b*) Simple *gases*—*e.g.*, carbon monoxide, oxides of nitrogen, ammonia, sulphuretted hydrogen.

(*c*) *Acids* and *alkalis*.

(*d*) *Metalloids*, or elements, which chemically form the border line between non-metal and metal—*e.g.*, arsenic and antimony compounds.

(*e*) *Metals* and *their salts*—*e.g.*, lead, mercury, copper, barium, zinc, chromium, iron, etc.

2. Organic Poisons. By organic is understood any compound, necessarily containing carbon, other than the oxides of carbon and the carbonates, often of animal or vegetable origin, but often synthetical—*i.e.*, capable of being made from its elements. Apart from artificial drugs this great division includes the active principles of poisonous plants.

(*a*) *Alkaloids*, distinguished by being bases—*i.e.*, forming

salts with acids. A most numerous class represented by, *e.g.*, strychnine, atropine, conine, taxine, etc.

(*b*) *Glucosides*, compounds which, by the action of dilute acids or enzymes, take up the elements of water, and are resolved, giving, along with other compounds, always sugars, *e.g.*, digitalin, helleborin, amygdalin, etc.

(*c*) *Acrid juices or resins*, amorphous, faintly acid, irritant substances, commonly contained in saps—*e.g.*, of euphorbia, and ranunculus.

(*d*) *Indefinite principles*, neutral in chemical respects—*e.g.*, anemonine, cicutoxine, picrotoxine.

(*e*) *Essential oils*—*e.g.*, turpentines (savin), tanacetone (tansy), camphor, mustard oil.

(*f*) *Toxines*—*e.g.*, ricine, abrine, venoms.

(*g*) *Cyanides*, hydrocyanic acid produced in plants from glucosides, but also prepared in other ways, and its salts.

(*h*) *Phenols*, hydroxy derivatives of the aromatic or benzene hydrocarbons, sometimes found in plants—*e.g.*, tannin—but mainly encountered in the tar of coal and wood distillation—*e.g.*, carbolic acid, cresols.

The extraordinary range and variety of chemical types covered by this incomplete scheme serves to accentuate the extreme difficulty of any correlation of chemical nature and physiological or toxic activity. That the effects are due to a chemical reaction between the agent and the cell substances, far from being a pious opinion, seems almost a truism. Of the nature of those reactions we are profoundly ignorant. So far, little has been done beyond the recognition of certain general correlations between activity and composition, thus, for instance :

1. Antimony, closely allied chemically to arsenic, resembles it in its effects on the organism.

2. In general the condition known to chemists as unsaturation (*i.e.*, the possession of unsatisfied or uncombined valencies by a molecule, rendering it able to combine directly with other substances), in comparison with saturation (*i.e.*, when all the valencies are actually in combination), is accompanied by greater physiological activity. In illustra-

tion, arsenic acid is saturated and is less poisonous than arsenious acid, which is unsaturated, and Ehrlich's researches tend to prove that a reduction from the pentavalent arsenic to the trivalent arsenious stage is a necessary precursor to toxicity of the arsenic compounds; the unsaturated hydrocarbon acetylene in distinction to the saturated ethane is poisonous; hydrocyanic acid is more unsaturated and more toxic than the sulphocyanides; the alcoholic compound betaine is far less toxic than the unsaturated and more reactive but similarly constituted aldehyde muscarine.

3. Among organic compounds similarity of type commonly denotes similarity of effects, which, however, grow less as complexity increases in a series of compounds. Thus the alcohols resemble one another, the lower being more active than the higher, and similarly phenols show general likeness to one another. But there are anomalies; *e.g.*, sulphonals containing only ethyl groups lack the hypnotic effect of those containing the analogous methyl group.

CONDITIONS GOVERNING THE ACTION OF POISONS.

A very large number of factors determine the action of a poison in a given case of which the more important are—*Absorption*, *distribution* and *accumulation* in the organs, and *elimination*. Minor factors are the *species*, *age* and *idiosyncracy* of the subject. A manifest condition precedent to all is the administration of a sufficient quantity, the *toxic* or *lethal dose*.

Absorption.—The absorption of a poison depends:—

1. On the physical nature of the poison.
2. On the channel of absorption.

1. Before absorption, which requires the formation of a solution, can occur, it is necessary that the poison be in a soluble and absorbable form when given, or that it becomes modified so as to fulfil these conditions after administration.

The *gaseous state* represents the most easily absorbable of all forms, when the gas enters the lungs. The very rapid action of gases, such as hydrocyanic acid, carbon monoxide, sulphuretted hydrogen, and volatile anæsthetics—*e.g.*, chloroform ether and nitrous oxide—illustrates this point.

Solids insoluble in water, in dilute acids, or in dilute alkalis, in general are not poisonous by the alimentary tract. Thus, the soluble salts of barium are very poisonous, but barium sulphate is insoluble and non-toxic. With lead sulphate the insolubility is not so pronounced, and therefore it has a definite though slow toxic action. Perhaps the best instance is that of arsenious oxide. When the coarsely powdered vitreous substance is freed from impalpable particles, large quantities may be given to dogs without bad effect.

Solutions or *solids soluble* in the body fluids are easily absorbed from the alimentary tract, provided that they are not precipitated—*e.g.*, by acid, or alkali, or albumin—and that they are diffusible by osmosis—*i.e.*, can permeate animal membranes. Thus, a solution of arsenious oxide, or of an alkaloid, is a readily absorbable and diffusible substance, but a dilute solution of silver nitrate is mainly precipitated as the insoluble silver chloride by the hydrochloric acid of gastric juice. The absorption of an alkaloid is retarded by the formation of the insoluble tannate when tannin is employed as a remedial agent, and most of the heavy metal salts give precipitates of insoluble albuminates whereby absorption is retarded. Toxines (except ricine and crotine) are not, or only very slowly, diffusible, and therefore not absorbed by the intact membranes. They are thus not toxic by the alimentary tract, or, like ricine, act far less intensely in this way. Another example of a soluble poison which is not easily absorbed is afforded by the alkaloid curarine. Moreover, the living cell wall is impermeable to certain dissolved substances — thus to the magnesium radicle, or ion, in solution—so that that metal is not absorbed from the stomach or intestines.

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fat are penetrant—such as alcohol, benzene, turpentines, phenols, and alkaline liquids. The latter point is important. An alkali saponifies a fat and emulsifies it, and thus alkaline solutions of phenols, and of arsenic, such as are used in dips, may penetrate and cause danger if the solution is too strong, or if it is left too long in contact. Very little arsenic would be absorbed from a solution of equivalent strength in an acid liquid.

Absorption from the broken skin or from a wound is naturally far more rapid than from the intact surface, and, varying with the nature of the wound, approximates to subcutaneous injection. The difference is well illustrated by Kaufmann's figures for the toxic doses of powdered arsenious oxide for the sheep, which are by the mouth 75 grains, and by application to a wound 3 grains.

(*c*) The *alimentary tract* offers a more suitable mucous surface, external to the body, than the skin, forming a very extensive, moist surface, covered by a delicate epithelium, and is the most usual channel of absorption of a poison. In contradistinction to the stomach and intestine, the membranes of the mouth and gullet are thicker, and swallowed materials do not rest in contact with them for very long, so that absorption by this channel is of less importance than from the stomach and intestines.

Among *carnivores* the stomach offers an acid medium to the ingested materials, thus favouring the solution of substances sparingly soluble in water, but yielding soluble salts with hydrochloric acid. Such are the majority of alkaloids, and many metallic oxides—*e.g.*, those of lead, barium, zinc, and mercury. Absorption takes place freely from the stomach of a carnivore. Thus, Taylor quotes results obtained by giving strychnine wrapped in paper to cats and dogs. In the case of a dog, death resulted after some considerable period—longer than three hours—and $\frac{3}{4}$ of a grain of a dose of 2 grains had been dissolved.

In the *horse* absorption from the stomach is not so rapid or complete as in the carnivores. Thus, Smith* draws

* 'Veterinary Physiology,' 1907 edition, p. 177.

attention to the lack of absorption of strychnine therefrom, after ligature of the pylorus. It cannot, however, be safely laid down that no absorption takes place, for traces of potassium ferrocyanide, given by injection into this viscus after ligature of the pylorus, have been found in the urine.

The first stomachs of the *ruminants* are not adapted to absorption, which is active from the fourth or digestive stomach. Bouley and Colin injected a solution of alcoholic extract of nux vomica in water into the abomasum of a bullock after pyloric ligature, and observed symptoms after five, and death after seven, hours. Craig* gave a goat 10 grains strychnine in water coloured by magenta, and obtained symptoms in twenty minutes, when, on slaughter and post-mortem, coloration extended over the rumen, reticulum, and omasum, and just entered the abomasum.

The small intestine, cæcum, colon, and rectum, all absorb rapidly, and quicker than the stomachs. Thus, strychnine injected into the small intestine may produce its results in a few minutes.

The general condition of the organs has naturally an influence on the rate of absorption. In all cases this is more rapid when the stomach is empty than when it is full. The magnitude of the dose, as well as the nature of the substance, is also clearly an important factor. It is evident that many variable conditions unite in making it impossible to answer the highly important medico-legal question, 'How long after dosage would symptoms set in?' with anything more than very approximate accuracy.

(*d*) *Subcutaneous* or *intravenous* injection is a method of introduction of poisons rarely, if ever, likely to give rise to poisoning, but of immense value in studying the toxic effects of drugs, and in medicinal treatment. The possibility of loss by vomition or otherwise is obviated, and, moreover, the general effects of the drug are more fully disclosed. Absorption in this way is rapid; thus, potassium ferrocyanide

* *Record*, 1911, p. 103.

or iodide injected under the skin of the face of a horse could be found after eight minutes in the urine (Colin).

The Mechanism of Absorption.—The underlying physical principle governing absorption is that of *osmosis*.

By osmosis is understood the passage of a dissolved substance through a membrane, such as parchment or the protoplasmic cell wall of the living cell, or the epithelial layers of the alimentary tract. In general, only relatively simple, soluble, and crystalline substances, such as salts, sugars, and urea, diffuse at all easily through such a membrane. They are called *crystalloids*. Substances which are of greater complexity, which are not crystalline, and which tend to form viscous or mucilaginous solutions, or suspensions in water, do not osmose, or, at any rate, only do so very slowly. Typical of this class are starch, proteins including enzymes, and glue. They are called *colloids*. Amongst inorganic compounds, silicic acid forms a typical colloidal solution. In the light of these facts one understands why proteins and starches must be resolved into simpler crystalloids—peptones and sugars—before absorption, and why toxines—*e.g.*, of ricine, snake venom—are not absorbed, or only very slowly, by the alimentary mucosa.

But the epithelium is not only capable of separating, by the process of osmosis, the crystalloid from the colloid, it is also *semi-permeable*—that is, will allow osmosis of certain substances and not of others. This may be illustrated by reference to salts. In water solution a salt (in part and to an extent dependent on the strength of the solution and temperature) is split up or dissociated into *ions*, which are held to be the carriers of electric charges, and which do not possess the ordinary properties of the atoms or groups of which they are composed. In illustration, sodium chloride splits into ions Na^+ and Cl^- not possessing the ordinary qualities of sodium and chlorine, existing independently of one another, and bearing charges of electricity, positive on the metal, and negative on the acid ion respectively.

Too great emphasis cannot be laid on the essential point

that the ions do not possess the ordinary properties of their components. It is only when the ions are discharged, as in electrolysis, or in the course of a chemical reaction, that the ordinary properties of the element or group are displayed. To suppose, for instance, that the existence of the sulphate ion SO_4^- in a solution of magnesium sulphate in the least degree confers the properties of ordinary sulphuric acid on the solution may prove most misleading.

The existence of SO_4 ions in a solution, together with metal ions, such as those of magnesium, zinc, and sodium, means that we have to do with a salt solution. In ordinary sulphuric acid solution we have whole molecules of H_2SO_4 and ions of SO_4 and hydrogen, all in proportions determined by the concentration. Similarly with other acids, such as nitric and hydrochloric, in solutions of which we have ions of hydrogen, and of NO_3 and chlorine, respectively. The existence of hydrogen ions in a solution means the possession of acid properties. Alkalis, such as caustic potash, KOH, ionise into K ion and OH ion, and similarly with other alkalis, and the existence of alkalinity in a solution is a fact parallel with the existence of OH or hydroxyl ions in it.

The simple physical laws of osmosis, in accordance with which a dissolved crystalloid tends to permeate the membrane, passing from the more to the less concentrated solution, adequately accounts for the absorption of dissolved alkaloids and organic poisons, which do not exercise local chemical reactions. If on the one side we have a solution of strychnine, and on the other a liquid free of that substance, the alkaloid will traverse the membrane. If the solution into which it passes is not removed, equilibrium will be established when the solutions on both sides are of the same strength, but if, as in the digestive system, this solution is continually removed, the osmosis will go on to completion.

The intestinal epithelium is semi-permeable to some salts, and, for example, will not allow of the passage of the magnesium ion. This metal is, therefore, not absorbed from

the alimentary tract, and consequently exercises different effects when given by the mouth than when injected. Similarly, zinc is not at all readily absorbed.

The case of the action of the heavy metal salts is, however, more complex, and attempts have been made to account for their general irritant effect in the light of the theory of ionic dissociation.

When the salts of the heavy metals—*e.g.*, lead, silver, mercury, copper, zinc—come into contact with the proteins of the cells and cell walls, for the most part they form an albuminate of the metal, which is insoluble, or soluble only in excess of albumin.

The general reaction for an albumin of feebly acid character would be, taking the case of a sulphate—

$$\text{Metal, } SO_4 + H_2 \text{ alb.} \rightleftharpoons \text{Metal-albuminate} + 2H^+ + SO_4;$$

i.e., there would be formed metal-albuminate and sulphuric acid, more or less completely ionised according to concentration, the whole being a reversible reaction, since the albuminate is decomposed by excess of acid. The irritant effect is ascribed to the acid thus liberated.

Irritant effects cannot be held to be solely due to the degree of ionic dissociation. Were this alone responsible, the greater the ionization the greater the irritation, in accordance wherewith the alkali nitrates, chlorides, and sulphates ought to be more irritant than the corresponding salts of copper, which is not the case, although ionic dissociation is more complete with the former than the latter.

A possible explanation of some value is to be found in the fact that, with the heavy metal salts as a class, a different type of dissociation occurs. That is *hydrolytic dissociation*, which means that water decomposes the salt into oxide and acid, as may be illustrated by reference to zinc chloride; thus—

$$ZnCl_2 + 2H_2O \rightleftharpoons Zn(OH)_2 + 2HCl.$$

The extent or degree of hydrolytic dissociation is generally small, and the reaction is reversible—*i.e.*, for every

concentration and temperature there will be a given proportionality between the four components of the system. Hydrolytic dissociation of the type shown is indicated by the marked acidity of the solution to litmus and other indicators.

The kinds of salt liable to suffer hydrolytic dissociation are—(1) Salts of weak bases and strong acids showing acid reaction—*e.g.*, heavy metal chlorides, nitrates, and sulphates; (2) salts of strong bases with weak acids, showing alkaline reaction—*e.g.*, carbonates of the alkali metals; (3) salts of weak bases with weak acids—*e.g.*, the organic acid salts of the heavy metals, such as zinc and lead acetates. The reaction of such heavy metal acetates is feebly acid.

It seems not unreasonable to associate irritant effect with hydrolytic dissociation. A solution of zinc chloride contains a fair proportion of free hydrochloric acid, and, similarly, the sulphates contain free sulphuric acid. In support may be adduced the well-known fact that the double alkali chlorides and sulphates—*e.g.*, potassium zinc sulphate, potassium copper sulphate, potassium zinc chloride, are less irritant and are also less hydrolysed than the normal sulphates. On the other hand, mercuric salts are feebly hydrolysed and also feebly ionised, yet they are irritant, or even corrosive, a fact which well illustrates the danger of ascribing to any one property an effect which is jointly due to several factors, the chief of which, in the case of mercury, appears to be the facility with which it forms compounds with proteins.

But considerations of this kind help us to understand why the double salts—the salts of the weak organic acids and the metal albuminates—are more or less devoid of irritant properties.

The formation of albuminates and their degree of solubility in excess of protein, or in salt solution, are very important factors as regulating the penetration of metals into the cells and tissues, and the speed of their absorption into the circulation.

By whatever chemical and physical processes the libera-

tion of free acid from an irritant salt takes place, the modern standpoint of physiology enables one to conceive of the mechanism of its action. Brought into a normally alkaline medium, the acid stimulates the cells to increased alkali secretion in order to counteract the acidity. Similar considerations hold for the introduction of alkali into a normally acid medium. This requires increased circulation of blood and increased cell activity, and whilst tonic when small quantities of acid are concerned, the effect becomes toxic with larger quantities.

Distribution and Accumulation.—It may now be regarded as fully established that all poisons, save those which act by the formation of gross lesions resultant upon the destruction of the cells (strong acids and alkalis), causing death by shock, exercise their effects only after entrance into the general, and in particular the arterial, circulation. In a few cases, such as those of arsenic and hydrocyanic acid, these poisons may actually be detected in the blood after administration. The fact that most poisons cannot so be detected is merely due to the lack of sufficiently refined chemical methods of separation and recognition. Thus, atropine produces its remote mydriatic effect, though it could not be chemically detected in the blood or nervous tissue. For this reason a characteristic physiological effect often constitutes a better test than a chemical reaction.

Before the poison absorbed by the above-mentioned channels can reach the capillary system and cells of the body it must traverse the liver and lungs, which thus play a protective rôle. Sulphuretted hydrogen may be introduced by the rectum, in doses considerably greater than those which prove fatal on respiration. After such administration the blood, arriving at the pulmonary mucosa, and carrying the dissolved gas, abandons it to the exhaled air, in which it may be detected by the smell, and by blackening a paper soaked in lead acetate solution.

The liver arrests most metals, phosphorus, and many alkaloids, so that on arriving thereat in the portal system, after absorption from the alimentary tract, the whole or a

great part, depending on the quantity, may be held back from the blood stream. The activity of the liver in this respect appears to be proportional to its glycogen content, for when this is reduced the liver no longer arrests the drug, and a very much smaller dose than the normal may be toxic. Some substances—*e.g.*, salts of sodium and potassium, alcohol, and digitalis—are not arrested by the liver. Metals are stored in an unknown condition, possibly as albuminates, in the liver, and many unstable poisons are modified; *e.g.*, ammonia is converted into urea, and some alkaloids oxidised, so that the liver does not exercise a merely passive function in its protective capacity. The condition as to health or disease of this organ is thus of importance, since when the glycogenic function is low, little arrest of a poison will occur. It is chiefly in the liver that *accumulation* takes place. Besides metals a few organic poisons, notably digitalin, helleborein, and strychnine, are cumulative, although not necessarily stored in the liver. To designate a poison as cumulative does not endow it with exceptional properties. The only difference between such and ordinary drugs is, that elimination is slower in the former than in the latter, though both may be stored. The difference is of degree, not kind, although important from the practical standpoint. The sudden display of symptoms, after more or less prolonged dosage, means, then, that elimination has not kept pace with absorption, or that absorption has, through some cause become more active, or elimination less so.

Elimination.—Poisons may be divided roughly into those which are slowly (cumulative) and those which are rapidly eliminated. In the first category we have, as the most important examples, the metals, which are arrested chiefly by the liver. From that organ they pass off in part by the blood, but mostly by the bile. From the bile a portion may pass into the fæces, and a portion be reabsorbed and carried back to the liver, thus establishing a gastro-hepatic circulation (Claude Bernard).

Most of the poisons, however, do not accumulate, and

elimination starts immediately after absorption. This is especially true for volatile poisons like hydrocyanic acid, alcohol, and the like, which are eliminated in great proportion by the lungs.

All the excretory channels are concerned in elimination, but the most important in this respect are the kidneys. The milk is a less important channel of elimination. The skin also plays a part, and the beneficial effects of drugs are in many cases attributable to their local excretion—*e.g.*, of balsams through the lungs, and of arsenic through the skin.

The time taken in elimination varies very greatly, is most rapid with volatile agents, and generally is quicker after injection than after ingestion.

An instructive and important example regarding arsenic is given by Taylor. The maximum of arsenic in the liver was attained in fifteen hours after ingestion, viz., 2 grains to 3½ pounds ; after fourteen days the proportion was 0·17 ; and after seventeen days nil (Geoghegan). Valuable as such data are, they must on no account be taken to be general and precise. They only serve to illustrate the relative speed of elimination, which, in a given case, will be modified by such factors as dosage, condition of the subject, and other circumstances.

VARIATIONS OF ACTION DUE TO SPECIES.

Wide differences in reaction towards a given poison are noticed in comparing the various species with one another. In part these variations are explicable on anatomical and physiological grounds, though by no means always. The differences in the digestive apparatus and in the nervous system serve to account for some variations. In ruminants ingested poison is immensely diluted in the rumen, is distributed evenly on rumination, and in general is absorbed more slowly than in the horse or carnivore. This affords greater opportunity for elimination, and

accounts partially for the usually high degree of resistance of the ox and goat.

The relative stage of development of the nervous system greatly affects the dosage and action of many drugs. In illustration, the dog is more susceptible to morphine than the rabbit and less so than man. Morphine acts as an hypnotic on man and the dog, but as an excitant and convulsive on the cat, goat, pig, ox, and horse.

But some variations cannot easily be explained on such considerations. Thus, the ox is considerably more sensitive to mercury and lead than the horse; the rabbit is insensitive to atropine; birds withstand large doses of strychnine.

VARIATIONS OF ACTION DUE TO THE INDIVIDUAL.

Age.—As a rule, young animals are more sensitive than adults, but the young cat is less susceptible to morphine than the old, and young animals are less affected by strychnine than older ones. On the other hand, according to Fröhner, whilst a dog of ten years can resist 1·7 grams of santonine per kilogramme, 0·2 grammes per kilogramme will kill a dog of a few weeks old.

Idiosyncracy.—Subjects apparently similar in all respects often display great diversity in relation to a drug. Thus, strychnine sometimes is dangerous in ordinary medicinal doses to dogs, on account of which, as also by reason of the accumulation due to tardy elimination, dosage of this drug ought to be kept down.

In poisoning, where a large overdose is almost always given, there is not so much room for the display of idiosyncracy as in therapeutic treatment.

Other factors of tolerance and habituation do not play a significant part in toxicology on the same grounds of the large dose commonly given. Habituation is displayed towards nervous poisons in man—*e.g.*, opium, nicotine, alcohol, and probably also towards arsenic, of which the mountaineers of Styria eat relatively large quantities. The

question as regards animals assumes interest in view of the fact that cases of poisoning by the local poisonous plants do not often occur amongst animals bred in a particular locality. But it is almost certain that is due to their avoidance of the dangerous herb. When fresh stock is put on the land, the animals eat indiscriminately, and poisoning occurs.

CLASSIFICATION.

The rational basis of classification is that of physiological action, but unfortunately no satisfactory scheme is available. In many cases the action is complex; thus there may be local effects and general effects, as with ammonia and aconitine. There may be much overlapping as regards the centres acted upon, making it difficult to assign a particular poison to a particular class. The course of poisoning by a plant is always complex. In the majority of cases poisonous plants owe their activity to alkaloids, which act after absorption. At the same time on account of other components (acrid juices, oils, and the like) they exercise irritant effects. The effects of a large poisonous dose are not necessarily similar to those exercised by moderate therapeutic administrations.

A useful, if broad, distinction may be made between—(1) corrosives, (2) irritants, (3) non-irritant nervous poisons.

Corrosives owe their action to their concentration, and are represented by the strong mineral acids and alkalis, phenols, and very concentrated solutions of many salts. A poison acting as a corrosive in a concentrated form may have an entirely different action in a diluted condition. Actual destruction by water abstraction, by decomposition, and solution of fats and proteins in the living cell mark the effect of a corrosive.

Irritants so modify the cell as to disturb its normal course of metabolism, ultimately causing inflammation. The irritant effect is general, not being limited to special cells of the organism, and poisons having a wide range of activity

are the protoplasmic poisons, such as mercuric chloride, phenol, and hydrocyanic acid.

Among irritants are included typically the salts of the heavy metals, but it will be remembered that to an irritant effect may often be added nervous effects, leading to the designation of *narcoto-irritant* applied in practice to so many vegetable poisons. And similarly with the metals there must be distinguished the local irritant effects and the general effects produced after absorption.

Non-irritant nervous poisons may be distinguished according to the centre acted upon. Local effects are not significant, and only after absorption do symptoms set in.

A fairly complete survey of poisons, arranged according to their physiological effects, is that of Rabuteau :

Hæmatic, acting on blood corpuscles : Cyanides, phosphorus, arsenic, alcohol, carbon monoxide, sulphuretted hydrogen.

Hæmatic, acting on plasma : Silver.

Neurotic, paralyso-motor : Curarine, aconitine, conine, cicutoxin.

Neurotic, spinal : Strychnine, cantharidin.

Neurotic, cerebro-spinal : Chloroform, ether, morphine.

Muscular : Solanaceæ, digitalis, veratrine, antimony, potassium, copper, lead, mercury, zinc.*

The chemical classification depends on the most convenient routine followed in an analysis.

For analytical purposes we class poisons as follows :

(*a*) Volatile poisons—*i.e.*, those which may be distilled either from an alkaline or acid solution, comprising phosphorus, hydrocyanic acid, carbolic acid and its allies, essential oils, alcohol, chloroform, ammonia, conine, and nicotine.

(*b*) Metals and metalloids : Lead, mercury, arsenic, antimony, copper, zinc, chromium.

* According to more recent opinion, it is doubtful whether the metals should be included in this section.

(*c*) Fixed, or non-volatile, bases and acids: Caustic alkalis, mineral acids, oxalic acid.

(*d*) Fixed, or non-volatile, organic poisons: The alkaloids and glucosides.

Further details regarding this subject will be given in a later section.

COMMON CAUSES OF POISONING.

It will be advantageous to refer at this point to some of the more usual causes of poisoning, although further details will be given for each particular poison later.

Poison is nearly always conveyed in food or water. Poisoning by subcutaneous injection, though more certain and less easily proved by analysis, is unknown as a malicious practice, and when it happens is the result of an accident in treatment.

Malicious poisoning is generally done by means of poisoned food, and affects chiefly dogs, foxhounds, and foxes. The agent is almost always an accessible poison, such as a rat paste or powder, and in the majority of cases strychnine. It should be remembered as regards foxes, for which a common bait is a poisoned rabbit, that it is no offence to kill a fox, but that the exposure of poison above ground constitutes an offence.

The malicious poisoning of the larger animals is fortunately rare. Of accidental poisoning the ox is the most frequent victim; this no doubt on account of its feeding habits. This animal is an indiscriminate feeder in comparison with the horse and sheep. All our experience tends to the conviction that the greater number of cases of poisoning of cattle are the result either of gross carelessness or culpable ignorance.

Sheep dips either in powder or solution, and weed-killers, are too often left exposed where animals can get them; troughs of solution, or water-carts, are left for weeks overlooked or forgotten. Very often a long and painstaking

search after the event discloses such a cause open to all observers. The same remarks apply to paint, which is eagerly eaten by cattle, and generally contains lead, though green copper arsenite is often used. Paint tins are left in fields. Paint splashes are not removed. Refuse is frequently thrown over the fence. A case of lead poisoning occurred recently due to old tarpaulin, crusted with oil paint, stripped from a roof and thrown into a field.

Bullet splashes have caused lead poisoning, and the impregnation of herbage with lead and other metals on account of smelting and similar operations sometimes leads to chronic poisoning.

Water is less often the vehicle. In spite of the considerable solubility of lead and zinc in rain water, it is not likely that a very large quantity of poison would be taken in this way, so that acute poisoning from this cause is rare. On the other hand, chronic poisoning may happen.

Another prolific source of trouble is the inveterate habit of dosing the animals common to so many attendants and even owners, who might be presumed to possess more judgment. Wholesale administration of condition powders and salts always does more harm than good, and often destroys life.

Vegetable poisons are more likely to be eaten by animals in the spring and early summer, when the tender green food is especially tempting after the winter fodder, or in a dry season, when herbage is scarce (*cf.* the acorn poisoning of 1911). Fortunately many poisonous plants contain less poison in the young than in the mature state, but this rule is not general. For instance, certain cyanide-producing plants are more poisonous in the earlier than the later stages of growth.

Poisonous plants in general display great variation of the proportion of poison. Two conditions of significance both tend to modify the poison content. They are latitude and cultivation.

In higher latitudes plants of the same species are often less poisonous than in lower and warmer situations; for

example, the aconite and cherry laurel. The rule is not absolute; thus rhododendron, veratrum, and the hellebores are flourishing and dangerous in Alpine districts. A scrutiny of the distribution of poisonous plants in Great Britain, however, soon satisfies one that they are less numerous in Scotland than in England, and in the latter country more often found in the south than in the north. Poisonous plants are particularly common in rocky and arid districts, especially near the sea. One is struck by the profusion of *Solanum dulcamara* and *nigrum*, of *hyoscyamus*, and of the horned or sea poppy in the more sandy coast localities of the South of England.

As regards warmer latitudes, poisonous plants are both more numerous and usually more virulent.

As to cultivation, this is well known to lead to a diminution in the generation of poison by a plant. Since the presence of poison is no doubt a measure of natural protection, one understands how this occurs; for Nature never wastes energy, and when the preservation of a species is assured, there is no need to elaborate poison. An excellent instance is the *Phaseolus*, or French bean, which is harmless, whilst the uncultivated variety, *Phaseolus lunatus*, contains a cyanogenetic glucoside. Aconite is another species whose poisonous properties diminish on cultivation.

The hemlocks are often eaten by cattle, and both cattle and horses frequently crop overhanging growths of yew, rhododendron, aconitum, and other plants. The throwing of garden clippings over the fence is a common cause of disaster. Those responsible ought to remember that common garden shrubs and flowering plants comprise many poisonous species, and it would be a safe rule to regard all as dangerous.

KINDS OF POISONING.

The course of poisoning follows three types—acute, subacute, and chronic—dependent on the dosage.

Acute poisoning manifests the intense symptoms, and

rapid denouement and termination consequent on the taking of a large dose. Thus, with irritant poisons, there may be burning sensations, nausea, vomiting (when possible), abdominal pain, diarrhœa, vertigo, and evidences of collapse; with nerve poisons, unrest, excitement, delirium, tremors, convulsions, difficult respiration, cyanosis, paralysis, coma, and the like.

Subacute poisoning results from smaller doses, and displays the same train of symptoms, less rapidly developed, less violent, and more protracted, extending over days or even weeks with eventual recovery or death.

Chronic poisoning resulting from the accumulated effect of repeated small doses, each inadequate to the production of serious symptoms, is not common among animals. Great differences may appear with one and the same poison as between the acute and chronic forms, as, for instance, in phosphorus poisoning in man. Chronic lead poisoning in animals is marked by persistent colic and constipation, and sometimes the formation of a blue 'lead line' on the gums; nervous symptoms are paralysis, convulsions, coma, and muscular wasting, and general debility and emaciation.

DIAGNOSIS OF POISONING.

Any case of sudden illness, or death, especially following on a meal, or after dipping, is commonly held to be one of poisoning. This idea is not by any means invariably right. It can only be verified by a full post-mortem inquiry, observation of the symptoms and history of the case, and chemical analysis of the organs. But when one or two animals are suddenly seized with violent symptoms, the presumption of poisoning has sufficient justice to make a post-mortem and analysis desirable. This is, perhaps, even more true when each of several animals known (as so commonly occurs) to move about and feed together are equally affected.

It is manifestly quite impossible to state general symptoms by means of which a case may be diagnosed as one of poisoning. Nevertheless, certain observations on the chief symptoms produced by common poisons may be of value.

(*a*) *Alimentary* symptoms comprise—Salivation, foaming, colic, retching, vomition, purgation (which may be bloody), bloody extravasation of the mouth, tongue, jaws, and fauces.

(*b*) *Circulatory* symptoms comprise—Accelerated or retarded throbbing or feeble heart-beat; hard, imperceptible or weak, irregular pulse; cold or hot dry skin; sweating.

(*c*) *Respiratory* symptoms comprise—Accelerated, retarded, intermittent breathing, with groaning, rattling, or gasping.

(*d*) *Motor* symptoms comprise—Trembling, quivering, cramp, stiffness or twisting of the neck, locking of jaws, epileptiform or convulsive seizures, paralysis of the hind or all the limbs, loss of feeling or great irritability of the skin.

(*e*) *Cerebral* symptoms comprise—Fear; shrinking on disturbance; frenzy and delirium, or dejection; hanging of the head; drowsiness; loss of sensibility and coma.

(*f*) Other symptoms are contraction or dilatation of the pupil, the eye being protruded or retracted in orbit; a fixed, anxious look; staring coat; suspension of lactation; repression or incontinence of urine, which may contain blood, albumin, bile or excreted substances, such as phenol derivatives in carbolic poisoning. The breath also may contain recognisable traces of volatile substances, especially hydrocyanic acid, and the mucous membrane of the mouth may show characteristic staining or erosion.

TREATMENT.

Since the particular poison causing a given illness is usually unknown, it becomes important to attempt to answer the question, What should be done in a case of suspected

poisoning? Although the special treatment may be impossible, the type of poison is indicated by the nature of the symptoms as above related—thus: as corrosive or irritant, acting locally on the alimentary tract; as depressant, tending to stop the action of the heart or respiration, or cause paralysis and coma; as convulsive, through excessive nervous stimulation. The main principles of treatment are—

1. *Prevent more poison being taken.* The affected animal and others liable to the same source of poison ought to be put into a safe place, and the food changed. Only known and safe food and water of proved purity (*ad lib.*) should be offered. Samples of suspected food and water ought to be taken. As far as possible, keep the patient quiet and unworried.

2. *Prevent absorption, or render inert any poison on the skin or in the stomach.* (*a*) Any skin application such as an ointment or dip must be washed off, preferably by soapy water.

(*b*) Clear the stomach, except in cases of strong corrosive poisoning, when perforation is liable to be caused. Emetics can only be used with the dog, cat, and pig, and thorough washing of the stomach is only practicable with the dog and cat. Fluids to dilute or neutralise the poison can only be given to the horse by drenching, unless voluntarily taken. Drenching and the hollow probang or stomach-tube are applied to the ox and sheep.

Useful emetics are—A *strong salt solution* (not advised for the pig); *mustard and water* (a dessertspoonful in 4 to 6 ounces); *zinc sulphate* (dog, grs. v. to x.; pig, grs. x. to xv.); *pulv. ipecac.* (dog, grs. x. to xxx.; pig, grs. xv. to ℥i.); *apomorphine hydrochloride* hypodermically (dog, gr. $\frac{1}{10}$ to $\frac{1}{5}$; cat, gr. $\frac{1}{30}$ to $\frac{1}{10}$.

To hinder absorption, *tannic acid* (horse, ℥ii.; ox, ℥iv.), *oak bark*, *oak galls*, or *logwood* (horse, ℥iv.; ox, ℥i.), in gruel; or charcoal (horse, ℥i.) in gruel or oil may be given. These remedies are primarily directed against vegetable alkaloids.

3. *Neutralise the effects of the poison taken.* Against irritants and corrosives use demulcents, such as oatmeal or starch gruel, linseed tea, carron oil (equal parts of linseed oil and lime water), olive oil, or castor oil (dog, ℥ss. to ℥ii.), petroleum emulsion, milk and eggs, or milk alone.

Abdominal pain may be relieved by giving opium tincture or laudanum (horse, ℥i. to ii.; dog, ʒss. to i.), or a hypodermic injection of morphine (horse, grs. v. to x.; dog, grs. ss. to ii.), or solution of chloral hydrate *per rectum* (horse, ℥ss. to i.; dog, grs. v. to xx.). It must be carefully remembered that many vegetable irritants exert narcotic effects after absorption. Care has, therefore, to be taken in using sedatives, which by still further depressing might cause death.

Against narcotics, paralysants, and depressants, such as opium and hemlock, stimulants are indicated. The following may be employed: Alcohol in the form of rectified spirits (horse, ℥ii. to iv.; dog, ʒi. to ii.), or as brandy or whisky (horse, ℥iii. to vi.; dog, ʒii. to iv.); ether hypodermically (horse, ℥i.) or *per rectum* (horse, ℥ii.; dog, ʒss. to ii.); draughts of strong coffee or tea; inhalations of ammonia.

If the animal is inclined to become drowsy and comatose, it must be kept moving with a whip, or by douches of cold water, and the application to horses or cattle of a liniment of ammonia and turpentine in oil is useful. With smaller animals artificial respiration may be tried.

Against convulsants (strychnine) use sedatives, such as opium, morphine, or chloral hydrate as above, or chlorodyne (horse, ℥i.; dog, ♏x. to xxx.), or inhalations of chloroform (℥i. on a sponge held to the muzzle for the horse; a few drops on cotton-wool for the dog). Nicotine given as tobacco infusion has been employed as an emergency strychnine antidote (see this).

4. *Promote excretion.* Unabsorbed material may be expelled by oily purgatives, such as linseed oil (horse and ox, 1 to 2 pints) and castor oil for the dog. Enemata promote peristalsis and excretion. The elimination of certain poisons is hastened by particular drugs; thus

potassium iodide is held to facilitate the elimination of lead and of mercury.

The best advice to owners is to do as little as possible, keep the patient quiet, summon professional assistance at once, and inform the veterinarian of all the circumstances and the measures already taken.

For cattle, one and a half times the dose advocated for the horse is to be used.

POST-MORTEM.

The post-mortem of a suspected poison case should be made with extreme care, particularly in view of possible legal action. Full notes of all circumstances relating to the surroundings of the subject, feeding, accessibility to sources of poisoning, or of persons likely to have malicious intent, and of the symptoms, should be made, verified, and preserved. Careful search may reveal recognisable or suspicious traces of poison, or of a poisonous plant. Any such material should be preserved and verified by *ex parte* evidence, which, whilst satisfactory in all cases, is almost essential in litigious cases.

The *lesions* are rarely—perhaps never—very characteristic, and the most common observation is of more or less acute gastro-enteritis. Such pathological changes as yellow atrophy of the liver in phosphorus and arsenic poisoning may be absent in very acute cases. In general pure alkaloid or other vegetable poisons do not produce irritation, but most mineral poisons and plants do so.

Search of the alimentary contents often discloses the cause. Hydrocyanic acid imparts its faint smell to all parts of a poisoned subject; similarly carbolic acid, chloroform, alcohol, and essential oils may be found. Phosphorus betrays its presence by its garlic odour and luminescence, but only when free, and it is hardly ever thus encountered in the dog.

Certain poisons impart colour; thus, copper salts give a greenish-blue, chromic compounds a yellow to orange or

green, nitric acid and picric acid a yellow colour. The blue (indigo, ultramarine, or Prussian blue) or black (soot) pigments of vermin powders are rarely detectable, because of the small quantity of the poison ingested.

CHEMICAL ANALYSIS.

Some details as to chemical toxicology are reserved to a later treatment in this volume. At this place, however, a few points of value to the clinician and chemist may be indicated. The analyst is at the mercy of the pathologist, who has it in his hands to render an analysis decisive, for or against, or to nullify the value of a laborious search.

The following suggestions are therefore made:

1. General details of the symptoms should be given, and particular note of the drugs administered in treatment. In a stomach the analyst may find a trace of an active drug, such as strychnine. Without knowledge of the treatment, he is confronted with the problem, Is this a residue of an original poisonous dose, or is it an unabsorbed fraction of a legitimate medicinal dose? With nothing before him save a jar of contents, and a card desiring an analysis, it is impossible to answer this question.

This particular case is not uncommon. In other instances, both lead and morphine have similarly been found, their presence being the result of legitimate medicinal dosage.

2. In the majority of cases the best material for research is contents of the stomach or intestines, not from the scientific, but from the practical point of view. In poisoning there is almost invariably a large overdose, and the unabsorbed excess can be separated and detected with facility from contents. In spite of the consensus of text-book opinion, our repeated experience, both in practice and experimental work, is that alkaloids cannot be satisfactorily separated from liver, kidney, or even urine. The case is different with metals and such poisons as cyanides, where

very delicate tests are available along with simple and quantitative methods of separation. It is, therefore, desirable to reserve contents, carefully selected, sealed, and labelled, or with a small animal the entire organs. A piece of stomach wall, carefully washed with water, is not a promising material.

3. Besides contents, portions of liver, kidney, blood and urine ought to be taken and separately sealed. To take a case in point. Arsenic is found in contents. The liver and kidney are then tested with positive results, thus giving satisfactory evidence of the giving of arsenic and its absorption. Or, if strychnine or an alkaloid is found in a stomach, and the other organs or urine can be shown to give evidence, in itself not conclusive so far as a clear chemical reaction is concerned, a satisfactory proof is afforded.

4. It is undesirable to add any preservative, such as carbolic acid, alcohol, glycerine, formalin, or the like. Practically all the ordinary poisons are stable, many quite permanent, for a sufficient length of time, and advanced decomposition does not affect the processes of extraction.

5. The proper sealing, labelling, witnessing, and transit of parts is expedient, as otherwise in contentious cases a large and expensive host of witnesses through whose hands the package has passed may be required in court.

6. If autopsy is not deemed necessary, the despatch of the uncut body or unopened stomach is most satisfactory.

MINERAL OR INORGANIC POISONS

ARSENIC.

Forms and Occurrence.—Arsenic is extremely widely diffused in nature, the chief sources being the yellow sulphides, *orpiment* and *realgar*, and *arsenical pyrites*, a double sulphide of iron and arsenic. It is, further, present in most natural metallic sulphides. When these are roasted or smelted, volatile arsenious oxide passes off and condenses in the flues, but in part may find its way into the air, into streams, on to the soil, and into herbage. When sulphuric acid is made from sulphur dioxide derived from pyrites, arsenic may be present in the acid, and may pass thence into substances prepared by its means; as, for example, into glucose, and eventually beer, and thus may give rise, as in the Manchester epidemic of 1900, to extensive arsenical poisoning. From smelting furnaces and the refuse of disused workings, arsenic may pass into vegetation, or into streams, as in the instance remarked by Dunstan of Liskeard under Lead (*q.v.*).

Arsenic enters into the composition of many everyday substances, and is used in certain preparations—*e.g.*, of some of the aniline dyes.

The most poisonous compounds of arsenic are the hydride and certain organic compounds, such as those of dimethyl arsine (*e.g.*, cacodyl oxide $[(CH_3)_2As]_2O$, and the chloride $(CH_3)_2AsCl$), but poisoning by these is unlikely outside the laboratory. There does not appear to be much solid foundation for the widespread idea that the hydride is slowly generated by the action of fermenting paste on wall-

papers coloured with arsenical pigments, although arsenic may be found in the dust of rooms so papered.

Arsenious oxide, *arsenious acid*, and its *salts* are by far the commonest forms of arsenic. The oxide, crystalline, vitreous, or amorphous, sparingly soluble in water and volatile on heating, is the so-called 'white arsenic,' also erroneously known as 'arsenious acid,' or simply as 'arsenic,' of trade. The sublimed oxide forms characteristic glistening octahedral crystals. From its likeness, when powdered, to flour accidents have occurred. It enters into the composition of certain *rat powders*, in which it is often mixed with barium carbonate, flour, and blue.

Of the *salts of arsenious acid*, *copper arsenite* is Scheele's green, and a double *copper arsenite* and *acetate* is Schweinfurt's green.

The alkali (sodium and potassium) salts are easily soluble, as also are the alkali thio-arsenites, in which oxygen is in part replaced by sulphur. Alkali arsenites are used as *weed-killers* and *wheat dressings*, and a common *sheep-dip* consists of alkali arsenite and thio-arsenite, along with sulphur. Other dips are of alkali arsenite, soap, sulphur, and sometimes iron sulphate. Dips usually contain about 20 per cent. of soluble arsenic and 3 per cent. of insoluble arsenious sulphide, which is held to exercise a protective effect during the intervals between dipping. After solution for use the strength of soluble arsenic lies between 0·25 and 0·5 per cent. The arsenites are the most toxic of the ordinary arsenical preparations.

Arsenic acid, its oxide and salts, are less commonly met with, and are less toxic, their action probably depending on their reduction from the pentavalent arsenic to the trivalent arsenious form. *Sodium arseniate* is sometimes used as the poison of fly-papers.

The common *medicinal forms* of arsenic are *Fowler's solution*, which is a 1 per cent. solution of arsenious oxide in 1 per cent. potassium carbonate, and *liquor arsen. hydrochl.*, which is a 1 per cent. solution of the oxide in dilute hydrochloric acid. To these must now be added the

numerous synthetical organic arsenic derivatives, for many of which science is indebted to the work of Ehrlich and his school. *Cacodylic acid*, $(CH_3)_2AsOOH$, is a long-known pentavalent arsenic compound, and is relatively inocuous in comparison with the trivalent cacodyl compounds above named. Atoxyl, used in trypanosomiasis, and '606,' used as a specific for syphilis, are more recent examples.

But atoxyl may cause poisoning, as in the case observed by Wooldridge,* who gave a bulldog 0·1 of a gramme in 10 per cent. solution on alternate days. After four doses the animal died, having shown symptoms of poisoning. According to Miessner,[10] cattle suffering from foot-and-mouth disease are susceptible to salvarsan ('606'), being killed by 45 grains intravenously.

In view of the importance and frequency of arsenical poisoning, the chief channels of administration may be here summarised: (*a*) Administration of the drug for malicious purposes, in mistake for other drugs, or in incorrect medicinal dose. (*b*) As the result of dipping (see below). (*c*) By local application, as in the treatment of warts and mange. (*d*) By the accidental partaking of dips, weed killers, rat powders, and the like. (*e*) Administration by horse attendants to improve the animal's condition and coat. (*f*) By contamination of water and herbage through the neighbourhood of metal smelting works or mine refuse.

Toxicity of Arsenic.—The garlic-smelling vapour of free, or metallic, arsenic is toxic, probably oxidising in part to arsenious oxide. Finely divided metallic arsenic is also dangerous when rubbed into the skin; but, in coarser division, chemically pure arsenic is harmless when given internally. Similarly, coarsely powdered white arsenic, which is sparingly soluble, can be given in large doses to dogs without ill effect. Thus 270 grains of coarsely powdered white arsenic failed to kill a dog, as also did 15 grains a day over four months; but $\frac{2}{10}$ grain of potassium arsenite and 2¼ grains of sodium arseniate proved fatal when given

* See Finlay Dun, 'Veterinary Medicines,' 1910, p. 284.

by the mouth.* In very fine division, or in solution, and particularly in the form of alkali salts, the arsenious compounds are most readily absorbed and most toxic. Absorption through the unbroken skin may occur when an arsenious solution has remained for a long time in contact therewith, and accidents have occurred to sheep-dippers from this cause.

Arnold Theiler,[11] experimenting in South Africa, has published the results of a large number of tests made to ascertain the safe dose of white arsenic (arsenious oxide), Cooper's dip (an alkaline arsenite and thioarsenite with sulphur), and bluestone (copper sulphate) for sheep. All these substances are common remedies for intestinal parasites, and are largely used and easily procurable. Theiler's results have added value by reason of the fact that he was able to use large numbers of animals in his tests. For each dose thirty sheep were used. All were watered twenty-four to twenty-eight hours before dosing, fifteen immediately after, and fifteen within twenty-four to twenty-eight hours after. Theiler does not appear to have reckoned on the sparing solubility of white arsenic, for he remarks on the extraordinary circumstance that doses varying from 7½ to 150 grains failed to kill in special tests designed to disclose the toxic dose. In the larger scale experiments, two of fifteen sheep, which had 60 grains each, followed at once by watering, died, one on the seventh and the other on the eleventh day, both showing perforation of the fourth stomach, peritonitis, and gastro-enteritis, with pale yellow liver. As abundant examples prove, these are not invariable signs of acute poisoning, and the results Theiler got must be held to illustrate slow poisoning, probably with lodgment of coarser particles in the alimentary tract. As regards Cooper's dip, Theiler concludes that 15 grains is a safe dose. He found that 30 grains killed 1 in 48; 45 grains killed 4 in 30; and 60 grains killed 14 in 15 watered at once after dosing, and 6 of 15 watered twenty-four hours after dosing. Whereas the

* See *Revue Générale de Médecine Vétérinaire*, February, 1911.

animals watered at once died within twenty-four hours, the others died after seven or eight days. This result is intelligible on the supposition that the dip is more easily absorbed after extensive dilution, but the author has observed symptoms speedily caused in cattle very soon after licking dry dip.

As regards mixtures of bluestone and arsenious oxide, Theiler found that 15 grains of each is a safe mixture. With a dose of 30 grains of each, five of thirty-six sheep had prolapsed stomach, and were slaughtered. For bluestone and Cooper's dip 15 grains of each is regarded as a safe mixture. Thirty grains of bluestone and 15 grains of Cooper's dip, and 30 grains of each, both proved dangerous mixtures, each killing one out of six sheep within twenty-four hours from gastro-enteritis.

A very remarkable result was obtained on adding common salt and sulphur to the mixture of bluestone and Cooper's dip. A mixture of equal parts of each of these four substances was made. A dose of 45 grains, containing $11\frac{1}{4}$ grains each of bluestone and dip, killed five of sixty-five sheep from gastro-enteritis, and a 60-grain dose, containing 15 grains each of bluestone and dip, similarly killed sixteen of eighty-eight animals. It thus appears that the addition of salt and sulphur increases the toxicity of the copper sulphate and arsenical dip.

In respect to the organic derivatives the researches of Ehrlich have well established the dependence of toxicity on the valency, or degree of oxidation, of the arsenic. It has been long known that arsenic acid is less toxic than arsenious acid, and, according to Ehrlich, the toxicity of a pentavalent arsenic compound depends on its relative ease of reduction in the living cells to the trivalent stage.* It is further clear that the position of the arsenic in the molecule is of great significance; thus the action of the hydride, AsH_3, is totally different from that of the acid, H_3AsO_3, in which the arsenic is part of the radicle or ion, AsO_3. It is thus not surprising that among the numerous compounds where pentavalent

* See abstract, *Veterinary News*, 1909, p. 155.

arsenic is united to organic groups, such as methyl and phenyl, orders of toxicity down to practical inactivity, dependent on the relative stability and resistance to reduction, have been noted.

These considerations make it clear that for practice the most dangerous arsenic preparations are those in which the arsenious acid is in a readily soluble form, as in alkali arsenite dips and weed-killers, and to a less degree with copper arsenites and finely divided arsenious oxide.

Absorption.—The absorption of arsenic is manifestly intimately connected with the form of administration. As stated, the coarsely powdered porcelain, or vitreous oxide, appears to be without action on the dog, and according to Kaufmann 45 to 60 grains of the dissolved oxide will kill a horse, whereas 675 grains of the solid would be required.

It is to be clearly remembered that solution is very slow from the solid oxide in the digestive tract, and that, in consequence, no exact connection between dosage and effects of this form can be traced. It appears possible —at any rate, it is worth investigation to ascertain—that much of the alleged arsenic toleration in man and animals is due to the fact that solid is taken, that absorption is very slight, and that the greater part of the drug is excreted in the fæces unchanged. Many of the conflicting statements as to dosage and effect are due beyond question to the inadequate recognition of this fact. It must, however, be remembered that by gradual dosage the horse acquires a measure of tolerance. Thus, in the Holmes treatment after a time 45 to 50 grains may be given against piroplasmosis, surra, dourine, and other affections due to trypanosomes. Moreover, Ehrlich has shown that strains of trypanosomes can be bred which are immune to arsenic.

In the dog, according to Henry Gray, long continued small dosage is more likely to cause toxic effects than a few large doses. After such prolonged dosage, irritation of the conjunctivæ, vomiting, and loss of appetite occur.

Absorption of arsenious acid takes place slowly through the intact skin. When introduced by a fresh wound, about

one-tenth of the ingested dose proves fatal—*i.e.*, about 60 grains for the horse and ox, as against 600 to 700 grains by the mouth. It will be observed that this dose is about equal to that of dissolved arsenious acid by the mouth.

The local effect of arsenic on mucous membranes is caustic, with formation of sloughy gangrene.

Absorption of dissolved arsenic from the mucous membranes of the digestive tract is very rapid, and the irritant effects are well marked with concentrated solutions. In a recently investigated case of acute arsenical poisoning, $\frac{1}{10}$ grain per ounce of unabsorbed arsenic was observed in stomach contents, whereas the contents of the small intestines were almost free of arsenic. This illustrates the rapidity of absorption and production of general toxic effects. Similarly, in acute cases arsenic is speedily found in the liver, although in less quantity than in subacute or slow-poisoning cases. Interesting data bearing on this point were obtained in investigating three cases of arsenical poisoning by dip of Jersey cows. In Case I. death was very rapid, within twenty-four hours of dosage; in Case II., five days; and in Case III., seven days elapsed. The figures tabulated show the proportions of arsenic in grains per ounce found in the parts named:

	Rumen.	Fourth Stomach.	Liver.	Spleen.	Period of Illness.
Case I. ...	$\frac{1}{6}$	$\frac{1}{40}$	Trace	Trace	Less than 1 day.
Case II. ...	$\frac{1}{30}$	$\frac{1}{15}$	$\frac{1}{900}$	—	5 days.
Case III. ...	$\frac{1}{24}$	$\frac{1}{36}$	$\frac{1}{300}$	$\frac{1}{360}$	7 days.

The noteworthy point is the evidence of the gradual distribution of the poison over the alimentary system, and its increasing accumulation in the liver.

With the passage of time the quantity remaining in the ingesta will dwindle. Thus, in a well-established case of cattle poisoning by weed killer, investigated in 1906, the arsenic was found to be very evenly distributed throughout

stomach and intestinal contents, and averaged the low proportion of $\frac{1}{128}$ grain per ounce, whilst the liver was arsenical to about the same degree.

After ingestion and absorption arsenic may be detected in the blood and most of the organs, thanks to the extreme delicacy of the methods of chemical research for this element. As with the heavy metals, arsenic is temporarily stored in the liver.

Elimination of arsenic is, however, rapid, and takes place chiefly by the kidneys. It probably does not undergo the process of biliary excretion, reabsorption from the intestine, and return to the liver, or gastro-hepatic circulation, recognised by Claude Bernard for lead.

Five or six hours after ingestion arsenic may be recognised in the urine and milk, and elimination is rapid, being complete within two to three days. Elimination also takes place by way of the mucous secretions and the skin, to which may be attributed the effects of arsenic in skin diseases.

Toxic Doses.—The toxic dose of arsenic depends upon the nature of the arsenical compound; upon its condition —solid, coarsely or finely powdered, or dissolved; upon the condition of the digestive organs and nature of the ingesta; upon the species, and individual, and idiosyncracy; and upon the mode of application. Consequently, great variation is to be expected, and the figures here quoted from Kaufmann must be regarded only as approximate. As to actual amount ingested, the fowl is, as in other cases, most variable, by reason of the relatively great doses which may be stored in the crop. The figures relate to arsenious oxide:

	Powder by the Mouth.	Powder by a Wound.
Horse	150–700 grains ...	30–60 grains
Ox	225–700 ,, ...	30–60 ,,
Sheep	75 ,, ...	3–6 ,,
Pig	7·5–15 ,, ...	0·3 grain
Dog	1·5–3 ,, ...	0·03 ,,
Fowl	1·5–1·65 ,, ...	0·03 ,,
Man	1·5 ,,	

Diagnosis.—The sudden appearance of violent symptoms of colic, thirst, straining, and purgation in large animals, or of vomiting in dogs, will arouse suspicion that irritant poisoning, probably by arsenic, has occurred.

Symptoms.—Numerous recorded cases have fairly established the general symptomatology of arsenic. There are to be distinguished the local irritant and remote nervous effects. The arsenic compounds do not precipitate albumin, and the irritant effect has thus a different mechanism from that due to heavy metal salts. In general, in acute poisoning of the herbivoræ one has to note—salivation, thirst, loss of appetite, vomition when possible, violent colic, fœtid diarrhœa of alliaceous odour, and sometimes bloody, exhaustion, collapse, and death. Apart from the extreme debility, there may be noted paralysis of the hind extremities, coldness of ears and horns, and a subnormal temperature, with trembling, stupor, and convulsions. The onset of symptoms and death may be extremely rapid, so that the animal may never be noticed to be ill. In more prolonged cases giddiness, muscular tremors, colic, and coldness will be prominent. The urine is albuminous and often bloody.

The notes by Bevan[1] give an excellent summary of arsenical poisoning. The effects due to absorption through the skin, noted by that observer—viz., a scalded appearance and sloughing in patches, especially round the eyes, over the scrotum of the bull, or udder and vulva of the cow—recall the parallel effects of arsenical eruption in man.

In dogs a large dose (3 to 10 grains) causes nausea, vomiting, moaning, hard and rapid pulse, painful evacuations, and death in convulsions in from six to thirty hours.

Chronic poisoning is less frequent and less well marked with animals than with man; few cases are to be found in the veterinary literature. As characteristic of it, there are noted diminution of sensibility, difficulty in movement, and eventually entire abolition of the motor and nutritive functions. Indigestion, thirst, great wasting, and chronic disease of the joints have been observed amongst animals living near smelting works in Cornwall and Wales.

An interesting case of poisoning by absorption is noted by Mahon,[2] in which arsenic was used in mistake for zinc sulphate in a case of greasy heels. The condition was obstinate, but ultimately yielded to treatment.

Wallis Hoare has observed colic and purgation caused in a horse by the application of a strong arsenical ointment to raw warts. The trouble gradually passed off after removal of the dressing.

Cases of poisoning by copper arsenite, or Scheele's green, used in colouring wall-paper, have been observed in the donkey and ox,[4,6] and a case of poisoning of sheep by lead and arsenic has been communicated by Dunstan.

As regards dips, a lengthy report of a legal action is given in the *Veterinarian* of 1858, in which the conflicting expert opinion and the difficulty of experimental verification are well illustrated. The plaintiff lost 850 out of 869 sheep within two or three days after dipping in a dip containing $2\frac{1}{2}$ pounds of arsenic per 100 gallons. He obtained a verdict, though others had safely used the same or similar dips; and Gamgee, Macadam, and Dun failed to secure experimental poisoning with even stronger solutions. The strength noted is regarded generally as safe, when properly used.

Most authorities are agreed that there is no danger of absorption of arsenic in toxic doses from dips through the unbroken skin. Absorption to a certain extent must, however, occur, and H. E. Laws,* of Messrs. Cooper, holds that the killing of the parasites is caused by their taking up arsenic from the blood stream of the host. In dipping the solution ought not to contain more than 5 pounds of arsenic per 100 gallons, each sheep being immersed for from forty to sixty seconds and requiring 1 gallon. The solution must be as completely squeezed out of the fleeces as possible, and the animals turned out, if possible, on to a dry road or large yard free of hay, litter, vetches, or green food. Accidents arise chiefly from the licking of the fleeces when the sheep are overcrowded, and from the drippings

* Private communication.

on to grass or fodder, which then becomes a vehicle of poisoning.

Post-Mortem Appearances.—Notable signs of arsenical poisoning are—intense rose-red gastro-intestinal inflammation with ecchymoses and extravasations[5,9]; fatty degeneration of liver, kidneys, heart, and nervous centres in protracted cases. Well-marked preservation of the organs highly charged with arsenic is characteristic.* But it is to be observed that the detachability of the lining mucous membranes disclosing injected submucous layers is not invariable, and depends on the concentration. Several cases are on record in which inflammation was not a prominent feature,[3,7] and some have been investigated where poisoning was caused by dilute weed-killer and where also inflammation was not notable. Such examples ought, however, to be viewed as exceptional. In some cases actual perforation may be found.

In cases of pigs poisoned by partaking of waste dip, Varnell (1859) observed inflammation with effusion of lymph of the membranes of the mouth and fauces, which extended to the larynx and trachea, with production of asphyxia.

Treatment.—This should consist of emetics and purgatives, with milk, egg-white, and lime water as demulcents. As specific antidotes, calcined magnesia and freshly precipitated ferric hydroxide are used. The behaviour of the latter towards dissolved arsenious acid is remarkable. When shaken up with a solution of arsenious acid, the hydrated iron oxide fixes the arsenic, and thus withdraws it from solution. This it does by the process of adsorption, which is physical, and not chemical. As to whether arsenic, thus rendered insoluble, is dissolved, and therefore absorbed by acid digestive juices, is uncertain, and is a point worthy of experimental investigation.

Magnesia is very efficacious in preventing gastric in-

* In a poisoned fox examined recently the stomach, which was heavily arsenical, was exceedingly well preserved, the other organs being, indeed, almost entirely decomposed.

flammation, probably by neutralising the acidity of the stomach.

In dealing with an emergency case of arsenical poisoning in the ox, horse, or sheep, one may precipitate tincture of iron perchloride with soda carbonate, filter through a handkerchief, and give *ad lib.* in warm water. Dialysed iron, $\frac{1}{2}$ to 1 ounce for dogs and 6 to 15 ounces for horses and cattle, may be given.

As demulcents, oil or equal parts of oil and lime water or linseed tea are given.

Hypodermic injection of morphia is desirable if there is much pain, and in case of prostration strychnine and ether, also hypodermically.

Chemical Diagnosis.—More attention has probably been bestowed on the methods of detecting traces of arsenic than has been devoted to any other analytical process. The epidemic of arsenical poisoning through beer led to the establishment of refined standard methods, and laid new emphasis on the extremely wide distribution of this element and the need of caution against its presence in laboratory reagents. It may, indeed, be not unfairly assumed that in many cases in which very small amounts of arsenic have been detected it occurred not in the materials examined, but in the chemical agents used for its detection. Complete destruction of organic matter by heating with strong sulphuric acid, or its partial destruction by means of hydrochloric acid and potassium chlorate, are standard methods, the latter being favoured by German toxicologists. A method also favoured by the latter experts consists in distillation of the volatile arsenious chloride from organic matter and hydrochloric acid. In our experience none of these methods offers any advantage over the older Reinsch process of concentrating the arsenic on pure copper by boiling with dilute hydrochloric acid. Very little chemical need be added, and the organic matter need not be destroyed. The delicacy of the method amply suffices, since very clear results are given by $\frac{1}{1200}$ grain of arsenious oxide in 4 ounces of organic matter. The arsenic

must be verified by heating the coated copper in a dry tube, when the cooler parts of the tube become coated with a glistening white crust, which shows the octahedral crystalline form under a low power, and by solution from the copper by means of pure sodium hydrate and hydrogen peroxide, or by pure sodium peroxide, and submitting the solution to the well-known Marsh test. The quantitative estimation of arsenic is usually performed by comparing the size of the mirror got in Marsh's test with sealed standard mirrors prepared from known quantities of arsenic. In this way absolutely indisputable evidence of the presence of arsenic may be obtained, and chemical diagnosis is a matter of certainty, with the single exception of the rare, though theoretically possible, case where death is just produced by a dose of such magnitude that the elimination of the last trace of the poison coincides with the time of death.

Medico-Legal.—It has been held that arsenic remains longest in the bones, and that it may be found in the hair. It may be recognised a very long time after death, but in cases of analysis of exhumed parts care is needed to exclude the possibility of arsenic having entered in traces from the soil.

In all analysis it is necessary to prove by blank experiment that the apparatus and chemicals used are free of arsenic.

The actual proportions found in practice vary very widely, as may be expected. The greatest proportion is often that noted in the crop contents of fowls. In cattle arsenic has been observed in the varying proportions of 4 grains to $\frac{1}{128}$ grain per ounce of ingesta, and it will be clear that these figures refer to the excess of poison, and bear no definite relation either to the original dose or to that absorbed part by which poisoning was caused. In those cases where the greater part of the dose is rejected by vomiting, recovery may occur; but, if death ensues, arsenic is generally to be found in the stomach walls and liver.

Unless the quantity of arsenic actually separable from

the viscera is relatively great, it is necessary, in order to legally establish a case of poisoning, to prove that no arsenical medicine has been given, that there has been access to an arsenical preparation, and that the symptoms and lesions are consistent with those of arsenic.

REFERENCES TO ARSENIC.

[1] L. E. W. Bevan, *Vet. Jl.*, 1908, p. 557.
[2] F. C. Mahon, *Record*, 1889-90, p. 120.
[3] C. Hoole, *Jl. Comp. Path.*, 1894, p. 379.
[4] Beeson, *Veterinarian*, 1871, p. 472.
[5] Tuson, *Veterinarian*, 1865, p. 419.
[6] H. Lepper, *Veterinarian*, 1865, p. 352.
[7] Varnell, *Veterinarian*, 1859, p. 510.
[8] Case of Dip-Poisoning, *Veterinarian*, 1859, pp. 223, 268.
[9] J. C. Truckle, *Veterinarian*, 1855, p. 142.
[10] Miessner, *Archiv wissen. und prakt. Tierheilkunde*, 1911, p. 602.
[11] A. Theiler, *Agricultural Journal of Union of South Africa*, 1912, p. 321.

ANTIMONY.

Preparations.—The compounds of antimony do not find much use in modern medicine.

Potassium antimonyl tartrate, or *tartar emetic*, prepared by boiling together antimony oxide and potassium acid tartrate, is the most convenient soluble antimony compound. Along with iron sulphate it is used as a vermicide, and is recommended by Noel Pillers as very effective against lumbricoids of the horse.

The native *sulphide*, or *black antimony*, purified by fusion or by digestion with ammonia, in order to free it as far as possible from arsenic, is still extensively used in the manufacture of the so-called 'condition powders.' These contain about 18 per cent. of antimony sulphide, together with nitre, sulphur, and a spice—*e.g.*, aniseed or fenugreek. The old-fashioned Kermes mineral is a mixture of sulphide with a little oxide.

The *oxide*, which is colourless, is sometimes used as a

paint. Like arsenious oxide, it is volatile, but gives an amorphous sublimate. A strong solution of the *chloride* is made by boiling the sulphide with hydrochloric acid, and constitutes butter of antimony. It is used as a caustic, and is characterised by giving a turbidity of the insoluble oxychloride on dilution with much water.

Actions of Antimony.—The sulphides and oxides are slowly dissolved by the digestive juices, and thus exercise similar, but less intense, effects to those of the soluble preparations. The soluble compounds act as gastro-intestinal irritants, causing, in *carnivora*, vomition, and in large doses also violent purging, weakness, collapse, and death.

According to Kaufmann, dogs are poisoned by 3 to 6 grains.

The antimony compounds do not appear to owe their emetic properties to an action upon the centres of vomition, but to their local gastro-intestinal effect; for, on injection, very large doses are required to produce emesis, and it is found that the antimony is excreted into the alimentary tract.

Like arsenic, antimony causes fatty degeneration of the liver, and the oxide is therefore given to geese to produce fatty liver for the preparation of *foie grasse*.

Ruminants are able to withstand very large doses, and some doubt has been expressed as to whether poisoning can possibly be produced by antimony; but extreme nausea, colic, and death have been observed in the horse, whilst actual vomition in the cow, following the administration of Kermes mineral, is on record.*

Finlay Dun† quotes valuable experiments on horses with tartar emetic. They show that such large quantities as 10 ounces of the drug given over a period of ten to eighteen days do not exercise any noticeable physiological effect. But a healthy horse, given 10 ounces of tartar emetic in solution in one dose, showed nausea, uneasiness, and pain, and died within about six hours.

* Winslow, 'Veterinary Materia Medica,' 1901, p. 215.
† 'Veterinary Medicines,' 1911, p. 268.

Concerning the toxic effect of antimony on carnivora there can be no question, and as regards the herbivora, there is sufficient evidence to warrant the opinion that the habitual use of antimonial medicines is objectionable, and may, particularly with young and delicate thoroughbred animals, cause poisoning. In consideration of the large quantities of antimonial condition powders, which are administered empirically as a matter of weekly routine by persons in charge of stock, this is a very important point, worthy of close attention.

G. Armitage* quoted a case of the death of pigs. They were stated to have had the usual food, exhibited severe abdominal pain, and made unsuccessful efforts to vomit, but there was no purgation.

On post-mortem the stomachs were gorged, and the mucous membranes showed intense inflammation, extending to the whole of the small intestine. The large intestine was not inflamed. In the stomach was found a deposit of black grains of antimony sulphide, and the opinion was formed that death had resulted from an antimony, nitre, and sulphur condition powder.

In 1909 a case was investigated in which a six months blood filly, apparently well overnight, was suddenly seized with violent scouring, and died very quickly. On post-mortem acute inflammation of the stomach and bowels was observed, the other organs appearing normal. Antimony was found in the viscera—it was admitted that the animal had been dosed with a condition powder—and no other cause of poisoning or death was discernible.

In another case investigated in 1905, doping heavily with antimony prior to a sale appeared the only explanation of the sudden death of a mare.

Treatment.—Antimonial poisoning is treated by removal of the cause by evacuation of the stomach and oily purgatives with demulcents.

Tannic acid precipitates tartar emetic, and is used as a chemical antidote.

* 'Vet. Records,' 1865, p. 337.

Morphine against pain, and stimulants are to be used as required.

Chemical Diagnosis of antimony is best accomplished by the aid of Reinsch's test, the deposit on the copper being distinguished from arsenic by yielding an amorphous sublimate on heating, and further by the well-known difference in behaviour in Marsh's test.

A solution of antimony in hydrochloric acid, in which are immersed pieces of zinc and platinum in contact with one another, gives a black stain of antimony on the platinum. This is a delicate and characteristic reaction, which may also be used for the separation of antimony from a hydrochloric acid extract of organic matters.

LEAD.

Forms and Occurrence.—The common preparations of lead likely to give rise to poisoning are—the oxides *litharge* and *red lead*, the latter being used as a paint and in plumbing; *lead acetate*, or sugar of lead, and the *basic acetate*—Goulard's solution; *white lead*, a basic carbonate, the common pigment, and the most usual vehicle of poisoning, also used in the manufacture of oilcloth and linoleum*; the *sulphate*, less frequently used as a pigment. *Metallic lead*, in the form of bullet splashes, has been observed to give rise to poisoning, but only after prolonged lodgment in the digestive system, during which the metal is corroded and absorbed. Metallic lead, through solution in water under certain conditions, may also be the cause of chronic lead poisoning, or plumbism. The conditions governing the solution of lead by water are—that the water is soft—that is, free of lime and magnesia salts—and aerated. It must contain dissolved oxygen and carbon dioxide, and the presence of nitrates further facilitates solution. The extent of solution may be gathered from the following figures,† which show the

* White lead substitutes, such as "lithopone," which do not contain lead, are coming into increasing vogue.

† Roscoe and Schorlemmer, 'Treatise on Chemistry,' 1897, vol. ii., p. 722.

number of milligrammes of lead dissolved by 500 c.c. of water from a bright surface of 5,600 square millimetres :

	Twenty-four Hours.	Forty-eight Hours.	Seventy-two Hours.
Distilled water	2·0 ...	2·0 ...	3·0
Distilled water +0·02 gramme ammonium nitrate per litre	13·0 ...	— ...	25·0

Such conditions are fulfilled by rain water, but ordinary hard water is without perceptible solvent action. The lead is at first dissolved by soft water, but eventually separates in the form of white flakes of basic carbonate.

Lead poisoning of cattle almost always results from eating white paint or red lead, and of dogs by licking wet paint, or by licking lead lotions applied externally.

Absorption and Elimination.—With the exception of the acetate, or sugar of lead, and the basic acetate, the preparations named above are insoluble in water, the oxides and carbonate dissolving easily in dilute hydrochloric acid,* the sulphate less readily, in conformity wherewith it has the least toxicity, and its formation by the exhibition of dilute sulphuric acid, or a soluble sulphate (Epsom or Glauber salt), is an antidotal measure.

It has been held that lead is absorbed as the chloride formed by the action of the gastric juices. Lead salts, however, precipitate albumins, and absorption as lead albuminate is more probable.

The relative insolubility of the lead salts and of the albuminate account for the fact that lead is one of the least corrosive metallic poisons. The question of poisoning by metallic lead has been the subject of some controversy (*vide* References), but is now held to be established. The exposure of bullet splashes to weather leads to superficial oxidation or rusting, whereby a coating soluble in acid will be formed. In like manner, effluviæ from lead-works may eventually impregnate herbage, and lead from the refuse of old disused workings gradually finds its way into the

* It will be remembered that lead chloride is sparingly soluble.

neighbouring streams, soils, and herbage.* Thus, in a case brought under the author's notice by Dunstan of Liskeard, the water of a stream was proved to contain both lead and arsenic—the latter being adsorbed by suspended iron oxide.

Lead is retained by the organs for a long time. The blue line on the gums, supposed to be caused by a deposition of lead sulphide, only disappears very slowly after withdrawal of the cause. The liver, kidneys, bones, nervous tissue, and muscle retain lead in the relative order given. According to Ellenberger the percentages of lead found in those organs of a sheep which during four months had received 164 grammes of lead acetate, were—

Liver	0·065	Nervous tissue ...	0·018
Kidneys	0·047	Muscle	0·0084
Bones	0·032		

Elimination is slowly effected by way of the bile, urine, salivary, mucous, and cutaneous excretions, and is stated to be hastened by the administration of potassium iodide.

Toxic Dose.—This cannot be regarded as satisfactorily established. Very usually a large excess of lead remains in the alimentary tract, cows frequently eating lead paint refuse by the pound. The dose is also determined by the nature of the preparation given, the general condition, idiosyncracy, and species of the subject. The following provisional figures may be quoted for minimum toxic doses of acetate of lead:

Ox	720 grains
Horse	7,500 „
Sheep	450 „

The relative tolerance displayed by the horse is a remarkable point, and is on a parallel with the comparative rarity of cases among those animals, the usual victims being in order — cattle, pigs, sheep, dogs. Birds are readily susceptible, but cases of poisoning of them by lead are rare.

* See on this point Taylor, 'Poisons,' 1875, p. 438.

Symptoms.—Numerous well authenticated cases on record in the literature enable a close picture of the symptomatology of lead to be indicated. But by reason of the nervous symptoms lead poisoning may easily be confounded with that due to some vegetable poisons. Toxic, or numerous small, doses first irritate and then paralyse both voluntary and involuntary muscles through the motor nerves.

Gastro-enteritis, colic, convulsions, coma, and death are very general effects of *acute poisoning* of cattle[1,2,4]. Noteworthy are signs of intense abdominal pain, grinding of teeth, nasal discharges, salivation, pallor of mucous membranes, constipation, with passage of hard black dung, fœtid breath, ropy urine, blindness, muscular tremors, and coma. The pulse is hard and thready, breathing acclerated, and temperature nearly normal or depressed, whilst the extremities are cold. In cattle there is delirium, alternating with a semi-comatose condition, in which the patient may assume an unnatural position, and make no attempt to alter it.

An interesting case of *subacute poisoning* with subsequent recovery of foals was quoted by D. Pugh in 1897.[3] The symptoms were listlessness, watery discharge from nose, eyes bright and prominent, tongue red, offensive breath. Pulse frequent, hard and weak, temperature 100·4° F. Refused food, fæces hard, urine highly coloured and ropy. Under usual treatment recoveries were made in from seven to twenty-one days. The cause was the scaling off of flakes of paint from a bucket.

The onset of symptoms may be slow—up to forty-eight hours—but is generally unknown, and the period of illness may be protracted, as, for instance, over nine days in a case communicated by Ainsworth Wilson, of which the subject was a calf.

Chronic Poisoning.—The blue line on the gums only appears in chronic poisoning, which is a rarer event in practice than the acute. In chronic poisoning, so common formerly among workers in lead, are to be noted the general digestive derangements, colic, constipation alternating with diarrhœa, and thirst; the nervous symptoms of paralysis,

convulsions, and coma observed in the acute form; the general symptoms of wasting, emaciation, debility, and the like. Rumination and lactation cease, and convulsions, coma, and paralysis precede death.

Some excellent examples of chronic or slow lead poisoning in cows, due to bullet splashes, were recorded by Tuson,[7] Broad,[8] and Watson in 1865. In these cases the period intervening between the ingestion of the lead splashes and illness was prolonged, amounting to as much as fifty-eight weeks. The symptoms observed were—abdomen tucked up, staring eyes, dull look, staggering, groaning; lactation ceased, appetite good, constipation alternated by diarrhœa; gradual wasting and prostration preceded death. The blue line was absent. The viscera generally were pale, the intestinal walls having a peculiar blue colour, and the abdominal cavity contained about 3 gallons of straw-coloured fluid. Metallic lead was found in the reticulum, and Tuson observed that this had been corroded by the digestive juices. These cases are further remarkable in illustration of the lodgment of the solid matter so permanently in the digestive system.

The *Veterinarian*, 1855, p. 609,[9] records an interesting legal action with comments, arising from the chronic poisoning of stock, due to lead-smelting operations in the Mendips. There were observed stunted growth; leanness; shortness of breath; paralysis, especially of hind extremities; swelling at knees; but no constipation or colic. It was adduced by Herapath, in evidence, that the blue line on the gums gave, on dissection and blowpipe reduction, visible beads of metallic lead.

Plumbism is remarkable from the length of illness, which may be protracted over weeks or even months. In horses lead causes roaring and dyspnœa by acting on the vagus nerve. Cases in point are given by the German authorities, and have also been observed by Shenton in this country.*

Post-Mortem Appearances.—Inflammation of the fourth

* See Dun, 'Veterinary Medicines,' 1911, p. 228.

stomach and intestines is a common, but not invariable, condition. Particles of lead, sometimes amounting to several pounds, as in a case recorded by Nash[5] (1894), may be found in the reticulum. It is usual to find pieces of lead paint of the size of beans, blackened externally, friable, and white internally. Where such pieces have been in contact with mucous membrane, the latter is also blackened and easily detachable, revealing inflammatory patches. Acute peritonitis, with formation of greyish-yellow false membrane, has been observed.

In a case observed by Lawson[6] (1884) the mucous membrane was detachable; no inflammation; liver bloodless and yellow; lungs engorged with black blood, inflamed and emphysematous; trachea and bronchi filled with a frothy spume.

In some cases the liver has been found engorged, and in others the organs have been found healthy. The production of inflammation depends on the nature of the preparation given. It is not shown when the dose is small or in the presence of an excess of acid; in the form of albuminate dissolved in acetic acid death is rapid and inflammation absent; but with solid carbonate dark red inflammation is to be anticipated.

Treatment.—Removal of the cause by emetics, by the pump, and by means of saline purges; as chemical antidotes, dilute sulphuric acid; or soluble sulphates, such as Epsom or Glauber salt; casein, given as milk, is recommended, in order to precipitate lead albuminate; tannic acid, as tea or coffee; and stimulants, *e.g.*, digitalis or ammonium acetate, have been used. In chronic plumbism the exhibition of potassium iodide is claimed to facilitate elimination.

Chemical Diagnosis.—The separation of lead from organic matter follows the lines indicated under the general scheme described in the section on Chemical Toxicology. Quantities of the order of $\frac{1}{600}$ grain of lead in 4 ounces of organic matter are recognisable with certainty by means of the well-known sulphuretted hydrogen coloration test. But in the absence of much free acid iron

responds and is always present, so that reliance should not be placed on this test alone, but with the formation of the characteristic yellow crystalline lead iodide, the two reactions afford absolute evidence. The precipitation of lead sulphate from the liquid got by heating organic matter with nitric acid is not reliable, calcium sulphate being always present, and, further, is not sufficiently delicate, owing to the solubility of the sulphate in acid and in ammonium salts.

As is to be expected, the quantities found in alimentary contents show very wide variation, as much as $2\frac{1}{2}$ per cent. of red lead having been found, and, on the other hand, as little as $\frac{1}{200}$ grain per 2 ounces in well authenticated cases. The detection of lead in the liver or kidneys, even in small quantities, affords better medico-legal evidence, representing absorbed lead, whereas medicinal doses—*e.g.*, of lead acetate—might easily be recognised in visceral contents, but not in the organs.

REFERENCES TO LEAD.

[1] *Vet. Record*, 1904, p. 559.
[2] *Vet. Record*, 1902, p. 14.
[3] D. Pugh, *Vet. Record*, 1897, p. 383.
[4] J. H. Parker, *Vet. Record*, 1896, p. 178.
[5] G. E. Nash, *Vet. Record*, 1894, p. 398.
[6] A. W. Lawson, *Veterinarian*, 1884, 447.
[7] Tuson, *Veterinarian*, 1865, pp. 6, 217, 423.
[8] A. Broad, *Veterinarian*, 1865, p. 222.
[9] 'Case of Lead Poisoning,' *Veterinarian*, 1855, p. 609

MERCURY.

Forms and Occurrence.—The most important soluble compound of mercury is the *bichloride* or *corrosive sublimate*, one of the most powerfully corrosive and bactericidal of the salts of the heavy metals. It is not often the cause of accidental poisoning. The *mercurous chloride* or *calomel*, being insoluble, is non-toxic—save in large

doses, or when elimination by purgation does not occur—and non-irritant, and is one of the commonest medicinal forms of mercury. *Metallic mercury*, which is harmless in large globules, is absorbed when in the finely divided form, and is thus extensively employed in such preparations as mercury with chalk, and in various mercurial ointments. Finely divided mercury is also sometimes incorporated with oil of tar and mineral oils in mange dressings. The sparingly soluble red *iodide* or *biniodide* also finds application as an ointment. As lotions, suspensions of the black *mercurous* and yellow *mercuric oxides* in lime water are used, and *zinc mercuric cyanide* is a powerfully antiseptic, non-irritant agent.

Ammoniated mercury or *white precipitate*, formed by acting on mercuric chloride with ammonia, is a non-irritant used as a dressing.

Absorption and Elimination.—The finely divided metal, as well as the soluble salts, is absorbed through the skin. Thus Fröhner* records a case of poisoning by absorption from blue ointment.

When finely divided mercury has access to herbage it may be eaten as such, or possibly may be converted into the oxide, as in the case quoted by Lander.†

In the stomach the soluble salts of mercury come into rapid and intimate contact with the tissues, and thus exercise the powerful corrosive effects due no doubt in part to the acid ion, though chiefly to that of mercury. The mercury albuminates, being readily soluble both in proteins and in sodium chloride, cause the drug to penetrate deeply into the tissues, and to pass into the circulation in the form of albuminate.

The metal thus becomes distributed throughout the body, and is stored mainly in the kidney and liver. It is eliminated from the organs by most of the excretory channels, chiefly through the intestines and kidneys. The elimination is in all cases very slow. For the most part calomel is converted in the intestines into the black sulphide, and

* *Vet. Jl.*, 1907, p. 448. † *Ibid.*, 1906, p. 498.

excreted as such in the fæces, very little of this salt being absorbed.

The toxic doses of mercuric chloride by the mouth are given by Kaufmann as—

Horse	120 grains	Sheep	60 grains
Ox	120 „	Dog	3–5 „

Symptoms.—Recorded instances of mercurial poisoning among animals are rare, and the data unfortunately by no means always complete or instructive.

Acute poisoning exhibits the usual sequelæ of nausea and vomiting where possible, with violent diarrhœa and straining, the stools being watery or bloody. These symptoms are followed by collapse, weakness of the pulse, irregular respiration, a subnormal temperature, and death by shock, or if there is survival for several days, acute gastro-enteritis, salivation, irritation of the kidneys, and death from exhaustion.

Characteristic of *mercurialism* is the salivation or ptyalism, the blanching of the membranes of the mouth, and loosening of the teeth, probably caused by the local excretion of mercury. Wallis Hoare points out the danger of using too strong mercurial lotions, and has encountered salivation and even death in dogs after the use of white precipitate ointment. Serious local inflammation of the mouth and eyes with formation of pustules has also been noted by the same authority in horses, caused by rubbing against a part dressed with the biniodide ointment.

In the case observed by Fröhner (*loc. cit.*) a horse was dressed over the ribs, back, and quarters with blue ointment. The symptoms ensued after seven days, when the patient displayed loss of appetite, diarrhœa, profuse nasal discharge, staring coat, pulse 68 and weak, temperature 102½° F. Pustules of 1 inch in diameter formed at the points of application of the ointment, diarrhœa and discharge increased, with extreme debility, depression, and dulness of sensation, followed by death on the ninth day.

In the case observed by Lander (*loc. cit.*) the mercury

had found its way on to the herbage as the result of a fire in an explosive works, but three months elapsed before the cattle began to die; so that here, no doubt, there was chronic poisoning, but, unfortunately, no record was given of the symptoms.

Examples of the toxic effect of calomel are given by Finlay Dun, who observed irritant and general effects in horses by 3 to 4 drachms; in cattle by 2 to 3 drachms; in sheep by 15 to 30 grains; and in dogs by 6 to 30 grains. Such doses cause colic and copious defæcation of green, or in dogs darker, fæces, and if repeated for three or four days fœtid diarrhœa, bad breath, soreness of mouth, loss of appetite and condition, low fever, dysentery, and death. A donkey was killed in sixteen days by fourteen daily doses of 1 drachm, having exhibited salivation, fœtid breath, soreness of the gums, and loss of appetite and general condition. After death the teeth were found to be loose, mucous membranes of mouth and air passages blanched, those of the stomach and intestines softened and covered in parts by mucus.

Post - Mortem Appearances. — Those observed by Fröhner were the inflammation of the mucous membranes, hæmorrhagic enteritis, necrosis, and perforation of the cæcum, peritonitis, catarrhal nephritis, and inflammation of the spleen, and are noteworthy in illustration of the fact that the gastric disturbances due to mercury are also produced when the poison has been absorbed otherwise than by the alimentary tract.

In acute poisoning there is formation of diphtheritic false membranes, particularly in the large intestine.

C. Hirst* has given a somewhat imperfect account of what was probably corrosive sublimate poisoning in the pig, in which rupture and perforation of the stomach and ulceration of the mucous coats were prominent effects.

In the *Veterinary Record*, 1902, p. 27, there will be found an abstract showing the post-mortem appearances observed

* *Veterinarian*, 1862, p. 143.

in the experimental poisoning of cattle with repeated doses of sublimate.

Treatment.—Chemical antidotes are white of egg, which precipitates albuminate, and thus checks the corrosive action. Sulphur, or liver of sulphur, acts by forming the insoluble sulphide. Potassium chlorate tends to counteract the salivation characteristic of chronic mercurialism, whilst potassium iodide is thought to hasten elimination.

When the injury is due to an ointment, the skin should be thoroughly cleansed with warm soda or soap solution.

Chemical Diagnosis.—Mercury is deposited on copper in the well-known Reinsch test, the delicacy being about $\frac{1}{600}$ grain of mercury in 2 ounces of organic matter. Deposition ensues even when one has to deal with mercurial organic compounds, such as must be formed in the liver, for these are broken down by the hydrochloric acid used. Distinct globules, which may be collected and weighed, are obtained on heating the coated copper in a dry tube. If only traces are present, the sublimate on heating the copper should be treated with iodine vapour, when the formation of mercuric iodide, yellow when freshly heated, and gradually changing to scarlet on keeping, affords a most characteristic reaction. Mercury is also separated as the sulphide in the nitric acid process of extraction of organic matter. The sulphide may be dissolved in hydrochloric acid with the addition of a scrap of potassium chlorate, and the solution of mercuric chloride then reduced to insoluble calomel, and eventually to metallic mercury by means of stannous chloride. This is also a delicate reaction, but is less characteristic than the iodide test.

Failure to obtain a positive result with the iodide reaction must be taken as a decisive negative to the question of the presence of mercury.

From the medico-legal standpoint it will have to be established that no mercurial preparation has been given. After perfectly safe and legitimate dosage of calomel, mercury is very easily found, especially in the alimentary

contents. And it must be remembered that it is rarely possible to ascertain the form in which the metal was given.

COPPER.

Forms and Occurrence.—The commonest salt of copper is the *sulphate*, or blue vitriol, or bluestone, which is often used as a dressing for grain against the depredations of birds and as a preservative, and thus may give rise to poisoning.

Copper preparations, such as Bordeaux mixture, are widely used as sprays against parasites of the vine and other fruit-trees.

The effects of *copper arsenite*, or Scheele's green, are more correctly referable to the arsenic than to the copper.

Copper is dissolved by liquids containing organic acids from copper vessels, and thus is sometimes taken up from cooking vessels. Salts of copper are further used to give a green colour to such preserves as pickles, but could not in this way give rise to poisoning among animals.

Copper subacetate, or verdigris, is formed by exposing copper to acetic acid vapour, and is occasionally used in medicine.

Absorption and Elimination.—Copper is not easily absorbed through the intact skin. In the stomach the salts of copper form albuminates, which are soluble in an excess of the albumin solution, and it is therefore absorbed fairly quickly, transported by the blood to the tissues, and deposited chiefly in the liver, lungs, and kidneys.

Elimination by the bile and urine follows very slowly, the metal being stored for several months.

Our laboratory experience indeed satisfies us that copper is normally found in the livers of the domesticated animals. Thus, in the dog it is present to the extent of about 1 in 40,000.

Physiological Effects.—Concentrated solutions, especially of the sulphate and chloride, act as irritants, more

dilute solutions exercising an astringent and antiseptic effect, contracting the capillaries, with arrest of secretions and disinfection of the surface.

Large doses produce amongst animals loss of appetite, nausea, colic, diarrhœa, and fatal gastro-enteritis.

Small doses continued over a long time eventuate in chronic poisoning. Thus, Ellenberger and Hofmeister gave sheep from 7½ to 45 grains of copper sulphate per day, and observed death in periods of from 50 to 114 days.

Baum and Seeliger* similarly experimented on sheep, goats, dogs, and cats, to which cuprohæmol (a compound of copper with hæmoglobin), copper sulphate, copper acetate, and copper oleate were administered over extended periods. They observed great emaciation, weakness, loss of appetite, cramp, and death.

The injection of non-irritant copper salts—*e.g.*, double alkali tartrates and albuminates—induces slow and weak locomotion, and later paralysis, in which the heart and respiration are involved. If the animal survives, violent and bloody diarrhœa, loss of flesh and appetite, albuminuria, icterus and anæmia may ensue.

The poisonous doses quoted by Kaufmann for the horse and ox are 300 grains each of copper sulphate. Dogs withstand daily doses of 10 to 15 grains of copper sulphate, but may succumb under the effects of 40 to 60 grains (Finlay Dun). Fifteen grains of the sulphate injected into the jugular vein of a dog killed in 12 seconds (Christison).

Theiler (see under Arsenic) found that 22 grains of copper sulphate is a safe dose for sheep, 45 grains and upwards causing death from acute gastro-enteritis.

Symptoms of Acute Poisoning.—Recorded instances of acute copper poisoning are rare. A case is given by Reimers† in which four foals (six months), having eaten wheat cured with copper sulphate, were estimated to have received about 9,000 grains, and after twenty-four hours all were ill, and one dead.

The symptoms observed were — sweating, muscular

* *Vet. Record*, 1898, p. 249. † *Vet. Jl.*, 1908, p. 215.

spasms, difficulty in standing and unsteadiness of hind-quarters, vacant look, pulse 105 per minute, temperature 106° F., the conjunctivæ dark red. There was loss of appetite and great thirst, and passage of greenish-yellow fœtid fæces.

In animals capable of vomition, the vomit and purge have a blue to green colour. Abdominal pain, collapse, a weakened pulse and respiration, terminating in coma, convulsions and paralysis, with death from exhaustion, indicate the usual forms of poisoning by corrosive metal salts.

Chronic copper-poisoning is stated to occur amongst animals in the neighbourhood of copper-smelting works. They show increasing emaciation, weakness, and general loss of condition. It is doubtful whether the disorder is not due to arsenic. Analyses of the livers of animals supposed to have died from this cause have been made and failed to reveal a larger proportion of copper than that normally found.

Post-Mortem Appearances.—In the case of the foals quoted by Reimers, the abdomen was greatly distended, and contained about a litre of reddish-yellow serum. The stomach was full of food; mucous membrane inflamed and thickened, that of the small intestines being also thickened and covered with hæmorrhagic patches; the liver enlarged, brownish-yellow, and friable; the spleen enlarged and kidneys congested; the heart was dark red, and covered with hæmorrhagic patches.

The post-mortem appearances of the chronic cases of Baum and Seeliger varied, and showed amongst others chronic catarrh of small intestine, with thickening of mucous membrane and swelling of lymph follicles. The liver and kidneys showed swelling, inflammation, fatty degeneration, atrophy, and necrosis. The organs manifested copious hæmoglobin deposits and subserous hæmorrhages on the heart; in one case there was general icterus.

Treatment.—As chemical antidotes to copper, potassium ferrocyanide, grape or milk sugar, iron filings, sulphur, and animal charcoal have been recommended. Egg albu-

min, milk, and burnt magnesia may be used. Besides elimination of the cause, mucilaginous and stimulating medicines are indicated.

Chemical Diagnosis.—The separation of copper from organic tissues follows the course indicated under the general scheme.

Delicate tests are—the formation of a dark blue liquid with excess of ammonia, and the formation of the reddish-brown ferrocyanide by means of potassium ferrocyanide in the presence of acetic acid. The latter test is of excessive delicacy, showing the presence of copper in insufficient quantity to respond to the ammonia test. From the remarks made above, it is evident that the detection of copper in the liver and kidneys is no evidence of copper poisoning, but its presence in the stomach contents of the herbivoræ, or in the vomit or fæces of a dog will, if the quantity is considerable, point to copper as the particular agent of the observed corrosive poisoning.

ZINC.

Forms and Occurrence.—Although metallic zinc and its compounds are widely encountered, the poisoning of animals by them is a rare event. The *sulphate* (white vitriol) finds use as an emetic, and is liable to be mistaken for Epsom salt, which has the same crystalline appearance. The *chloride* is a very soluble and deliquescent substance, having a powerful corrosive action, and is not given internally. The solution in water is faintly acid in reaction, and constitutes Burnett's fluid, used as a strong disinfectant for unheathly wounds. A concentrated solution of zinc chloride is used in plumbing, and a mixed solution of the chloride with sulphurous acid used to be employed as a disinfectant (Tuson).

Each of these salts is irritant, and causes poisoning. The double salts with potassium or ammonium are less powerfully irritant.

The *oxide* and *carbonate* are extensively used as pigments —zinc white—and in antifouling preparation for ships. They and the salts of weak acids, such as *zinc acetate* and *zinc benzoate*, are astringents, and are used internally in medicine.

Zinc cisterns and galvanised vessels yield zinc to soft water under the same conditions as those governing the solution of lead. It is not to be apprehended that poisoning would arise from this cause.

The metal is also dissolved, probably by means of organic acids, from zinc-lined troughs, and on this account is frequently found in forage and foodstuffs.

In spite of the wide diffusion of zinc compounds poisoning is only likely to occur from the accidental administration of zinc chloride, or of the sulphate. In the latter case dogs and cats promptly reject the dose by vomiting, and under proper treatment a fatal termination is unlikely.

Absorption and Elimination.—The insoluble compounds of zinc are not very easily absorbed, being found only in traces in the organs after lengthy dosage. The greater part of a dose of the oxide is excreted as sulphide in the fæces. The soluble and irritant salts are absorbed, and may be found in the liver, kidneys, and spleen. Elimination takes place chiefly by the kidneys, but zinc is stored, and only slowly eliminated from the liver. Thus, the author found zinc in the liver of a calf which had received 100 grammes of zinc potassium chloride (equivalent to 42 grains of pure zinc chloride) three weeks before death. One ounce of the organ contained $\frac{1}{40}$ grain of zinc; there were traces in the kidney and bile, but it was absent in the spleen.

Toxic Doses.—Half an ounce of zinc sulphate daily for a fortnight gave no marked effect on horses,* though larger doses caused loss of appetite, nausea, and diuresis.

In experiments with zinc potassium chloride the author found that 100 grammes (nearly 4 ounces) caused illness, but were not fatal to a young calf which had already received several smaller doses.

* Finlay Dun, "Veterinary Medicine," 1910, p. 239.

A full-grown sheep was not seriously affected by 20 grammes, but was killed by 60 grammes of the same salt.

When given intravenously zinc sulphate acts rapidly; thus 30 grains depressed the heart's action, and killed a dog in a few seconds (Christison).

Symptoms.—Poisonous doses of zinc salts produce the general symptoms of acute metal poisoning, and are not marked in animals by remote effects.

In the case of the calf above referred to, the administration of 100 grammes of zinc potassium chloride caused at once blowing and distress, the animal lying down. After twenty-four hours the temperature was 98° F., pulse 88 strong, fæces watery, abdomen tucked up, back arched, and there were rigors of fore and hind quarters. The symptoms passed off slowly, the animal remaining in an emaciated condition.

In sheep and pigs the irritant salts produce loss of appetite, frothing, nausea, dulness, and general loss of condition.

Post-Mortem Appearances.—These are of acute gastro-enteritis. A sheep poisoned by zinc potassium chloride showed slight inflammation of the first and third stomachs, but intense croupous inflammation, with fibrinous exudate, of the fourth stomach. The whole of the alimentary contents was very fluid and watery, and there was diffuse but slight inflammation throughout the small and large intestines and cæcum. Kidneys and liver were normal, and the bladder empty. The lungs were highly engorged.

Treatment.—Alkali carbonates tend to render the zinc salts insoluble, and may be given as antidotes. Gastro-enteritis is combated by demulcents. Vomitories or the pump are used to remove the cause.

Chemical Diagnosis.—Zinc is separated from organic matters in the systematic analysis by means of nitric acid. Other metals having been removed or proved to be absent, it is easily recognised by giving the colourless sulphide as a precipitate when ammonium sulphide or sulphuretted hydrogen is added to the ammoniacal solution.

A delicate test consists in the precipitation of colourless

zinc ferrocyanide by means of potassium ferrocyanide from neutral solutions, or in the presence of acetic acid.

As a point of medico-legal value, it may be observed that traces of zinc are very often found in alimentary contents, but must not be taken as indicative of poisoning in the absence of concordant symptoms and lesions. When, however, the metal is found in the liver, there are stronger, though not absolutely conclusive grounds for suspicion.

SILVER.

Forms and Occurrence.—Metallic silver is not important from the standpoint of pharmacy and toxicology. The commonest soluble compound is the *nitrate*, very easily soluble in water, and stable on heating, being fused and cast into sticks for use as *lunar caustic*. The halogen salts (the chloride and bromide) are very extensively used in photography, but are not dangerous. The very dangerous cyanide is also used in photography and largely in silver plating. *Colloidal silver* is the metal reduced in the presence of solutions of colloids, and is soluble. It is used in medicine, and, like the organic salts the *lactate* or *actol*, and *citrate* or *itrol*, does not act as an irritant. An efficacious non-irritant silver preparation is *protargol*, a compound of silver with albumose.

Poisoning by silver is rare, and the acute form follows the administration of large doses of soluble salts. Accidents in the case of the dog may result from the swallowing of a stick of lunar caustic. Smaller and repeated doses give rise to the condition known as *argyria*.

Absorption.—Soluble salts of silver form albuminates like those of the heavy metals, and these are but slowly absorbed. The astringent and caustic actions of the nitrate are thus confined to the parts in contact. In the stomach the salts are decomposed by the hydrochloric acid giving silver chloride, which is very sparingly soluble in acids and water, but slightly dissolved by sodium chloride solution.

The greater part of a dose of silver nitrate is thus rendered unabsorbable, but in contact with organic matter the chloride is reduced, and the silver converted into black silver sulphide, which passes into the fæces.

Nevertheless, some proportion is absorbed, and in the case of prolonged dosage is deposited with blackening of the skin, especially when exposed to light, in the condition known as *argyria*.

Silver is stored in the liver, spleen, pancreas, and bones, and is mainly excreted through the bile.

Toxic Doses.—Dogs are poisoned by from 30 to 60 grains of silver nitrate. The larger animals would doubtless require doses of considerable magnitude, but data on the point are wanting.

Symptoms.—Large doses of silver nitrate cause the symptoms of gastro-enteritis, with vomition of blood-streaked clots in dogs. Great prostration is caused, with weakening of the heart's action, and often paralysis, convulsions, and death from shock.

Chronic poisoning, *argyria* or *argyrism* is often attended in animals by the same blackening of the skin as in man. There is chronic indigestion, loss of appetite, weakness, anæmia, and emaciation.

Post-Mortem Appearances.—Beyond the signs of gastro-enteritis the lesions due to acute silver poisoning are not characteristic. With large doses, and when vomition has not been profuse, flakes of discoloured silver chloride might be noticed. The bowel contents are black from the presence of silver sulphide.

In chronic poisoning there is fatty degeneration as with arsenic, antimony, and phosphorus.

Treatment.—Sodium chloride is a chemical antidote to acute silver poisoning, acting by formation of silver chloride. It must be remembered that silver chloride dissolves slightly in salt solution, so the dose given should not be disproportionately large, and should be diluted. Demulcents and opium should follow the ordinary measures to secure removal of the cause.

Chemical Diagnosis.—Silver is dissolved in the nitric acid extraction process, and separated according to that scheme, as sulphide along with the other metals (*q. v.*). In seeking for silver the sulphides are to be extracted with warm nitric acid (not hydrochloric as in the ordinary routine), and the characteristic tests for silver performed on the solution of the nitrate. The precipitation by hydrochloric acid of flocculent silver chloride, colourless, but blackened on exposure to light, is characteristic. It should be further shown that the chloride is soluble in excess of ammonia, whereby it may be separated from the sparingly soluble lead chloride, which does not dissolve in ammonia. From the ammonia solution silver chloride is reprecipitated by acidifying with nitric acid.

Other tests are also delicate, but most of them not very characteristic. Thus potassium chromate from a neutral solution gives reddish-yellow silver chromate, but confusion with the yellow chromates of lead and barium is possible. Potassium iodide gives yellow silver iodide, which in very small traces is not easily distinguished from other insoluble iodides, such as those of bismuth, copper, and lead.

Phosphates and arsenites give yellow precipitates of the corresponding silver salts, and arseniates give brown silver arseniate. All these are only formed in the absence either of free acid or ammonia, and are of little value in practical toxicology.

Silver also coats copper in Reinsch's test, and on warming the copper with dilute nitric acid both metals dissolve to form the nitrates. Hydrochloric acid precipitates silver chloride from the solution of the mixed nitrates.

BARIUM.

Forms and Occurrence.—Barium is the most toxic of the metals of the alkaline earth series—calcium, strontium, and barium—and cannot replace calcium in its relations to life; for instance, in respect to blood coagulation, and bone

formation. Barium is, however, stated to be deposited in the bones in barium poisoning. Of the salts, the *sulphate*, or *heavy spar*, is insoluble both in water and acids, and is consequently inactive. The *carbonate* is soluble in hydrochloric acid and is therefore converted into the chloride in the acid stomach. It is used as a component of some arsenical rat powders. The *nitrate* and *chlorate* are both soluble, and are used to make green fires in pyrotechny. The *chromate* is used as a yellow pigment, and barium also finds a limited application in glass making. By reason of the great density of the barium compounds, the sulphate is sometimes used to bulk fabrics, and has been found in the coatings of cheeses. But there is no ground for regarding this as likely to cause poisoning. In veterinary therapeutics barium chloride is given intravenously in 8 to 20 grain doses to the horse, or 1½ to 2 or even 3 drachms by the mouth, and 75-gramme drenches to cattle. It is employed in impaction of the colon, and causes violent contraction of the intestine.

Toxic Doses.—Sixty grains prove poisonous to dogs, and horses have been killed by five daily doses of 75 grains of the chloride. These isolated examples must be taken with reserve. By injection far smaller amounts are dangerous.

Symptoms.—When concentrated, the barium salts act as irritants, but are not easily absorbed from the alimentary tract. By whatever channel given, barium acts as a powerful purge, and when possible causes vomiting. There is staggering, loss of control of movements, and difficulty in standing.

Barium acts on the heart like digitalis, the ventricular contractions being slowed, and the heart eventually arrested in systole. Given intravenously, barium causes clonic and tonic convulsions, and the same general symptoms of vomition and purging.

Post-Mortem Appearances are not characteristic. Some inflammation of the stomach is seen after large doses of the soluble salts. Congestion of the lungs, kidneys, and brain will be observed.

Treatment.—Soluble sulphates, such as those of sodium or magnesium, are indicated as chemical antidotes, operating by the formation of the insoluble barium sulphate. Removal of the cause by purgatives, emetics, and the pump is required. When given intravenously the prognosis is grave. The depressant action is combated by stimulant and excitant drugs, and oxygen has been recommended.

Chemical Diagnosis.—Since the sulphate of barium is so very insoluble in acids, the salts of this metal are converted into the sulphate and lost in the residue of the nitric acid extraction process used for lead. Special search may be made for barium in a hydrochloric acid or nitric acid residue, the former being preferable, as nitric acid might oxidise barium sulphide to sulphate. By boiling the clear extract in hydrochloric acid with calcium sulphate solution, or dilute sulphuric acid, a white precipitate of the sulphate is got. This is collected and fused in a crucible with a mixture of potassium and sodium carbonates. After washing with water, the residue of barium carbonate is dissolved in acetic acid and special tests applied, viz., formation of insoluble barium sulphate with calcium sulphate, or sulphuric acid solution; formation of yellow insoluble barium chromate by addition of potassium chromate. To distinguish from lead, the acetic acid solution is shown to give no black sulphide with sulphuretted hydrogen and no insoluble iodide with potassium iodide.

CHROMIUM.

Forms and Occurrence.—*Chromic acid* is a powerful corrosive which destroys all tissues. The corrosive effect is shared to a less extent by the orange *potassium bichromate*, and still less by the yellow *potassium chromate*. The green basic, *chromium oxide*, is stated to be harmless. Several cases of poisoning by potassium bichromate, which is widely used in the arts, have been recorded in man, and the salt appears to operate occasionally less by reason of its

irritant than by reason of its indirect nervous effects. *Chromates* of *lead* and of *barium* are yellow paints very commonly used, and large quantities of chromate are used in the chrome tanning process for leather.

Toxic Dose.—That for the horse is given by Kaufmann as 450 grains, for the dog 45 to 60 grains, but according to a foreign abstract* 300 grains proved fatal to a horse.

Effects.—Chromates are never given internally, but are absorbed through wounds or through the skin, producing dyspnœa, general lowering of the temperature, acceleration of the pulse, convulsive movements, followed by weakness, insensibility, and death. In the case referred to above, 300 grains of potassium bichromate were given to the horse in the morning in mistake for sodium bicarbonate. In view of the fact that bichromate is orange and bicarbonate colourless, this seems a most extraordinary mistake, unless, indeed, the bicarbonate had been coloured by a yellow dye.

There was no appetite in the evening, and on the next day there were observed stiffness, frequent pulse, heart excited and irregular, temperature 101½° F., respiration slow, mucous membranes cyanotic, abdomen painful, intense thirst. Later the breathing became hurried and short, the temperature rose to 102·7° F., and the stiffness passed off; but in spite of treatment the animal died forty hours after ingestion of the poison.

Post-Mortem Appearances.—On post-mortem the conjunctival membranes were found to be covered with hæmorrhagic spots, the buccal membranes having small, shallow ulcers; the mucous membranes of the stomach showed numerous blackish spots, the small intestines were covered with a diphtheritic layer and contained a blood-coloured fluid, as also did the large intestines; the membranes of the lungs, heart, kidneys, bladder, and spleen were destroyed.

Chemical Diagnosis.—The chemical diagnosis is not difficult. In the above-mentioned case no potassium bichromate was found in the intestinal fluid, but it must be

* *Vet. Record*, 1906, p. 290.

remembered that chromic acid and the bichromates suffer reduction to the lower basic oxides in the body.

Salts of chromium are extracted in the nitric acid process from organic matter, and in the subsequent treatment green chromium hydroxide separates along with the iron, and may be recognised by well-known tests, which need not be described here.

IRON.

Iron filings are stated to act as a mechanical poison similar to powdered glass, but such cases are rare, if not entirely absent, from our literature. Nor are there recorded cases of poisoning by *sulphate of iron* (copperas, or green vitriol). This salt is not an active irritant; it produces violent pain, vomiting, and purging in the human subject.

Only a very small proportion of ingested iron is absorbed, and it is extremely doubtful whether this salt would produce death, at any rate in the larger animals. It is now agreed that a small proportion of a dose of an iron preparation is absorbed, probably in the form of albuminate, but the greater part of the material is excreted as iron sulphide in the fæces. When iron albuminate or iron sodium tartrate, which do not coagulate albumin, are injected, poisoning results, but this is a case which does not come within the range of practice. Absorption from the intestines is so slight and so slow that poisoning does not arise when iron is given by the mouth. A case is recorded by Wallis Hoare* of death of cows by iron perchloride. The symptoms noted were dulness, loss of appetite, quick and weak pulse, hurried respiration, cessation of lactation, and obstinate constipation. Administration of magnesium sulphate caused inky-black evacuations. On post-mortem examination the fourth stomach was found to be thickened and perfectly black, with some erosion of the mucous membrane; the intestines were slightly congested, and the contents black. Analysis revealed an abnormal proportion

* *Vet. Record*, 1893, p. 118.

of iron, stated as equivalent to 285 drachms of iron perchloride per 1·5 gallons of liquid contents. As regards this case the effect must be ascribed to the astringent and irritant action of the perchloride on the alimentary system.

PHOSPHORUS.

Chemical Characters.—Of the numerous derivatives of the element, only the '*ordinary*,' free, or white phosphorus, and less frequently the *hydride*, and *hypophosphorous* acid are of significance in toxicology. Poisoning by the very highly toxic *hydride* is not likely to occur outside the laboratory, although, when *calcium carbide* contains traces of *calcium phosphide*, phosphoretted hydrogen is given by the action of water; and, moreover, calcium phosphide itself is a moderately accessible substance.

Ordinary phosphorus forms a waxy solid, of characteristic garlic odour, very inflammable, and oxidised rapidly with emission of a glow in the dark. Red, or amorphous, phosphorus is formed from the ordinary on heating, is insoluble in oils and carbon bisulphide, and is not toxic.

Preparations.—Those most likely to be met with are phosphorus *rat* and *mice pastes*, which are made by incorporating finely divided phosphorus with a suitable grease, and colouring with blue. Although often marked as non-poisonous to cats and dogs, most of the accidents with these animals and fowls are due to them. The grease protects the phosphorus from rapid oxidation and spontaneous ignition, and also facilitates its absorption on ingestion. *Phosphorus matches* are less likely vehicles, and are being rapidly superseded by so-called 'safety matches,' whose heads are free of phosphorus.

Toxic Dose.—Great uncertainty exists as to the toxic dose of phosphorus, and the figures given are to be accepted with reserve. A great deal depends on the state of subdivision, a more finely divided preparation being more easily absorbed. It has, indeed, been stated that coarse particles

are harmless. For the horse and ox, 8 to 32 grains; pig, 2·5 to 5 grains; dog, 0·7 to 1·5 grains; fowl, 0·3 grain.

Absorption and Elimination. — Dissolved or finely divided phosphorus is absorbed as such, absorption being facilitated by the emulsifying action of the alkaline bile. Phosphorus is in part oxidised in the alimentary tract, to which is ascribed its irritant effect. In the blood stream it is carried to the tissues, and is eventually oxidised to phosphoric acid. No positive evidence of the formation of the lower phosphorous acid in the blood has been obtained. It is excreted as phosphates in the urine. The free phosphorus in the blood is also in part given off in the lungs, and causes the exhaled air to smell of phosphorus and to glow in the dark.

Symptoms.—Phosphorus acts as a local irritant on the mucous membranes, but is only slowly absorbed, the onset of symptoms being delayed some hours, and in exceptional cases days, after taking. Uneasiness, nausea, vomiting, and eructation ensue; the vomit, fæcal and urinary excretions may be luminous in the dark, as also may be the breath. There is fever, thirst, and abdominal pain. In the second phase jaundice and nervous effects, delirium, convulsions and coma, precede death, which may not occur until after the lapse of several days. Jaundice is an almost invariable concomitant, being attributable to the enlargement of the liver cells preventing the flow in the bile-ducts.

Slow phosphorus poisoning in the dog might be compared with canine typhus (Stuttgart dog disease; infective gastro-enteritis).

In birds, which are frequent victims of phosphorus poisoning, there is great stupor, the patient being huddled up, beak open, comb blanched; thirst, diarrhœa, convulsions, and coma precede death.

The chronic phosphorism, with its well-known necrosis of the jaw, observed in workers in phosphorus is rare among animals.

Post-Mortem Appearances.—Besides inflammation and

ulceration of the mucous membrances of stomach and intestines, there is to be observed well-marked fatty degeneration, especially of the liver, of the heart, and even of the skeletal muscles. The liver is friable and yellow with occasionally red patches. The bile ducts may be enlarged so as to obstruct the flow of bile and thus cause jaundice. Perforation of the stomach has been observed in the dog after taking phosphorised oil. The alimentary contents are usually liquid and dark brown in colour, and the intestines often heavily charged with bile.

Treatment.—The stomach should be emptied, if necessary, by the tube. As an emetic copper sulphate is used. It may be repeated as an antidote, being supposed to remove the poison in the form of copper phosphide. A well-known antidote is old—that is, oxidised—French turpentine, but doubt has been recently expressed as to its value. Purgation may assist in the elimination of the poison. Oils promote absorption, and must be avoided.

In treating the dog give 3-grain doses of copper sulphate in water every five minutes till vomiting is caused. Thereafter 1-grain doses every quarter of an hour, and if rejected combine this with morphine (Wallis Hoare).

Chemical Diagnosis.—The garlic odour and luminosity of vomits clearly indicate phosphorus poisoning. In the case of the fowl the crop acts as a storehouse of phosphorus, which can be detected (as free phosphorus) after death; but in the dog and cat at the period of death the phosphorus will either have been eliminated or more or less completely oxidised. Material containing free phosphorus is boiled with water acidulated with dilute sulphuric acid, and the vapour is luminous in the dark if phosphorus is present (Mitscherlich). In a case investigated in the laboratory (1911) a sow had died from phosphorus poisoning by having eaten the bodies of fowls similarly poisoned. The ingesta of the sow's stomach contained fragments of fowls, and free phosphorus was present in the proportion of $\frac{1}{6}$ grain per ounce of ingesta. The gizzard of one of the

dead fowls gave ½ grain phosphorus per ounce. After seven days' exposure the material in each case no longer contained free phosphorus. After the phosphorus has been fully oxidised to phosphoric acid its detection is impossible, phosphates being normally present in ingesta and organs. In the lower stages of oxidation the detection is, briefly, as follows: (*a*) Act on the material with zinc and dilute sulphuric acid, passing the gas through silver nitrate solution. This yields a precipitate of silver phosphide. (*b*) Collect the silver phosphide, and introduce it into a hydrogen generating apparatus, passing the hydrogen over solid caustic potash. The flame is green, due to the presence of phosphoretted hydrogen (Dusart and Blondlot).

In this way evidence is easily obtainable. For instance, in a recently observed case no free phosphorus or lower acid was found in the viscera of a dog; and in the dried-up vomit several days after emission no free phosphorus was present, but lower acids were easily recognised.

In medico-legal work no case of phosphorus poisoning can be established unless either free phosphorus is found in the ingesta or vomit, or failing this unless in the same materials the lower acids are detected. The symptoms and lesions must also be consistent with phosphorus poisoning. It is hopeless to attempt to prove that the quantity of phosphate is excessive, phosphates being found in all animal matter, and in the case of the dog in particular in large and variable amount on account of the eating of bones.

AMMONIA.

Forms and Occurrence. — The only compounds of ammonia of significance in toxicology are *free ammonia* and *ammonium carbonate*. Free ammonia is encountered in the form of the solution in water—the so-called ammonium hydrate — or *liquor ammoniæ fortissimum*, of the Pharmacopœia. It contains in concentrated condition

36 per cent. of ammonia, has the specific gravity 0·88, and evolves ammonia gas on exposure to air. Diluted ammonia is a 10 per cent. solution. Ammonia gas can be easily condensed to a liquid by pressure, and is used to produce cold in refrigerating plants by the rapid evaporation of the liquefied gas. Dilute ammonia solutions along with soap form popular cleansing agents and adjuncts to the bath. Along with turpentine or vegetable oils and soap, ammonia, or the carbonate, forms valuable embrocations. Ammonia is present in coal tar liquors and coke oven affluents, but the proportion is not high enough to cause danger. The poisonous effects of such liquors are due to other constituents, chiefly creosote. Mishaps occur sometimes through mistakes in dispensing, strong ammonia being taken instead of ammonium acetate, dilute ammonia, or nitrous ether. Such errors lead to serious results, involving injury to the mouth, pharynx, and œsophagus (Wallis Hoare).

Toxic Doses. — Ammonia and ammonium carbonate, especially the former, possess a high degree of toxicity, although it is not possible to accurately state the doses. Kaufmann gives 480 grains as toxic for the horse, 1,000 for the ox, and 30 for the dog; of the carbonate, for the horse 1,200 grains. Hertwig* found that half an ounce of the strong solution, when diluted, had no bad effect on horses, but that when concentrated 1 ounce killed in sixteen hours, and 3 ounces in fifty minutes. These figures agree with those quoted by Kaufmann.

Effects.—Strong ammonia acts as a *corrosive poison* like the fixed alkalis, destroying the tissues by dehydration, solution of the epidermic and epithelial cells, liquefaction of albumins, and saponification of fats.

Ammonia is readily *absorbed* by the skin, lungs, and mucous membranes, and exerts on all animals a stimulation of the reflexes, marked by general excitation. When injected, ammonia produces general tetanic convulsions, which are absent in a muscle after severance of the motor

* See Dun, 'Veterinary Medicines,' 1911, p. 167.

nerve. In toxic doses coma, insensibility, and paralysis follow the first transient period of violent excitation. Ammonia and its salts are transformed in the liver into urea and excreted as such in the urine.

The corrosive effects of strong ammonia are marked in the mucous membranes of the mouth and pharynx, and in addition the simultaneous inhalation of the gas gives rise to the bronchial disturbances characteristic of it, the swelling of the membranes of the larynx and trachea often causing asphyxia. The tendency to the formation, as a secondary effect, of false membranes is noteworthy.

The effect of ammonia on the blood is first one of deoxidation, followed by dissolution of the corpuscles, and formation of hæmatin from the hæmoglobin. The proteins combine to form soluble ammonia albuminates, and the blood becomes incoagulable, and dark brown or black in colour.

Symptoms.—Recorded instances of death from ammonia poisoning among animals are rare, but reference is made in the *Journal* of 1906, p. 526, to a case observed in Belgium by Verlinde. A steel cylinder of compressed ammonia fell from a dray, and, being smashed, involved the driver and team for some time in an atmosphere heavily charged with ammonia gas. Similar accidents have been recorded amongst operatives of ammonia refrigerating plants. The *symptoms* observed in the horses were—cauterization of the mucous membranes of eyes, nostrils, and cornea; the buccal epithelia exfoliated in large flakes, exposing raw and inflamed surfaces, from which blood exuded;* bloodstained discharge from both nostrils, which were partially obstructed by œdematous swelling; the eyes were closed, eyelids swollen, and tears flowed; respiration laboured and panting. On the next day the observations were—dull, depressed, pulse 60, respirations 26, temperature 39° C., no appetite. Intense photophobia keratitis and muco-purulent conjunctivitis, with profuse yellow discharge mixed with flakes of epithelium

* The writer once experienced the same effects in the laboratory through incautiously sucking ammonia solution from a pipette.

from both nostrils. Frequent short, painful cough and loud, sibilant râles, and dull patches pointed to bronchitis, congestion, and pulmonary œdema.

On the third day in the first case the pupil became visible and appetite improved. About the twelfth day the animal became convalescent, but bronchial complications lasted some time. The second animal was, however, worse, broncho-pneumonia and septicæmia set in, and it was slaughtered on the tenth day.

Post-Mortem Appearances.—In the above cases there were observed patches of purulent broncho-pneumonia; the bronchi of greyish colour, the smaller filled with purulent casts containing débris of mucous membrane. The lower borders of the lungs were particularly implicated.

In a case of poisoning of a dog, observed some years ago, the stomach showed violent inflammation, was distended with gas, and contained brownish bloody fluid. Death had occurred in two days after bloody vomiting at the end, but the symptoms unfortunately were not under expert observation.

Treatment.—Ammonia poisoning is treated by dilute acids, preferably diluted vinegar, as a chemical antidote; demulcents, honey, and belladonna as a stimulant.

Chemical Diagnosis.—The recognition of free ammonia is a matter of great ease from the well-known smell and other properties of that substance. In the above-mentioned case the gas contained in the stomach was ammonia, and 2 ounces of the fluid yielded ½ grain of ammonia on distillation. In seeking for ammonia a precaution is necessary, since volatile bases of ammoniacal odour may be found in putrefaction. The distinction, however, is easy, since ammonium chloride does not fuse on heating, but sublimes, and is insoluble in strong alcohol, properties not shared by the chlorides of the organic bases.

In cases such as those of poisoning by vapour it need hardly be remarked that chemical diagnosis is superfluous and impossible, for long before death, which results as an after effect, the absorbed substance will have been eliminated.

As a point of medico-legal significance, it must be remembered that ammonia is developed in putrefaction. Judgment will therefore need to be exercised in expressing an opinion.

STRONG ACIDS AND ALKALIS.

Effects.—The effects of concentrated alkalis (caustic soda and caustic potash) and acids (sulphuric, nitric, and hydrochloric) are due to the profound chemical action which they exercise upon the living tissue, leading to its destruction, to intense corrosion, and local lesion, followed by vomition, colic, purgation, exhaustion, and death by shock.

All these agents attract water, and thus act as powerful dehydrants, a property which is, however, particularly characteristic of strong sulphuric acid. The alkalis, further, decompose fats and proteins; whilst the acids—in particular nitric acid—coagulate the latter.

Corrosion of the mucous membranes of the mouth, tongue, and pharynx will be observed.

When strong sulphuric acid is swallowed it causes retching, and, if possible, vomition of bloodstained matter, with shreds of mucus. The local irritation and swelling of the lips, tongue, fauces, and throat, are very marked, and cause suffocation.

Characteristic of nitric acid is the intense yellow coloration of the epithelium, which has been known in man to extend through the whole of the alimentary tract.

Dilute sulphuric or other acid is often given to horses to improve condition. This it fails to do, and an overdose causes acute poisoning. If this treatment is persisted in, chronic disturbance of the digestion, with serious loss of condition and general health, results.

Concentrated alkali (caustic potash or caustic soda) destroys the membranes, and sometimes causes perforation. According to Hertwig, 2 drachms of caustic potash in 6 ounces of water killed a horse in thirty-six hours, and

Orfila observed the death of a dog in three days after having 32 grains.

Treatment. — The treatment of alkali poisoning is by means of very dilute acids, preferably vinegar, followed by fatty oils, and later sedative or stimulating agents; whilst in acid poisoning dilute alkali—*e.g.*, chalk, burnt magnesia, soap solution, weak soda solution—is indicated, and later mucilaginous and stimulating agents.

Cases of poisoning by these agents are rare, and should they occur offer little difficulty in diagnosis and treatment.

The question of alkali contamination of water may from time to time demand attention. In a recent legal case it was held to have been proved that a stream had been contaminated with caustic soda, but there could be no doubt that the death of a heifer which had access to the stream was certainly not due to caustic alkali.

An extremely interesting case of poisoning by hydrochloric acid gas is reported by R. J. Stordy.* The fumes, which were of volcanic origin, emanated from a small hole in a gully in the Kelong Valley, Africa. Around the hole were the carcasses of buffaloes. Fowls tied near the hole were rapidly affected, whilst a man who put his head in it was seized with a severe headache and vomiting. Sheep, dogs, and cows placed near the hole immediately began to heave violently, collapse, and die—the sheep in a few seconds, the cows within half a minute. Inhalation of ammonia assisted resuscitation immediately after the first collapse. The gas proved to be that of hydrochloric acid extending at a maximum height of 18 inches from 20 to 30 feet of ground round the fissure.

Post-mortem revealed dark coloured difficultly coagulable blood, and the heart in all cases arrested in diastole.

Chemical Diagnosis.—Valuable indications are afforded in nitric acid poisoning by the yellow colour imparted to the tissues, which may, however, be marked by blood. The reaction of the contents to test papers is a valuable guide, and the degree of alkalinity or acidity should be

* *Jl. Comp. Path.*, 1908, p. 75.

determined and compared with that of the normally alkaline rumen or intestine content and the normally acid stomach content respectively. To separate the acids or alkalis from organic matter the process of dialysis through a parchment membrane with water should be used. The suspected compound may then be tested for in the purified dialysis liquid.

COMMON SALT.

Toxic Doses.—It is generally accepted that large doses of sodium chloride, or common salt, may lead to poisoning, and it is stated that from 4·5 to 7 pounds may prove poisonous to cattle; from 2 to 4·5 pounds to the horse; and from 4 to 8 ounces to the sheep and pig.

A pig had five daily doses of 1 ounce, six of 2 ounces, and six of 3 ounces of salt consecutively, mixed with its food. Although the 3-ounce doses were not eaten readily, no abnormal effects were observed (Lander).

It is only, however, in respect to the pig that salt poisoning assumes importance, and in our literature there are numerous references to poisoning of that animal by this agent.

Sources.—Sometimes the vehicle of poisoning is solid salt—*e.g.*, from salt trucks—but more often the poisoning is due to the partaking of liquors from the salting or boiling of meat or from salted potatoes.

Arising no doubt from the general acceptance of salt poisoning as liable to affect pigs, there is a widespread idea that household waste liquors of all sorts may kill these animals. In a very large number of alleged salt or soda cases, analysis of the stomach contents fails to reveal an excess either of sodium chloride or of carbonate.

It is not possible to state the doses of salt necessary to kill the pig, and some recorded examples are not free from doubt; thus it is not unlikely that brines may contain organic poisons derived from the decomposition of proteins; and in salted potato cases there is the possibility of poisoning

by the alkaloid solanine contained in diseased potato. Nor is the mechanism of salt poisoning well understood, for, apart from the gastric disturbances naturally to be anticipated, there appear to be definite nervous effects.

All concentrated solutions of salts when taken into the stomach and intestines cause an increased inward flow of water from the surrounding serum by reason of osmosis. This may set up irritation and vomiting. There can be no doubt that very large doses of many salts can produce dangerous or even toxic effects, although the salt in question has no specific action.

Whilst, however, the question of salt poisoning demands exact study, the actual records are sufficient to establish its existence as a matter of practical fact.

Brine poisoning is more obscure, since that fluid contains other constituents than salt. Herring brine is held responsible for some cases of poisoning abroad. Besides salt it contains nitre and the volatile toxic base trimethylamine. Since its toxicity, at first great, diminishes with keeping, it is probable that bacterial action occurs with production of toxic substances, and the nervous symptoms are more marked in its action than with salt.

Symptoms.—From the recorded instances the general symptoms of salt poisoning in *pigs*[2,5,6,7,10] involve loss of appetite, thirst, champing and salivation; in some instances diarrhœa, and in others scanty, hard fæces; the animals sit like dogs on their hind legs, and then roll over on to their sides; there are vertigo and convulsive movements, dilatation of the pupils, and blindness; the convulsions increase in frequency, and the subject loses power over the hind quarters; temperature normal, ears and skin cold; death takes place in convulsions. The onset of symptoms is rapid, and death occurs within three days.

Suffran[1] describes a case in which thirteen out of fifteen *fowls* died after eating a salted potato mash—the symptoms setting in within twelve hours. The birds fell from their perches, showed signs of great thirst and weakness, and there was a viscous discharge from the beak. Experi-

mental poisoning of fowls by the same author showed the dose to be 4 grammes per kilo body-weight by the injection of salt solution into the crop, and the symptoms observed appeared to show a special action on the muscles, illustrated by progressive difficulty in walking, with eventual inability to stand. But there was no true paralysis, because the muscles still exhibited reflex action. Death was attributed to asphyxia, due to loss of power of the respiratory muscles.

The symptoms of somnolence, hyperæsthesia, and vertigo seem further to indicate an action upon the nervous centres.

In the *Veterinarian*[4] is an abstract from the German relating to the deaths of *cattle*.

Twelve kilos (about 27 pounds) of salt dissolved in water was given to seven cattle, and there was observed agitation, loss of appetite, suspended rumination, thirst, a slight increase of pulse, normal evacuations. On the next day the animals were recumbent; head and back legs extended; foaming, body cold, moaning, moderate tympanites; evacuations liquid, greenish, and mixed with mucus and blood. Death occurred within twenty-one to twenty-four hours.

Since the practice of giving large doses of salt to cattle in the treatment of red water has come in, dangerous effects of depression, abdominal pain, extreme thirst, and often collapse have been observed after doses of 1 to 1½ pounds, especially if not well diluted (Wallis Hoare).

Post-Mortem Appearances.—General inflammation of the mucous membranes of the stomach and intestines is to be observed, and injection of the cerebral membranes appears to be characteristic. The blood[8] is dull red, rapidly coagulating, the clot separating easily from a whitish slimy serum.

Treatment.—In a case observed by Sir Charles Cameron[3] a cure was effected by means of emetics and stimulants. Demulcents, with opium and stimulants, such as camphor and ether, against the general weakness, are indicated.

Cerebral symptoms may be treated with bromide or chloral hydrate.

Chemical Diagnosis. — Salt may be separated from organic matter most readily by diffusion or dialysis. Cameron found 3 grains of sodium chloride in a teaspoonful of the semi-fluid contents of the stomach, whilst Herapath[9] found in similar contents 42 grains to the pint. In the case of the fowls[1] salt formed 14 per cent. of the crop contents. As is to be expected, these proportions are very high, and they show that the chemical diagnosis of salt poisoning is a matter presenting little difficulty, even allowing for the fact that sodium chloride is a normal constituent of the body fluids.

REFERENCES TO SALT.

1 Suffran, *Jl. Comp. Path.*, 1910, p. 71.
2 Lamoureux, *Vet. Jl.*, 1890, p. 116.
3 C. A. Cameron, *Veterinarian*, 1871, p. 883.
4 Abstract, *Veterinarian*, 1870, p. 635.
5 H. Pyatt, *Veterinarian*, 1862, p. 768.
6 T. Gregory, *Veterinarian*, 1859, p. 251.
7 W. Robinson, *Veterinarian*, 1859, p. 124.
8 Reynal, *Veterinarian*, 1856, p. 356.
9 Lewis, *Veterinarian*, 1856, p. 518.
10 G. H. Lepper, *Veterinarian*, 1856, p. 434.

NITRATES.

Sources and Doses.—The nitrates of sodium and of potassium have both been known to give rise to poisoning, the general character of which is not unlike that of salt poisoning, and attention was draw to the question at an early date.[5,6] Sodium nitrate is very commonly used in the form of Chili saltpetre as a manure, and may be confused with Glauber salt (or sodium sulphate). The toxic doses of the nitrates are, for large animals, at least 8 ounces. Prominence must, however, be given to the observations of S. M. Smith,[1] who gave potassium nitrate to two bullocks in 8-ounce doses per diem, increased

to 10 ounces twice, and then to 12 ounces three times, without ill effects. Against this is to be set the occurrence of several well-authenticated cases in the literature, and in 1909 a case was brought under the writer's notice in which three bullocks died after having had 1 pound each of a mixture containing 80 per cent. of potassium nitrate and 20 per cent. of magnesium sulphate. Wallis Hoare communicates a similar case, in which potassium nitrate was sold in mistake for Epsom salt. Four cows having a pound each died. As with salt, the question of nitre poisoning merits further research.

Symptoms.—The general symptoms observed are gastro-intestinal irritation, trembling, convulsions, tetanic convulsions, dilatation of the pupil, and paralysis of the voluntary movements.

In a case observed in Germany[3] three cows had had 15 ounces of sodium nitrate, and there was observed agitation, trembling, salivation, loss of power, protruding tongue, and eyes turned in orbits.

In the cases observed by Wallis Hoare (*v. supra*) there was violent pain, collapse, tympanites, but no purging.

Batchelder[4] records the death of 130 out of 226 lambs which had each had 1 ounce of sodium sulphate and 1 ounce of potassium nitrate.

Post-Mortem Appearances.—There are observed irritation of the digestive and urino-genital channels; the venous blood is red and not coagulated. In one case[2] there was no evidence of disease, but an immense quantity of water in the digestive organs.

In Wallis Hoare's cases the abomasum was inflamed and of claret colour, small intestines injected, and kidneys congested.

The **Treatment** should consist of measures similar to those adopted in the case of salt.

The **Chemical Diagnosis** also follows the principles described under Salt. After evaporation of the dialysed fluid, sodium or potassium nitrate may be easily recognized

and estimated by the aid of the numerous and well-known tests for those substances.

REFERENCES TO NITRATES.

[1] S. M. Smith, *Vet. Record*, 1898, p. 85.
[2] Abstract, *Vet. Jl.*, 1892, p. 97.
[3] Abstract, *Veterinarian*, 1870, p. 635.
[4] Batchelder, *Veterinarian*, 1869, p. 98.
[5] Surginson, *Veterinarian*, 1838, p. 85.
[6] Saussol, *Veterinarian*, 1836, p. 530.

SULPHUR.

In large doses sulphur is poisonous, cases of death in the horse and dog being on record.

Impure sulphur is liable to contain arsenic and free sulphuric acid; but the poisoning is not to be attributed to these, for in one case sulphur which had caused the death of horses was proved by analysis to be free of these impurities.

Free sulphur is unacted upon by the acid gastric juices, but in the intestines it is to a small extent converted into the soluble and absorbable alkali sulphides, and is further reduced to sulphuretted hydrogen, which enters into the circulation, so that not only is sulphuretted hydrogen given off *per rectum*, but also may be detected in the exhaled air.

It is to be remarked that sulphuretted hydrogen is intensely poisonous, an atmosphere containing 1 per 1,000 of that gas being held to be rapidly fatal to man.

The formation and absorption of these products no doubt accounts for the nervous effects of sulphur poisoning, which, for the rest, is characterized by the production of superpurgation and its attendant weakness and collapse.

Toxic Doses.—The purgative dose for the horse and ox is given by Kaufmann as from 7 to 14 ounces. In one case[1] horses were killed by 8 ounces each, in a second[2] by 10 ounces each, and in a third[4] by 10 to 14 ounces each.

Symptoms.—The onset of symptoms is fairly rapid—from three to eight hours after dosage. In the *horse*, dulness, pain, and diarrhœa, with black or grey liquid fæces smelling of sulphuretted hydrogen, and highly coloured acid urine, sometimes albuminous, have been observed. The conjunctivæ and other mucous membranes are injected, the respiration rapid and laboured, pulse weak and rapid, temperature 104° F., extremities cold; great depression and tottering gait are followed by death within a few hours, or gradual recovery under treatment.

In a case of a *dog*,[3] eight hours after administration of a large unknown dose of flowers of sulphur, there was violent colic, diarrhœa, and vomition, the ejections being at first bloody, and afterwards consisting of pure blood; pulse almost imperceptible and coma. In this case there was recovery after twenty-four hours.

Post-Mortem Appearances.—Intense inflammation of the gastric and intestinal mucous membranes, which are sometimes gangrenous. The blood is dark coloured and fluid; viscera and lungs engorged; fibrinous clots in the portal venous system, spleen, and liver; all the tissues and fluids of the body smell strongly of sulphuretted hydrogen. Particles of sulphur may be noticed in the stomach, intestines, and fæces.

Treatment.—In one case[1] castor oil, eggs and milk, chlorodyne and whisky, were successful in curing horses so treated. In another case[2] milk, flour, gruel, white of eggs, subnitrate of bismuth, and rice water enemata, were employed.

Chemical Diagnosis.—Particles of sulphur may be recognised by inspection. If no such particles are found, the material is thoroughly dried in the steam oven, extracted with carbon bisulphide, and the sulphur recovered by evaporation of the filtered solution. In this way sulphur in the proportion of $2\frac{1}{2}$ grains per ounce and $2\frac{1}{3}$ grains per ounce has been recovered from the stomach and colon contents respectively of a horse. It may also be mentioned that the extraction of sulphur is frequently a valuable guide

as to the sources of arsenic (from sheep dip) or antimony (from condition powders) in cases of poisoning by those agents.

REFERENCES TO SULPHUR.

[1] H. W. Percy, *Vet. Jl.*, 1910, p. 29.
[2] Ales, *Vet. Jl.*, 1907, p. 254.
[3] M. Hebrant, *Vet. Record*, 1900, p. 167.
[4] Mosselman and Hebrant, *Vet. Record*, 1898, p. 249.

HALOGEN ELEMENTS AND THEIR COMPOUNDS.

Forms and Occurrence.—The halogen elements (excluding fluorine, which has no special interest to practical toxicology)—namely, chlorine, bromine, and iodine—though not found in the free condition in nature, are employed in the arts to a limited extent, and free iodine is a valuable drug. Chlorine and bromine rank as amongst the most chemically active elements, and are both, in the state of vapour and diluted with air, most powerful disinfectant agents. In Germany stable disinfection by evaporation of bromine is practised. Neither the gaseous chlorine nor the volatile liquid bromine is encountered outside special work, and both are most dangerous substances to handle. Liquid bromine has a most powerful corrosive action, destroying the skin and mucous surfaces, and causing wounds which heal with difficulty. The solutions in water—viz., *chlorine water* and *bromine water*—find some application in pharmacy as caustic disinfectants.

Bleaching powder (*chloride of lime* or *calcium chlorohypochlorite*) and *sodium hypochlorite* (or *eau de Javelle*), the so-called chlorinated alkalis, owe their efficiency to the ease with which they part with chlorine, and thus effect oxidation. Bleaching powder is given internally to the horse and ox in doses of 450 and 750 grains respectively, and in proportionately smaller amounts to sheep. It must be remembered that contact with acids, even when very weak and dilute, generates free chlorine, and large doses are therefore liable to cause injury.

The simple salts, such as *sodium chloride* and *potassium bromide* and *iodide*, are common in use, and have great therapeutic value. All are liable to cause dangerous symptoms in large doses.

The oxygenated salts, *potassium* and *sodium chlorates*, *bromates*, and *iodates*, are increasingly toxic in the order named, and it is very probable that the presence of iodate in Chili saltpetre accounts in part for the toxicity of that substance (see Nitrates).

Symptoms.—Poisoning by the above-named substances is rare, and a brief summary of the general effects of the agents named will therefore suffice.

Chlorine and *bromine*, in the condition of concentrated vapour, are very dangerous, and primarily attack the respiratory system. They cause intense pulmonary irritation by directly attacking the mucous membranes. There is suffocation, cough, violent retching, and discharge of bloody mucus. In fatal cases a comatose condition precedes death. The pulmonary troubles may eventuate in broncho-pneumonia, and even with recovery there is great loss of condition and resistant power.

Treatment must include immediate removal of the cause. Small quantities of sulphuretted hydrogen or ammonia act as direct chemical antidotes against both chlorine and bromine. Vapour of alcohol or ether is, however, preferable, as the above agents are also themselves very toxic.

Chemical Diagnosis will not be possible if any length of time elapses before death, and, indeed, is scarcely necessary, since there can be little doubt as to the cause. The faint odour of chlorine or bromine may be observed. To test chemically the parts are distilled into water, and the halogen recognised in the watery distillate. Bromine imparts a brown or orange colour to water, and extraction with chloroform gives a heavy orange-coloured lower layer. Both agents liberate iodine from solutions of iodides, such as potassium iodide, and this gives a blue colour, discharged on heating and reappearing on cooling, with dilute starch solution. The test is not characteristic of chlorine and

bromine, since many substances—*e.g.*, nitrous fumes and hydrogen peroxide—also liberate iodine from iodides.

When given internally *bromine* is very poisonous. According to Law, 120 grains killed a dog in five hours; 10 to 12 drops in an ounce of water intravenously proved suddenly fatal (Orfila). *Iodine* in doses of 5 to 6 drachms by the mouth killed dogs in a few days (Orfila), a half-ounce of iodine caused colic in a horse (Tabourin), and 2 drachms intravenously killed (Patu). Hertwig gave horses 40 to 60 grains of iodine twice daily for fourteen days, without causing death, and larger doses of several ounces have failed to injure cattle.

Very large doses of these agents cause acute abdominal pain, diarrhœa, which has the bromine or iodine odour, general weakness, vertigo, and convulsions.

Iodism, following protracted administration of full doses, is marked by catarrh of the nostrils, throat, and alimentary organs, suppressed urination, weakness, emaciation, scaly skin eruptions, and the hair falls off in patches.

Boiled starch is recommended as an antidote in fixing free iodine, but is of doubtful value, and certainly useless, apart from its demulcent effect, for bromine.

Bromides of sodium and potassium, which closely resemble common salt in appearance, are more dangerous than it (see Common Salt). But the doses required to exercise toxic effects are very large, and not likely to be the cause of accidental poisoning. There is so great a difference in the cost of the iodides and bromides as compared with chlorides that risk of substitution of them for chloride is very greatly minimised. One ounce of bromide to a horse causes listlessness, muscular feebleness, unsteadiness of gait, impaired reflex movements; the pulse is feeble, respiration slowed, rectal and cutaneous temperatures are diminished, and secretion of urine increased. Dogs display similar symptoms after 45 grains (Finlay Dun). The vomition, diarrhœa, and diuresis are general effects of salt administration.

Bromism, with cerebral depression, increased secretion,

anæmia, weakness, and eczematous eruptions, results from prolonged administration of bromides.

Like the bromides, large doses of *iodides* cause enfeeblement of the heart and cerebral and spinal depression, prolonged exhibition causes *iodism*, and large doses are fatal to dogs, which show depression and gastric irritation due to salt administration (Finlay Dun). But large doses of iodide are given to horses and cattle. Thus, in actinomycosis of cattle 2 drachms twice daily cause no toxic effects even after prolonged treatment, and in obstinate cases 2 drachms potassium iodide with 5 grains mercury biniodide twice daily for several weeks are used (Wallis Hoare).

Iodoform, so valuable as an antiseptic dressing, is rarely given internally. Not being easily absorbed from the skin, it does not often give rise to poisoning; but this may happen with dogs through licking. In dogs and cats it causes gastric irritation, vomiting, muscular spasms, a lowered temperature, enfeebled heart action, and narcosis. There is albuminuria, and in chronic poisoning emaciation and fatty degeneration of muscles and glands. According to Fröhner, dogs are killed by 15 grains per 2 pounds body weight by the mouth, by 20 to 30 grains subcutaneously, or 7 grains injected into a serous cavity. An old cow died in thirty-six hours with spasms and narcosis after an ounce and a half (Finlay Dun).

In any case of poisoning by iodoform no difficulty ought to be experienced in diagnosis, because the drug will be known to have been accessible, the odour is characteristic, and the symptoms are in accordance.

CARBON MONOXIDE.

Occurrence.—Carbon monoxide, or carbonic oxide, is to be clearly distinguished from carbon dioxide, or carbonic acid. It is inflammable and chemically neutral. Carbon monoxide is formed in the incomplete combustion of carbon,

and is contained in 'producer gas' to the extent of about 23 per cent., in 'water gas' about 44 per cent., in 'Dowson gas' 24 per cent., in 'coal gas' from 6 to 11 per cent., and is further also formed in the explosion without complete combustion of fire-damp in mines, and is thus a constituent of choke-damp.

Toxicity.—Estimates as to the toxic proportion of carbon monoxide in air vary, but according to some authorities (Gruber and Hempel) it is less than 1 per cent. On this account, and in consideration of the numerous technical and domestic employments of the above-named gases, a description of carbon monoxide poisoning is desirable. But recorded cases of death amongst animals are very rare.

Absorption and Effects.—Carbon monoxide is rapidly absorbed by the pulmonary mucosa, and passes into the blood stream in the form of carboxyhæmoglobin, a combination of the monoxide with hæmoglobin, which is more stable than the oxygen compound (oxyhæmoglobin). It is therefore an exceedingly toxic compound, since the stability of the carboxyhæmoglobin prevents oxygenation of the blood, and poisoning in an atmosphere containing over 3 per cent. is almost instantaneous.

In smaller proportions there is vertigo, and loss of power and muscular tremors. Respiration is difficult, rapid, and stertorous. The heart's action is intermittent, power over the sphincters is lost, and death ensues in coma.

Post-Mortem Appearances.—The most notable post-mortem appearance is the bright redness of the blood, which gives a pink or violet froth, but the lesions are not characteristic.

Treatment in suspected cases such as of coal gas or choke-damp poisoning will take the form of inhalation of oxygen, in the hope of dissociating the carboxy-hæmoglobin, by means of excess of oxygen. Electrical treatment, the positive pole in the rectum and the negative in the mouth, has been recommended as valuable for small animals.

Chemical Diagnosis.—The best test for carbon monoxide

depends on the character of the absorption spectrum of carbon monoxide blood, which may be observed in a dilution of 1 of defibrinated blood in 1,000. This test may therefore be applied to blood post-mortem, or used for air by agitating the suspected sample with normal defibrinated blood of the above strength. The absorption spectrum of carboxyhæmoglobin is similar to that of oxyhæmoglobin, viz., two bands in the yellow, but displaced a little to the right. The difference is in the fact that reducing agents such as ferrous salts, but better ammonium sulphide, reduce oxyhæmoglobin, with fusion of the two bands into one, larger and indistinctly outlined, whilst reduction does not alter the carbon monoxide blood spectrum.

A chemical method of detection consists in passing the suspected gas over gently warmed pure iodine pentoxide. Even with very small proportions of carbon monoxide iodine is liberated which is absorbed in potassium iodide solution, and recognised by the starch or other of the well-known tests for iodine. The reaction is not absolutely conclusive, since such hydrocarbons as acetylene (which is also present in coal gas) behave similarly.

ORGANIC POISONS AND DRUGS

HYDROCYANIC OR PRUSSIC ACID.

Occurrence.—Hydrocyanic acid is one of the most powerful known poisons, and poisoning by it may arise not only through the use of the acid and its salts in the arts—*e.g.*, in the cyanide gold extraction processes, in electroplating, and in pharmacy—but also because hydrocyanic acid is, under certain circumstances, generated from many plants. In the vegetable kingdom hydrocyanic acid occurs in combination in the form of cyanogenetic, or cyanide-producing, glucosides.

A glucoside is an organic compound of vegetable origin which, by hydrolysis with dilute mineral acids, or by the agency of certain vegetable enzymes, is decomposed, yielding always sugars and at the same time other compounds. *Amygdalin* $C_{20}H_{27}NO_{11}$, the glucoside of the bitter almond, is an excellent typical example. The almond seed also contains the enzyme or ferment *emulsin*. On macerating the seed with water the emulsin is brought into contact with the dissolved glucoside, and causes the decomposition represented by the equation $C_{20}H_{27}NO_{11} + 2H_2O = 2C_6H_{12}O_6 + C_6H_5CHO + HCN$—*i.e.*, the amygdalin yields glucose, benzaldehyde (or oil of bitter almonds), and hydrocyanic acid. In smaller proportions amygdalin is also found in the peach, plum, cherry, and apple seeds, and the formation of the poisonous prussic acid from such sources appears to have been known to the early Egyptian priests. The cherry laurel—*Prunus laurocerasus*—one of the commonest ornamental shrubs, contains amygdalin in the leaves, and gives

rise sometimes to poisoning. In the South-Eastern United States the *Prunus caroliniana*, laurel cherry, or mock orange, which is cultivated as a hedge, and *P. serotina*, or wild black cherry, an Eastern forest tree, both similarly have proved dangerous. In South Africa *Dichapetalum cymosum*, gift-blaar or poison leaf, is a most dangerous cyanogenetic plant, having caused losses in the Transvaal, Bechuanaland, and Rhodesia. J. T. Dumphy[9] studied the effects on sheep, and states that 1½ ounces of the leaves is sometimes, and 2 ounces always, fatal to them. They eat it if kept starved, but once having been affected and recovered they refuse it.

In addition to the above mentioned, many of the *Leguminosæ*, in particular *Phaseolus lunatus* (Java or Rangoon bean, Haricot de Lime), and species of vetch (*Vicia*); certain of the *Gramineæ*—*e.g.*, the millet, or sorghum, and the maize; and, amongst the *Linaceæ*, common flax, *Linum usitatissimum* and its varieties (*L. catharticum*, etc.) also contain cyanogenetic glucosides, such as phaseolunatin (or linamarin), which yields on fermentation glucose, acetone, and hydrocyanic acid.

The quantity of this glucoside, which is unevenly distributed throughout the whole of the plant, varies widely; thus it is present in sufficient quantity in the young millet to cause poisoning, but is absent or in insufficient quantity to prove dangerous in the mature plant. Losses of British horses by the eating of young millet occurred in our first Egyptian campaign. In the *Phaseolus* (or Java bean) the percentage of glucoside in the wild East Indian varieties, which vary in colour from pale reddish-brown to purple, amounts to rather more than 0·1 per cent.[6]

The white beans of the same species contain only about one-tenth part of this proportion, and in the cultivated varieties the proportion is very small, clearly pointing to the fact that the production of the glucoside is a means of natural defence. From the practical point of view only the dark coloured, and especially the purple, beans are to be regarded with suspicion. In 1906 considerable poisoning

took place through the importation of the wild *Phaseolus* from the East under the name of Java or Rangoon beans.

As regards the mechanism of poisoning by these materials, it is necessary to emphasise several important points:

1. In the dried material the enzyme and glucoside are not in contact, and therefore no fermentation with formation of hydrocyanic acid occurs.

2. After mastication the pulped mass at the body temperature is in a most favourable condition for fermentation, and the disengagement of hydrocyanic acid is therefore rapid in the rumen or stomach.

3. A moist heat of over 60° C. destroys the enzymes, but it must be remembered that their destruction by dry heat requires prolonged heating at at least 100° C., the cyanogenetic glucosides in themselves neither appearing to be particularly poisonous nor to be acted upon by the various digestive ferments.*

In spite of hot pressing, which would be expected to destroy the enzyme, the great majority of linseed cakes still contain active enzymes. Linseed cake rarely, however, yields more than 0·025 per cent. of hydrocyanic acid, although as high a percentage as 0·055 has been observed. It will therefore be only under exceptional conditions that linseed can prove poisonous, as has been shown by feeding experiments.[7]

As regards the preparations likely to be encountered in pharmacy and the arts, the B.P. hydrocyanic acid is a 2 per cent. and Scheele's acid is a solution of from 4 to 5 per cent., whilst the very volatile and exceedingly poisonous anhydrous acid is never encountered outside the laboratory.

Potassium cyanide is the commonest salt, and is also very poisonous. It is a colourless crystalline solid, having a faint smell of prussic acid, and yielding an alkaline solution in water.

Potassium cyanide appears to be slightly less toxic than

* In McCall's experiments (*loc. cit.*) meal of Java beans appears to have poisoned even after one hour's boiling.

the equivalent quantity of the acid—at any rate, on intrathoracic injection; but this difference is probably due to the fact that potassium cyanide solution does not disengage prussic acid vapour, and is therefore less rapidly absorbed by the pulmonary system. By the acid gastric juices hydrocyanic acid is, however, readily liberated from potassium cyanide.

It must also be remembered that it is the cyanogen acid radicle or ion CN which is poisonous, and therefore the complex cyanides, *e.g.*, potassium ferrocyanide, and the sulphocyanides, *e.g.*, KCNS, are practically harmless.

Notable also is mercuric cyanide, $Hg(CN)_2$, which is not easily dissociated—*i.e.*, does not readily yield hydrocyanic acid, and is therefore no more toxic than mercuric chloride.

Toxic Doses.—Kaufmann quotes for the horse 6 grains in the form of the 2 per cent. solution, and 0·6 grain similarly for the dog. Finlay Dun states similarly that 4 to 5 drachms of the 2 per cent. acid (equivalent to 6 grains pure acid) may kill a horse in an hour. As regards potassium cyanide, Kaufmann gives the dose for the horse as 60 to 120 grains (equivalent to 25 to 50 grains of pure acid). For the dog he gives 4·5 grains (equivalent to 1·75 of pure acid). The very much larger doses of cyanide as compared with free acid are no doubt needed, because liberation of the acid is necessary when the salts are given. A heifer[7] withstood 22·5 grains pure acid in the form of potassium cyanide, but was killed by 30 grains taken by the mouth, and for the rapid destruction of dogs by intrathoracic injection on the average $1\frac{1}{5}$ grain of free acid in the form of Scheele's acid are commonly used.

As regards doses of cyanogenetic feeds, such as Java beans, assuming 20 grains as the minimum toxic dose for cattle, and an average hydrocyanic acid yield of 9 grains per pound for dangerous Java beans, it will be evident that at least 2 pounds of beans will be required. In the case of linseed an average hydrocyanic yield of 1·75 grains per pound may be expected, and thus at least 11 pounds of cake would be needed to produce poisoning. Even such

an unusually large feed would probably fail in this effect, since the gradual evolution of the acid leaves time for elimination and consequent diminution of the total effect.

Absorption and Elimination.—Hydrocyanic acid and its soluble salts are absorbed through the skin, speedily producing the general symptoms. Similarly, the vapour is rapidly absorbed through the lungs, and thus acts more quickly than by the other channels of absorption. The respiration, at first stimulated, is speedily inhibited. In the circulation the venous blood, at first redder, is eventually darker. In a test tube hydrocyanic acid forms a combination with hæmoglobin, thus preventing the absorption of oxygen. This effect appears to be absent in the living animal, and it is likely that prussic acid destroys the oxidising enzymes (oxydases) of the blood; but it must be remembered that hydrocyanic acid exercises a similar retarding effect upon the activity of such inorganic ferments as colloidal or finely divided platinum. When, for example, hydrogen peroxide is being decomposed with evolution of oxygen by means of finely divided or colloidal platinum, the addition of a cyanide greatly checks the rapidity of the change (Bredig).

In addition, experiments upon the frog, which is very resistant to ordinary asphyxiation, tend to show that the poison also exercises a paralysing effect on the central nervous system—rather more upon the medulla and lower brain than upon the cerebral cortex.

Elimination takes place through the lungs, the exhaled air having a faint almond-like odour. In the blood it is possible that ammonium formate is produced.

Symptoms.—Very large doses are exceedingly rapidly fatal, owing to the arrest of the heart in diastole. Toxic doses usually exercise a brief powerfully stimulant effect, followed by depression, paralysis, and diminution of blood tension. Given by the alimentary tract, the diluted acid causes salivation, vomition if possible, and diarrhœa with the herbivoræ. There are convulsions, spasms, vertigo, paralysis, stupor, and cessation of respiration before that of the heart-beats.

Several cases are on record relating to Java bean poisoning. Thus, McCall[3] gave Java bean meal to a *collie*, a *cow*, and a *horse*, that supplied to the horse having first been boiled for one hour. Fifteen to twenty minutes elapsed before the appearance of symptoms, which ended fatally in the case of the dog within two hours, and in the cases of the cow and the horse within four hours.

Damman and Behrens[4] describe the following symptoms: vertigo, tympany, and falling, with a fatal issue in nearly every case. Mosselmann[1] describes the effect of about one pound of the beans on four *oxen* and two *heifers*. There was a preliminary period of great excitement and salivation. After two hours the animals were swollen, with slight diarrhœa, quick pulse and respiration, muscular spasms, and in one case paralysis of the hind quarters. There was rapid recovery.

C. Aggio[2] observed the poisoning of *ewes* by cherry laurel. There was loss of appetite, vomition, inability to rise, and several deaths.

Adsetts[5] describes a similar case in the *horse*. There was an indistinct and feeble pulse, mucous membranes ingested, difficult respiration, uneasiness, prostration, coldness of the extremities, loss of appetite, constipation, diminished urination, and acute pain; protracted over three days, these eventuated in death.

It will be readily perceived from these examples that one has not to deal here with simple cyanide poisoning. It is well known that the train of symptoms and the post-mortem lesions in cases of the ingestion of a plant often suggest irritation which is by no means typical of the pure active principle. The difference between the effects of the yew plant on the one hand and the active principle taxine on the other hand form a good similar example. The difference is obviously referable to the fact that often these plants contain, in addition to a specific active poison, irritant substances; *e.g.*, cherry laurel contains an essential oil analogous to turpentine.

Post-Mortem Appearances. — In the laurel cases

inflammation, not only of the gastro-intestinal tract, but also of the heart, was observed. Such irritation is not typical of cyanide poisoning or that due to Java beans. Animals poisoned by hydrocyanic acid display congestion of the central nervous system and the lungs; fluid, black, and oily blood; the cavities of the heart contain bubbles of gas, and all parts of the corpse have a faint smell of bitter almonds.

Treatment.—The poison should be removed by emetics or the pump, and measures taken to combat prostration—*e.g.*, stimulants and warmth. Atropine has been recommended, and also the injection of sodium sulphide and sodium thiosulphate, in the hope that the comparatively harmless sulphocyanides may be formed. Ammonia and chlorine have been further recommended. According to Claude Bernard, experiments on rabbits show that under ether considerably larger doses than those ordinarily toxic may be withstood, so that if an animal were kept under anæsthesia it is possible that there might be time for the elimination of the hydrocyanic acid.

The classical antidote is freshly precipitated ferrous hydrate, which is made by mixing iron sulphate and *liquor potassæ*, and which may be given *ad lib.* It must not be made with carbonates, for ferrous carbonate does not easily form ferrocyanide, and the production of this harmless salt is the object of the treatment. But the antidote is useless when symptoms have set in, and there is rarely time to apply it in any case.

Chemical Diagnosis.—The tests for hydrocyanic acid are excessively delicate. Since hydrocyanic acid is volatile, the separation from tissues is effected by slow distillation in a current of steam of the material, acidified with tartaric acid or dilute sulphuric acid, the hydrocyanic acid being collected in the distillate.

The best test is the absolutely characteristic formation of Prussian blue by the action of precipitated ferrous hydroxide on a caustic alkali solution of the cyanide; this leads to the formation of ferrocyanide, and after acidifica-

tion the ferric salts present yield, with the ferrocyanide, Prussian blue. It has been shown[8] that the delicacy of this test reaches the $\frac{1}{3000}$ part of a grain; of equal delicacy, but not characteristic, is the production of a more or less dark yellow to red-brown coloration by warming with alkaline picrate. By the aid of these tests prussic acid may be detected post-mortem, not only in the viscera, but also in the blood and brain. It is interesting to observe that in the case of a rabbit killed forty minutes after the administration of a non-toxic dose of $\frac{1}{60}$ grain by the mouth, cyanide was present in the stomach, but could not be recognised either in the blood or brain.

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[6] Dunstan and Henry, *Journal of Board of Agriculture*, 1907-08, p. 726.
[7] Lander, *Journal of Board of Agriculture*, 1910-11, p. 904.
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CARBOLIC ACID AND ALLIED PREPARATIONS.

Sources and Preparations.—*Carbolic acid* [phenol (C_6H_5OH)] is a constituent of the tar either of coal or wood. The proportion of carbolic acid in crude coal tar is about 0·5 per cent., and it is separated in the distillation in the so-called middle oil. The three isomeric *cresols* ($C_6H_4CH_3OH$)—*ortho*, *meta*, and *para* respectively—form about 3 per cent. of the heavy or creosote oil of tar distillation. In wood tar there is also found guaiacol ($C_6H_4OCH_3OH$), the methyl ether of orthodihydroxybenzene. Of these various phenols, carbolic acid is the only one prepared

in a pure condition on anything like a large scale. The complex mixture of the various phenolic bodies is commonly known as *cresylic* or *tar acid*, and finds several technical applications. *Creosote* or *tar dips* are mixtures of tar acids with the hydrocarbons that accompany them in the tar oils with hydrocarbon oils, alkali, and resin soaps. Pyridine bases derived from the coal tar are also present, and the following is a good example of the composition of such a dip:*

	Per Cent.		Per Cent.
Water	8·20	Rosin acids ...	18·73
Soda	2·27	Phenols	14·11
Pyridine	2·25	Hydrocarbons ...	54·44

It may be pointed out at this stage that the solubility of carbolic acid in water is about 5 per cent., and that of the cresols about 2½ per cent.; but that these compounds are soluble in caustic alkalis, whilst the neutral hydrocarbons are emulsified, yielding turbid opalescent solutions with soaps.

Based upon these principles is the preparation of such disinfecting agents as creolin and lysol, which essentially are feebly alkaline mixtures, having as their active principles phenols, and capable of forming emulsions by reason of the resin or fatty soaps which they contain. In spite of frequent claims to the contrary, all phenolic preparations must be regarded as poisonous; thus with a carbolic dip the dilution for use should be such that the phenols do not exceed 0·75 per cent.

Tar oil, or *oil of pitch* distilled from wood tar, resembles creosote, but contains chiefly guaiacol and its allies.

Oil of tar with vaseline, or mineral lubricating oil, is a very common mange dressing for horses, and sometimes finely divided mercury is also added.

Crude creosote is very widely used for the impregnation of wood sleepers and fence spiles.

From all these sources, and even from tar itself, poison-

* United States Department of Agriculture, Bulletin 107, 1908.

ing may arise; and under this general heading must also be included cases of poisoning due to the contamination of water by coke oven and gasworks effluents.

As regards tar, it must be remembered that crude tar contains some 3 per cent. of cresols or tar acids, nearly all of which passes into water when the tar and water are mixed with one another. In a similar way water may take up sufficient cresols from creosoted sleepers to cause poisoning. But creosote, as it contains a larger proportion of cresols, is naturally more dangerous than crude tar. Cases of poisoning by both these means have been investigated.

Effluent waters from coke ovens and gasworks contain sulphocyanides and tar acids. An example of a coke oven effluent, analysed in 1909, showed:

Sulphocyanide ...	...	...	1·82 grains per gallon.
Cresols (tar acids)	...	...	5·81 ,, ,,

Large quantities of such a water would be required to cause death. Nevertheless, an effluent of such composition must be regarded as a dangerous contamination to drinking-water.

Wallis Hoare saw a case of a cow (poisoned by effluents from a disinfectant factory) in which the milk smelled of carbolic acid, as also did the dejecta. Recovery after purgation and olive oil took place slowly.

Toxicity.—The toxicity of phenol varies according to the channel of absorption. The lethal dose for the horse by the alimentary tract is about 1 ounce, for the dog from 1 to 2 drachms, although 15 grains is said to have caused death.

Of the cresols, the *ortho* compound is more poisonous than carbolic acid, *para*-cresol still more so, but *meta*-cresol less. All the phenols thus appear to be powerful poisons of the same order as hydrocyanic acid and arsenic. It is exceedingly difficult—in fact, impossible—to give quantitative data regarding poisoning by absorption through the skin, but in the case of creosotic sheep-dips the Departmental Committee found that a 1½ per cent. tar acid dip caused

grave symptoms, one sheep dying about three hours after dipping, whilst a ¾ per cent. dip was safe.*

Kaufmann further cites cases of death in horses by the external application over the whole body of 2 and 3 per cent., and of fowls by 5 per cent., lysol.†

Wallis Hoare communicates a case in which 2-ounce doses of lysol were prescribed for parasitic gastritis in sheep. According to the owner the treatment killed more animals than the disease!

Symptoms. — Concentrated carbolic acid precipitates albumins, with which it appears to form a loose combination, and is more penetrating to the tissues than the majority of corrosives. The concentrated acid is a violent corrosive, and its local effects may prove fatal from shock and collapse after the ingestion of large doses. In the dilute condition carbolic acid manifests after absorption marked effects on the central nervous system, illustrated by weakness, stupor, and tetanic convulsions, similar to those produced by strychnine, choreic movements, followed by paralysis of the locomotor system, and death. With very large doses the collapse may be immediate without convulsions.

In one case[2] carbolic acid had been poured upon the food of *cows*; they showed loss of appetite; the bodies distended by food; constipation; respiration slow; temperature 105° F.; weak and very quick pulse; soreness of mouths; salivation; and, in two cases, partial coma.

In another case[5] *horses* which had drunk water containing carbolic acid showed a blanching of the buccal membranes, staggering, twitching muscles, eyes staring and pupils dilated, and incipient coma.

In a third case[3] a *horse* had had by mistake 8 ounces of creolin in water, and after twelve hours showed dulness and general restlessness; quick pulse; temperature above normal; mucous membranes pale; the urine was almost black, and had a tarry odour. After three days' prostration

* Report of Committee on Sheep-Dipping, 1904.
† *Traite de Thérapeutique*, 1901, p. 133.

symptoms of acute enteritis set in, and the animal died in violent abdominal pain.

A bull *terrier*,[4] after eating a mouthful of carbolised bran, nine hours later displayed muscular twitchings, followed by paralysis of the hind quarters; abdominal distension; difficult respiration; the mucous membranes of the mouth white and hardened. The symptoms lasted about four hours, with occasional attacks of convulsions, and recovery took place within twenty-four hours. The amount of carbolic acid taken could not have exceeded 1·25 grains.

A *dog*[1] having a crushed foot was dressed with ointment and given four pills which contained creosote, after which there was vomiting and diarrhœa, the ejecta smelling of creosote; a weak and quick pulse; temperature 105° F. Under treatment with stimulants, alternating with sodium sulphate, there was gradual recovery.

An interesting case[9] refers to the poisoning of *cats* by carbolic disinfectant powder. In this case clonic and tonic spasms, dilatation of the pupils, salivation, irregular and feeble heart action, well illustrate the nervous symptoms of carbolic acid poisoning.

Hobday made a series of very careful observations on the dangers of disinfectants, and investigated creolin,[8] chinosol,[7] and izal.[6] Chinosol is a derivative of the base quinoline, but for convenience may be included here. Hobday's results confirmed the toxicity (in spite of exaggerated statements to the contrary) of these agents when improperly employed. With delicate breeds of dogs and cats Hobday advises that the total application of creolin should not exceed 10 to 15 minims. In poisoning by it he indicates subnormal temperature, paralysis of hind legs, followed by complete paralysis, prostration, and clonic spasms, well marked in limbs, jaws, and eyelids. Death from collapse follows coma.

Cats are more susceptible than dogs to chinosol, and it should not be injected in doses exceeding $\frac{1}{16}$ grain and $\frac{1}{8}$ grain per pound body weight of the cat or dog respectively.

The chief symptoms are—Sneezing and coughing and increased salivation; temperature subnormal; staggering gait, commencing with loss of motor power of hind quarters; great prostration and death from heart failure.

Izal is less dangerous than creolin, but may poison, showing similar symptoms. Applications of it to the skin ought not to exceed 10 to 15 minims per pound body weight of dogs or cats.

According to Dollar,[10] who investigated the clinical value of 'Jeyes' and creolin, these agents are safe as ordinarily used with the horse. With the dog his observations on the whole agree with Hobday's, and he advises 10 to 15 minims of creolin per pound live-weight as the upper limit for fine-skinned, delicate, and in-and-in bred dogs, such as toy varieties, although as large a proportion as 40 minims may be safe for adults and mongrels. He found that a cat of 7 pounds weight, three years old, after well rubbing with 5 drachms of 'Jeyes' in 5 per cent. solution was poisoned, and died within fourteen hours. But he forcibly argues that this preparation is no worse than any lotion or any other efficacious agent.

Post-Mortem Appearances.—Following internal administration there is observed intense irritation of the gastro-intestinal mucous membranes; that of the rumen may become detached, but this is a frequent post-mortem observation of no special significance. Frequently there is a strong smell of carbolic acid in the viscera; the pharynx and œsophagus exhibit pallor.

In creolin poisoning, one notes the smell of the agent; the heart cavities contain dark blood clots, and the small vessels, particularly of the brain, are congested.

As characteristic of chinosol are the smell and colour of the agent, and the presence of frothy saliva in the pharynx, œsophagus, and stomach.

Treatment.—Carbolic acid after absorption is eliminated in the form of sulphuric acid derivatives or sulphocarbolates in the urine. These compounds are comparatively harmless, and in using sodium sulphate as an antidote it

is intended to facilitate their formation. Sucrate of lime and the intravenous injection of ammonia are both also recommended. In the case of the cats above referred to, 20 grains of zinc sulphate speedily acted in one instance, and was followed by egg albumin in milk, and every three hours by a mixture of chlorodyne and lime water. In a second case, the emetic proving ineffectual, the animal died. With the large animals strong purgatives and whisky have proved successful when the purge acted. The horse[5] made a speedy recovery after oil of turpentine.

Chemical Diagnosis.—Carbolic acid and its allies, being volatile, may be recovered from organic matter by distillation from the acidified mass in a current of steam. They are recognised in the distillate by their odour, by the ferric chloride coloration (carbolic acid gives a violet, creosote a smoky tint), and by the formation of the sparingly soluble bromination compounds—a crystalline solid in the case of carbolic acid, and gummy in the case of the cresols.

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[8] F. Hobday, *Jl. Comp. Path.*, 1896, p. 1.
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STRYCHNINE.

Forms and Occurrence.—The alkaloid *strychnine* occurs along with *brucine* and *igasurine* in the seeds of certain species of the Loganiaceæ ; the *Strychnos Nux Vomica* in the East Indies ; the *Strychnos Ignatii* in the Philippines ; the upas-tree (or *Strychnos Tieuté*) in Java ; snake-wood tree (or *Strychnos Colubrina*) in the East Indies : and *Strychnos*

Gautheriana in Tonquin. In the *Strychnos toxifera* of Guiana strychnine is associated with brucine and curarine.

The *nux vomica* seed, incorrectly often called the 'nux vomica' or 'strychnine bean,' forms to the number of fourteen or fifteen the seeds of the fruit, resembling an orange in size, and having a harmless pulp. The seeds have an average weight of 23 grains, and are about the size of a shilling. They have a concavo-convex shape, with a well-defined umbilical centre, are covered with soft velvety hairs, and are brownish-grey in colour. The taste is acrid and intensely bitter. The beans are rarely the cause of poisoning, but it is stated that they have been known to be accidentally incorporated into oil cakes. The powdered bean forms the nux vomica whence is also derived the extract and tincture of the pharmacy.

The content of strychnine in the bean varies between 0·5 and 2 per cent. Brucine is present in larger proportion, and both it and igasurine show the same action as stimulants to the motor nerve centres, but are considerably less active. Strychnine, or nux vomica, is frequently encountered outside the pharmacy. Vermin powders very commonly consist of mixtures of strychnine with starch or flour and blue, or soot, and such powders are the commonest causes of poisoning amongst dogs, cats, and foxes. Strychnine preparations have also been used to protect stacks from vermin. The free and general sale and use of strychnine rat powders leads to so many cases of poisoning of the smaller animals that strychnine occupies the first place, numerically, among the poisons. Cases of poisoning of the large animals by strychnine are, however, rare. Since its discovery in 1818 strychnine has also acquired an evil repute as an agent of malicious poisoning in the human subject.

Absorption and Elimination.—Strychnine is absorbed, without local effect, slowly from the intact skin, giving rise to general symptoms. Absorption is rapid by way of the mucous surfaces, or on injection. From the stomach strychnine is easily absorbed at a rate depending on the

condition of the organ, the absorption being more rapid from an empty than from a full stomach. Free strychnine is but slightly soluble in water, the salts, especially those of the organic acids, such as the tartrate and acetate, being more freely dissolved. The absorption from the rumen is, as might be expected, not considerable, and no doubt the alkalinity of the contents of that organ is partly responsible for this. The precipitation of strychnine by alkalis makes it very inadvisable to prescribe with them, *e.g.*, with *liq. arsenicalis*, etc. The last dose may contain an excess of subsided strychnine, and prove poisonous. The period between dosage and the onset of symptoms is often a question of great medico-legal significance. Manifestly, it cannot be precisely answered, and it rarely happens that one has the opportunity of observing the point. It will depend on the nature of the dose — *e.g.*, as to whether given as solid, as a salt, or in solution; on the quantity administered; on the state of the digestive organs; and on the individuality and species of the subject. Doses given hypodermically may be expected to act within ten minutes, and from one-half to two hours might reasonably be stated as the interval in the case of the administration of solid strychnine in lethal doses by the mouth to the dog.

By whatever channel absorbed, strychnine is quickly transported by the blood to the central nervous system and organs.

Strychnine is not *eliminated* very rapidly. It passes into the saliva and urine, but the elimination is not complete even in three days. It thus results that the drug in therapeutic quantities exercises an apparently cumulative effect, so that the administration of the tenth dose may kill, whereas the first had scarcely any appreciable effect. For this reason—viz., that at least three days are required for elimination—it follows that the idea of an increased susceptibility to strychnine is fallacious. The effect is that of a fresh dose plus the fractional residues of previous doses.

Toxic Doses.—The toxic doses of strychnine and of powdered nux vomica are given by Kaufmann as follows:

	Strychnine.	*Nux Vomica.*
Horse	3·0 to 4·5 grains	300 to 450 grains
Ox	3·0 to 6·0 ,,	300 to 525 ,,
Pig	0·15 to 0·75 grain	60 to 90 ,,
Dog	0·075 to 0·30 ,,	7·5 to 15 ,,

The dose for a dog is thus roughly in ordinary fractions between the $\frac{1}{15}$ and $\frac{1}{3}$ of a grain.

The relative sensibility is shown by Kaufmann's figures displaying the number of milligrammes of strychnine per kilogramme of body weight:

Man	0·40 mg.	Dog	0·75 mg.
Rabbit	0·60 ,,	Fowl	2·00 mgs.
Cat	0·75 ,,		

The limits above set forth will probably with justice be regarded as low, especially in the dog, where $\frac{1}{100}$-grain doses are given with caution to toy varieties; indeed in modern practice strychnine is given only in very small and carefully regulated doses to these animals.

Hodgkins[1] records typical strychnine convulsions and death in a toy spaniel which ate an Easton syrup tabloid after a meal, the dose of strychnine being probably $\frac{1}{32}$ grain.

Similarly, strychnine spasms followed by recovery have been noticed in a fox terrier after two laxative pills for human use found to contain $\frac{1}{60}$ grain each of strychnine.[2]

According to Bock[4] the injection into the jugular vein of 10 c.c. of a glycerin solution containing 6 grains of strychnine, destroys a horse in three to four seconds without the convulsions witnessed when a water solution is employed.

The dosage for birds is very erratic. Thus, Youatt[3] records the administration of a total of 95 grains of strychnine in twelve weeks to an owl, the dose increasing from $\frac{1}{8}$ to $\frac{2}{3}$ grain, when death resulted.

Guinea-pigs and some monkeys appear to be remarkably insusceptible to strychnine when given by the mouth.

Action and Symptoms.—Strychnine acts as a powerful stimulant to the central motor cells, and thus affects chiefly the spinal cord. The reflex irritability is greatly increased, probably by reason of a reduced resistance to the passage of peripheral stimuli along the sensory nerves. When the peripheral nerve-endings are paralysed by cocaine, injection of strychnine fails to produce the tetanic convulsions, and severance of the posterior roots of the spinal nerves in frogs has been shown by Claude Bernard to inhibit the convulsions, save when the nerve ending is stimulated. In a frog in which the anterior portion only is effused with strychnine solution, stimulation of the hind extremities is followed by ordinary reflexes, but stimulation of the anterior extremities leads to general tetanic convulsion. After the intense stimulation depression and paralysis follow.

Under the influence of strychnine an exceedingly slight external stimulus, such as a current of air, induces a normal reflex, immediately followed by the characteristic general tetanic spasms, during which the back is curved (opisthotonus), respiration arrested, and the muscles are tense. Death results from asphyxiation; usually the respiration ceases after two or three spasms.

Animals tend to avoid light, and display marked hyperæsthesia. During the spasms the rigidity of the extended limbs is so great that a small animal may be lifted in a perfectly straight position by one extremity.

In *horses* symptoms set in some hours after such doses as 5 to 6 grains, and involve an accelerated pulse, laboured breathing, abdominal pain, sensitiveness to touch, and tetanic spasms. From 1 to 2 ounces of nux vomica are required to poison the horse.

Macqueen* observed strychnine symptoms in the treatment of paralysis in the horse by doses of strychnine, increasing from 1 grain to 5 grains twice daily. Twitching of the superficial muscles is a preliminary warning. Within about twenty minutes after a further dose the horse rears, falls, and makes galloping movements, so that it moves

* Private communication.

backwards on its side in a circle. The spasm is followed by a period of quiescence. If relieved by tobacco there is recovery.

Cattle withstand relatively large doses. Thus Macgillivray, quoted by Finlay Dun,* gave an old cow in all 90 grains in solution, and this induced a few spasmodic tremors, which lasted about twenty minutes. Dun (*loc. cit.*) gave a small red cow, affected with pleuro-pneumonia, in all 47 grains within two hours and a quarter. The pulse rose to 160; the symptoms were quickly induced, and included nausea, attempts to vomit, laboured breathing, and the typical tetanic rigidity.

Dogs become very uneasy, whine, are nauseated, and often vomit. This is an important point, and emphasis must be laid on the fact that vomiting does not always save the patient. The rectal temperature rises 2° to 4° F., and general tetanic spasms occur with increasing violence at intervals of one, two, or more minutes, until death, which is rapid after the first onset of symptoms.

As to differential diagnosis, it may be recalled that strychnine spasms are clonic, whereas those of tetanus are tonic. But tetanus in the dog is very rare, whilst strychnine poisoning is very common.

Post-Mortem Appearances.—These are the appearances of asphyxia, the venous blood being dark and fluid, lungs and cerebral meninges engorged. The left heart is often firmly contracted and nearly empty. Very rarely, and in protracted cases, the intestines may show a little patchy congestion. The *rigor mortis* is a post-mortem appearance often held to be very characteristic, but its absence is not to be taken as a decisively negative sign. The feet, or the claws of birds, are generally incurved, and the muscles of the jaws are rigid. In one of the best observed cases, quoted by Taylor from the observations of Caspar of Berlin, the corpse was described as like a thousand others. The duration of *rigor mortis* among dogs is certainly not invariably more prolonged in consequence of strychnine poisoning. It may pass off within twelve hours.

* 'Veterinary Medicines,' 1910, p. 506.

Treatment consists in removal of the poison, when possible by emetics,[7] $\frac{1}{10}$ grain apomorphine hypodermically having induced vomition in three minutes[3] in a dog. The patient should be kept quiet and protected from external stimuli as much as possible. McCall records[3] a successful cure of a dog by chloroform inhalation, $\frac{1}{10}$ grain apomorphine, and after vomition $\frac{1}{6}$ grain morphine repeated two or three times.

The best physiological antidote is chloral. Howe[5] gave 6 grains chloral hydrate in solution to a dog, repeated in one hour and then in three hours. There were no fits after the first dose, and next day the dog was better. As an emergency treatment an infusion of tobacco has been successfully employed (Macer[6]).

W. C. Prudames* in treating strychnine poisoning of stag hounds, found it necessary in some cases to inject $\frac{1}{2}$ grain apomorphine to secure vomiting. Followed by 20 grains chloral by the mouth this proved effective.

There is danger in giving antidotes by the mouth, for if a spasm occurs the liquid may pass into the trachea, and cause suffocation or pneumonia. It ought also to be borne in mind that after absorption emetics may do more harm than good. According to Wallis Hoare, a large dose of chlorodyne and a full dose of castor oil have proved successful, probably by both hastening expulsion and retarding absorption.

Chemical Diagnosis.—Strychnine is obtained as a residue from organic solvents in the course of the systematic separation of vegetable principles, according to the scheme outlined later. When a large dose has been given, there may be obtained a crystalline deposit weighing several grains, but very often only a gummy smear rendered impure by the organic matter, always separated during the extraction, may result. The recognition of strychnine depends on three tests: (1) The substance must have an intensely bitter taste, a solution of 1 grain of strychnine in a gallon still exhibits distinct bitterness. (2) Strychnine dissolves

* Private communication.

without colour in concentrated sulphuric acid, and the introduction into the solution of a fragment of potassium bichromate yields a characteristic violet colour, passing quickly into rose pink, which is persistent for some time. The colour is produced in streaks on moving the bichromate crystal through the liquid. (3) When bichromate solution is added to a solution of strychnine in dilute acetic acid, the sparingly soluble strychnine chromate separates in crystals. The precipitate gives with concentrated sulphuric acid the same colour reaction as described under (2).

Although strychnine is amongst the most stable and easily recognised alkaloids, error is possible. When organic impurities are considerable, reduction of chromate and sulphuric acid may occur with simulation of the strychnine colours, and, moreover, those colours proper to strychnine may be effectually marked. Further, many residues, especially those derived from herbivorous stomach contents, give a precipitate with chromate in dilute acetic acid, which is, however, amorphous, although with concentrated sulphuric acid it gives a red colour. In such doubtful cases the physiological action of the extract ought to be tested on a mouse, for which $\frac{1}{1200}$ grain of strychnine is lethal within ten minutes.

REFERENCES TO STRYCHNINE.

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[2] Anon, *Vet. Record*, 1906, p. 356.
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[4] Bock, *Vet. Jl.*, 1907, p. 447.
[5] N. Howe, *Vet. Record*, 1898, p. 325.
[6] J. Macer, *Veterinarian*, 1870, p. 209.
[7] Gowring, *Veterinarian*, 1870, p. 209.
[8] Youatt, *Veterinarian*, 1840, p. 28.

MORPHINE AND OPIUM.

Forms and Occurrence.—Opium is the inspissated juice of the opium poppy, *Papaver somniferum*, which is native to Southern Europe and the Levant, though cultivated and

occasionally found wild in England. Poisoning of animals by eating the opium poppy is, however, unknown.

The juice contains some fifteen alkaloids most of which are present in very small proportions. The table displays the chief of these with the average percentages in which they are found in opium.

Hypnotic.	Per Cent.	*Convulsant.*	Per Cent.
Morphine	10·0	Thebaine	0·15
Codeine	0·3	Papaverine	1·0
Narceine	0·02	Narcotine	6·0

The other alkaloids are not important.

Poisoning by morphine is rare amongst animals, this alkaloid affording a good illustration of the variation in the action of a cerebral poison according to the degree of nervous development. Doses ordinarily given to dogs for anæsthetic purposes would prove fatal to a man. Horses and ruminants are less susceptible, and since in the horse the brain is relatively less highly developed than the cord, the spinal are more marked than the cerebral effects.

Toxic Doses.—Kaufmann gives the toxic doses on subcutaneous injection as:

Horse	45 to 75 grains
Ox	75 to 120 „
Small dog	1·5 „
Large dog	15 „

These doses are probably too low; for the horse 75 to 100 grains and for the dog about 4 grains per pound body weight are, however, fatal. Wooldridge uses $\frac{1}{4}$ to $\frac{1}{3}$ grain for anæsthesia in small, and 2 grains in large, dogs. It is very doubtful if poisoning by the mouth ever occurs, and it has been proved to be impossible to kill a pigeon in this way. Doses have been given experimentally to the pony reaching 60 grains by the mouth, without evoking marked symptoms, and after slaughter there was no sign of action beyond impaction of the stomach contents due to gastric paralysis.

Absorption and Elimination.—Morphine is somewhat slowly absorbed from the alimentary mucosa. When given hypodermically, it is excreted into the stomach. In a recent experimental case the writer found morphine by means of Pellagri's test in the urine, and in the abomasum of a calf which had received 30 grains of morphine under the skin thirty hours before death. In the system morphine is rapidly eliminated, being oxidised in the blood stream to oxydimorphine, or at any rate modified chemically to a substance closely allied to morphine.

Symptoms.—In the *horse* and *ox* toxic doses of morphine give rise to a period of excitement marked by restlessness, bellowing, laboured breathing, full pulse, sweating and dilatation of the pupil. Later, coma sets in with general loss of sensibility, slow pulsation and breathing, and a subnormal temperature. There is arrest of the digestive functions leading to nausea, indigestion, and tympany.

The excitant effects of morphine on the *horse* are sometimes seen when it or opium is given in colic. The animal moves in a circular direction ('circus mode of progression'), an effect which used to be ascribed to the disease, but is in reality due to the drug (F. Smith). The action on the *ox* is similar, and the excitement often gives the impression of madness or marked delirium.

When poppy-heads are eaten the gastric symptoms become more pronounced.

Sheep display similar symptoms to cattle.

In the *cat* an hypnotic effect is rarely seen, the action being that of motor excitement.

In *dogs*, as in man, a toxic dose induces excitement and reflex irritability, vomiting, contracted pupil, sometimes convulsions. Thereafter follow coma, respiratory failure, and death.

Post-Mortem Appearances are not characteristic, being those of asphyxia.

Treatment consists in removal of the cause by emesis, the pump, and purgation. The depressant effects are combated by caffein hypodermically, or in cerebral

excitement cold applications to the head. The stimulant action of small doses of atropine seems useful. Potassium permanganate, slightly acidified, is valuable to destroy unabsorbed morphine.

Chemical Diagnosis.—Morphine is separated in the course of the alkaloid research by means of amyl alcohol extraction from an ammoniacal solution. Morphine possesses many reactions, few of which have any value in practical toxicology. Ferric chloride gives a purple colour with solutions of morphine, but the test is not delicate, a fair amount of morphine being needed. Many of the reactions are reduction processes, and therefore most unreliable, if not absolutely misleading. A good test is that of Pellagri, which depends on the formation of apomorphine, and is thoroughly diagnostic. The suspected residue is warmed with concentrated hydrochloric acid and a few drops of sulphuric acid until heavy fumes begin to be evolved, cooled, diluted and neutralised with sodium bicarbonate. The solution is usually pale pink at this stage. Solution of iodine is added very slowly in small quantities, when an emerald green colour develops. On shaking with ether, two layers form, the lower aqueous layer is green, and the upper ethereal layer red.

Several experiments have been made at the Royal Veterinary College to test the reliability of the methods of extraction and recognition of morphine. A pony had 15 grains morphine in ball, and was killed after four and a half hours. The urine was tested and gave a good reaction for morphine by Pellagri's test. Another animal was killed three days after receiving 60 grains, and on analysis gave no reaction from ingesta, and doubtful tests from fæces and urine. No Pellagri test was got in extracts from the lungs, liver, and stomach of a dog which had had 15 grains of morphine acetate subcutaneously.

In opium the alkaloids are combined with meconic acid, which is separated in the routine process, and gives a dark red colour with ferric chloride, stable to acids. The

recognition of meconic acid is usually taken as satisfactory evidence of the presence of opium.

COCAINE.

Occurrence.—Cocaine is the active alkaloid of the coca plant, *Erythroxylon coca* indigenous to South America. In small doses it is a powerful stimulant, causing exhilaration and enhanced muscular activity, for which reasons it is used by the natives when performing long journeys without food. On the same grounds horses are sometimes 'doped' with cocaine, or cocaine preparations, such as powders containing 90 per cent. cocaine and 10 per cent. strychnine. Cocaine is valuable as a powerful local anæsthetic, especially in eye diseases. For local anæsthesia in dogs Wooldridge uses $\frac{1}{10}$ grain per pound body weight, with a maximum dose of 2 grains.

Poisoning may result from accident or from excessive 'doping' and depends on the general action of cocaine as a convulsant, producing spasms, which may be confounded with those due to strychnine.

In the *horse* toxic doses (60 to 100 grains) produce restlessness and excitement, salivation, dilatation of the pupil, acute mania, and intense excitement. In the *dog* there is first observed anxiety and fear, then exhilaration, followed by weakness, muscular twitching, rhythmical movements, convulsions and stupor, dyspnœa, feeble pulse, and weakened respiration. The respirations diminish in amplitude but increase in frequency, and cease from 20 to 25 seconds before the heart.

Examples of cocaine poisoning are rare, not at all likely to occur with the horse or ox, and not often with the dog.

At the present time cocaine is often replaced by the substitutes such as *novocaine* and *eucaine*, or conjoined with *adrenaline*. This acts by causing anæmia of the part injected, and thus preventing the carriage of the cocaine to the vital organs (Wallis Hoare).

The **toxic doses** in grains per pound body weight are approximately—

Horse	$\frac{1}{24}$ grain
Ox	$\frac{1}{30}$,,
Dog	$\frac{1}{12}$,,

but these are probably too low.

In the system cocaine is largely oxidised, and only in part excreted by the kidneys, which renders its detection as a dope a matter of some difficulty.

Treatment of cocaine poisoning requires rapidly acting stimulants, such as nitroglycerin, strychnine or atropine, together with the usual steps towards securing elimination.

Chemical Diagnosis.—Cocaine is separated in the general alkaloid scheme, and may be recognised by producing local anæsthesia on the tongue, and by the characteristic formation of a purple crystalline precipitate with potassium permanganate, a test which fails, however, unless the alkaloid extract is fairly pure.

ESERINE OR PHYSOSTIGMINE.

Occurrence.—The alkaloid eserine occurs in the seed of *Physostigma venenosum*, Calabar bean, chop nut, or ordeal bean, native to West Africa, and used by the natives as a primitive method of trial by ordeal. Eserine is not likely to give rise to poisoning among animals, but is used somewhat extensively as a powerful defæcant in obstruction of the bowels. In this capacity caution is needed, as some subjects are very sensitive to the toxic effects. Crude eserine is further liable to be contaminated with calabarine, an alkaloid which causes severe muscular tremors. The dose of eserine as sulphate or salicylate is from 1 to $1\frac{1}{2}$ grains for the horse hypodermically.

The **toxic doses** by subcutaneous injection are, according to Kaufmann:

Dog	$\frac{1}{10}$ to $\frac{1}{15}$ grain
Horse	$2\frac{1}{2}$ grains
Ox	5 ,,

The doses are probably underestimated, for Winslow ('Veterinary Materia Medica') gave a horse two doses of 3 grains each within twenty-five minutes, and evoked symptoms with recovery in two hours.

The same author quotes a case of an aged horse which had suffered for a week from impaction of the colon. He was given 12 grains of a commercial extract of Calabar bean, fell almost immediately, perspired, exhibited muscular tremors, and died within a few minutes.

Symptoms.—After a toxic dose of eserine there are powerful muscular tremors resembling convulsions of central origin, eserine acting like strychnine in augmenting the reflex activity of the cord. The excitement is followed by paralysis, eventually affecting the respiratory muscles, and death results from asphyxia. The animal falls and the breathing is rapid, laboured, and stertorous, and in the later stages feeble and irregular. There is increasing salivation, sweating, vomition when possible, and increased peristalsis with expulsion of dung and gas. The myotic action of small doses is often replaced by mydriasis after large toxic administrations.

Post-Mortem Appearances.—The large intestine is empty, anæmic, wrinkled, and hard. Bladder empty and contracted, as also is the uterus. The muscles and motor nerves retain sensibility for some time after death (Kaufmann).

Treatment consists in emetics or the pump. After respiratory failure life may be prolonged, or even saved, by artificial respiration (Kaufmann). Atropine is antagonistic to eserine, and should be given subcutaneously. In distinction to eserine, atropine inhibits secretion, diminishes reflex excitability, paralyses the alimentary organs and bladder, accelerates the heart by paralysis of intracardiac vagi, and is mydriatic (Kaufmann), but large doses of atropine are to be avoided, as aggravating the eserine effects.

Alcohol, digitalis, and ammonia may be given by the mouth, whilst strychnine is stimulant to the respiratory centres.

Chemical Diagnosis.—Eserine is separated in the ordinary procedure for alkaloids. Its solutions on keeping assume a reddish colour, without loss of activity. The myotic effect may be observed, but other myotics such as pilocarpine and muscarine must be excluded.

Eserine gives the following colour tests: Sulphuric acid, yellow; bleaching powder solution, red; bromine, red; but none of these is characteristic.

PILOCARPINE.

Occurrence.—The leaves of *Pilocarpus jaborandi*, indigenous to Brazil, contain the alkaloid *pilocarpine*, which is usually prescribed in the form of the nitrate.

Pilocarpine is allied in its physiological actions to eserine, and more closely to *muscarine*. Muscarine is the active alkaloid contained in the fungus *Agaricus muscarius*, or the fly-blown agaric. This plant is used by the Siberian natives to produce a kind of intoxication, and by long use they appear to gain a degree of tolerance to its toxic effects.

Effects and Symptoms.—Pilocarpine stimulates glandular excretion and involuntary muscle. Horses salivate copiously; after 3 grains subcutaneously the horse champs its jaws and salivates freely, but does not sweat; the bowels move freely by stimulation of the involuntary intestinal muscles (F. Smith). With large doses the mucous secretion of the bronchi is so great that taken along with the contraction of the tubes, great dyspnœa, which may be fatal, is produced. According to Kaufmann horses are poisoned by 5 grains subcutaneously, but cattle are less sensitive. The dog and cat are more sensitive, Fröhner stating that ¾ grain killed a dog weighing 132 pounds by pulmonary œdema (Finlay Dun).

Poisoning by pilocarpine could scarcely occur as the result of accident or malice. It is only likely to happen under treatment, and it is not therefore necessary for

present purposes to do more than signalise its possibility and main features. Atropine, which stops secretion and paralyses involuntary muscle, is clearly the proper physiological antagonist, and is the remedy to be used in the contingency of an overdose of pilocarpine or eserine.

Chemical Diagnosis.—An alkaloid suspected to be pilocarpine may be tested for as follows: A small quantity of the chloride is shaken in a test-tube with a crystal of potassium bichromate, 1 to 2 c.c. of chloroform, and 1 c.c. of 3 per cent. hydrogen peroxide. After a few minutes the chloroform layer becomes blue-violet to indigo, according to the quantity of pilocarpine (Helch).

IPECACUANHA AND EMETINE.

Occurrence.—Ipecacuanha is the dried root of the *Cephaëlis ipecacuanha*, indigenous to South America, and owing its activity to the alkaloids *emetine*, which is present in the dry root in the proportion of 1 to 2 per cent., and in smaller proportions *cephaeline* and *psychotrine*. In pharmacy emetine is rarely used, the extract, syrup, and wine of ipecacuanha, being the usual forms.

Effects.—There is very little chance of poisoning being caused in the ordinary applications of this drug, large doses of which are needed to give toxic symptoms. Thus, 3 ounces are quoted by Winslow as having killed a horse, whilst Finlay Dun gives 3½ ounces. As regards pure emetine Winslow gives 2 grains as fatal to a dog, and Finlay Dun gives for the dog ½ to 8 grains, and for the cat ⅓ grain. The latter authority describes 2 grains swallowed by a dog as having caused violent vomiting, increased mucous secretion from the respiratory and alimentary membranes, inflammation of the stomach and intestines, stupor and death in twenty-four hours (Magendie). The emetic effects of these drugs are probably due to local gastric action, for when given under the skin, emetine is excreted into the stomach, and may be found in the first vomits (Winslow).

GELSEMIUM.

Occurrence.—Gelsemium is the root of *Gelsemium sempervirens*, or yellow jessamine, native to the Southern United States. The drug contains two alkaloids, *gelsemine* and *gelseminine*. According to Cushny* gelsemine exercises an effect on frogs like strychnine, but not on mammals, and gelseminine has an action on mammals almost exactly like that of conine. According to the same authority confusion of the two names occurs. Gelsemium is, however, very little used in practice, and cases of poisoning thereby are exceedingly unlikely to be encountered.

Effects.—The general effect is of paralysis of spinal and not cerebral origin. Probably the convulsant action generally observed is due to the gelsemine, the main symptoms being due to the paralysant action of gelseminine. In poisoning one observes muscular weakness, staggering and falling, with convulsive movements of the head, fore, and sometimes hind legs. Respiration is slow, pulse feeble, and temperature reduced. Consciousness is preserved, and death occurs from asphyxia with almost simultaneous arrest of the heart.

The *lesions* are those of asphyxia, as with conine. Stimulants such as strychnine, atropine, digitalis, and alcohol, along with general measures of elimination are indicated as remedies.

Chemical Diagnosis.—Preparations of gelsemium root are identified by testing for gelseminic acid (β-methyl-æsculetine) a substance present in many of the *Solanaceæ*. It is left as a residue after evaporation of a chloroform extract from acid solution, and is characterised by giving a beautiful blue fluorescence in water or watery alcoholic solution.

VERATRINE.

Occurrence.—Veratrine is contained in the *Veratrum album*, a species of the *Colchicaceæ* found in Alpine districts,

* *Pharmacology*, 1905, p. 265.

but not in Great Britain, but the chief source of veratrine is the Mexican *Sabadilla officinalis*. With it are associated the alkaloids veratroidine and jervine.

Toxic Doses.—Veratrine is an exceedingly poisonous alkaloid, the toxic doses given by Kaufmann being:

By the Mouth.

Horse	...	...	...	...	15 to 45 grains
Ox	...	...	...	...	15 to 45 ,,
Dog	...	...	...	...	1 to 5 ,,

According to Cornevin, 1 gramme per kilogramme body weight of the fresh root of *Veratrum album* kills the horse, and 2 grammes per kilogramme the cow.

Symptoms.—*Dogs* to which veratrine is given hypodermically salivate profusely, perform movements of mastication and deglutition, and vomit profusely (eventually mucus). Similar symptoms affect *horses* and *cattle*, and actual vomition occurs with the latter. In all cases there is profuse purgation, and frequently excessive urination. After a period of excitability there is calmness, prostration, inability to rise, and inco-ordinated movements of the members. There is an increase in muscular contractibility accompanied by a marked prolongation of the period of relaxation.

Poisonous doses lead to a weak, irregular pulse, owing to the effects on the heart muscle, inhibitory apparatus, and vasomotor centres. The respiration is deep and slow, and death occurs in convulsions or paralysis.

J. B. Cresswell* saw a horse seriously poisoned three hours after having been given a ball containing *Veratrum album* as a remedy for grease. There was continual retching, but no actual vomiting; pulse 86, irregular and feeble; respiration 68.

Post-Mortem Appearances.—These are more or less extensive inflammation and hæmorrhagic patches in the pyloric end of the stomach and in the intestines. The bladder is empty, kidneys inflamed, and liver often but not always congested. The blood is black and fluid.

* *Veterinarian*, 1886, p. 227.

Treatment consists in elimination of the cause, respiratory stimulants, and warmth. Artificial respiration, if possible, is effective. Carbonates and demulcents are indicated, tannin as an alkaloid precipitant, and morphine against the nausea and gastric irritation.

Cresswell (*loc. cit.*) gave 3 ounces of whisky and 3 ounces of ammonia carbonate hourly for six doses, and then two-hourly. In twelve hours there was improvement, and, under tonics and stimulants, recovery.

Chemical Diagnosis.—Veratrine is remarkable in causing violent sneezing when a little of the powder is sniffed. The quantities isolated in a toxicological research are rarely sufficient for this excellent test.

A characteristic reaction is that given by warming with hydrochloric acid, which yields a green passing to red colour. The red coloration with sulphuric acid is also given by the constituents of hellebore. Sulphuric acid and sugar yield with veratrine successively a yellow, green, and violet colour.

The general effects of veratrine ought to be observed by injection in a mouse.

CURARINE.

Occurrence.—The alkaloid *curarine* is contained in the curara, wourara, wourali, or arrow poison, *Strychnos toxifera* of Guiana, and is associated therein with strychnine and brucine.

Effects.—Curarine paralyses the peripheral endings of motor nerves. The first parts to be affected are the limbs, then the trunk and head, and finally the respiration, which, with poisonous doses, is gradually enfeebled, and ultimately ceases. Consciousness and intelligence are unimpaired. For the horse 32 to 48 grains subcutaneously are fatal, and for the dog $\frac{5}{6}$ to $3\frac{1}{3}$ grains.*

When taken by the mouth curarine is absorbed slowly.

* Finlay Dun, 'Veterinary Medicines,' 1910, p. 533.

It is eliminated rapidly, and unaltered in the urine, by whatever channel it is given.

Artificial respiration is successful in combating curare poisoning mainly by reason of the rapid elimination of the alkaloid.

As with the other medicinal alkaloids, there is little likelihood of curare poisoning occurring outside of possible overdosage.

YOHIMBINE.

Occurrence.—Yohimbine is an alkaloid derived from the bark of the *Coryanthe yohimbi* (Schumann), found in the Cameroons.

It is given in the form of the chloride in doses of grains $\frac{5}{6}$ for the stallion, $1\frac{1}{4}$ for the bull, $1\frac{1}{2}$ for the cow, $\frac{1}{6}$ for the sheep, $\frac{1}{400}$ to $\frac{1}{250}$ for small dogs, $\frac{1}{60}$ for dogs from 20 to 50 pounds, and $\frac{1}{2}$ for dogs over 50 pounds. These doses may be repeated three times a day (Finlay Dun).

Effects.—Yohimbine has recently assumed importance as a powerful non-irritant aphrodisiac drug, which excites the spinal erection centre and congests the genital organs.

Poisoning has not often been observed, but Finlay Dun* states that dogs have been killed by $\frac{1}{2}$ grain, displaying dyspnœa, depression of the heart, salivation, diarrhœa, a low temperature, partial paralysis, and convulsions.

Although poisoning could not occur save as the result of careless dosage, it is well to point out its possibility, especially in view of the rather wide use of the drug, and the likelihood of inexpert employment.

Chemical Diagnosis.—Yohimbine gives with strong sulphuric acid and potassium bichromate a dirty greenish-blue colour, rapidly passing to dirty green (not characteristic). When yohimbine is mixed with a drop of a solution of benzaldehyde in alcohol (1 to 4), and a drop or two of sulphuric acid is added, the mixture is at first dark brown, then gradually (first at the edges) becomes cherry-red, and finally violet.

* 'Veterinary Medicines,' 1910, p. 618.

COCCULUS INDICUS.

Occurrence.—The seed kernels of *Anamirta paniculata*, or Levant nut, well known as *Cocculus indicus*, contain a chemically neutral active principle, *picrotoxin*, which is probably a glucoside. The Levant nut has an interesting toxicological history, but cases of poisoning by it of the large animals are not described, although it is stated that a small percentage of malicious poisonings of cattle in India are due to it. Taylor* relates cases of picrotoxin poisoning which will show the possible vehicles. The principle is very bitter and intoxicating, though in large doses it causes intense pain and frequent vomiting. It used to be employed as an adulterant to beer, and Taylor cites cases of poisoning by this means. It was also used as a fish poison, to poison wheat for the destruction of birds, and by robbers to render their victims powerless or "hocussed" (Taylor).

Cushny classes picrotoxin, as regards its effects, along with cicutoxin and œnanthotoxin, the active principles of *Cicuta virosa* and *Œnanthe crocata* respectively (see these).

Chemical Diagnosis.—Picrotoxin is yielded to organic solvents from the acid liquid in the systematic search for poisons. Sulphuric acid and ammonium molybdate (Fröhde's reagent) gives a gold to saffron-yellow colour. If picrotoxin is evaporated to dryness with a little strong nitric acid, the residue just moistened with concentrated sulphuric acid, and caustic soda added, a red colour is produced.

CANNABIS INDICA, OR INDIAN HEMP.

Cannabis indica is the dried flowering or fruiting tops of the female plant of *Cannabis sativa*, native to India, and is officinal in the British Pharmacopœia. In India the drug is rarely used for homicidal purposes, but is sometimes employed to produce narcosis and facilitate the commission

* 'Poisons,' 1875, p. 678.

of crime. In that country three forms are used to secure pleasant dreams—viz., bhang, the powdered ears and stalks; ganja, the dried flowering tops; and charas, the resin extracted from the green plant. The first two are used to prepare drinks and sweetmeats, and the third (charas), or a similar preparation, sometimes containing datura and called 'majun,' is used for smoking.

Ganja is used in India as an anodyne—*e.g.*, in the shoeing of, or surgical operation on, the horse, and is nearly, if not equally, as good as morphine. Rutherford, who used the drug in India against equine colic, states that it is as rapid as opium, and has the advantage of not arresting the action of the bowels or causing delirium. It is also stated that the duration of the narcosis is longer.

The toxic doses for animals must be large. In the literature there are few, if any, references to poisoning of animals either by the plant or extract. Hobday, quoted by Finlay Dun, states that doses of 10 grains to 2 drachms of extract given to *dogs* cause stupor, with paralysis of the hind quarters, which might last two days, but are not fatal.

F. Smith and Rutherford both used Indian extract in veterinary work, and the latter (quoted by Finlay Dun) observed the effects of doses of 1 to 8 drachms in bolus to horses. Only the 1-drachm doses caused preliminary excitement. The general symptoms were dulness and sleepiness, pulse and respiration slowed, food neglected. When trotted, the animals moved as if drunken, sideways and unsteadily. Defæcation was suppressed, and the dulness lasted up to thirty-five hours, after which recovery ensued.

From these valuable observations it is clear that fatal poisoning by Indian hemp need not be seriously apprehended in its applications to animals.

SANTONIN AND WORMWOOD.

Occurrence.—The shrubs santonica (*Artemisia maritima*) and wormwood (*Artemisia absinthium*) of the order Compo-

sitæ, both grow in Great Britain, but are not likely to be eaten by small animals. The effects of the active principles on larger animals are not serious. Wormwood yields *oil of absinthe*, a narcotic poison causing trembling, stupor, and convulsions in dogs. It is used as a remedy for worms. Its consumption in the form of absinthe gives rise to the very grave chronic absinthism in man.

Santonin, derived from the flower heads of *A. maritima*, is very commonly used as a vermicide for round and thread worms, the doses for dogs being from $\frac{5}{6}$ to 3 grains, and for cats and small dogs from $\frac{1}{3}$ to $\frac{5}{6}$ grains.

Symptoms and Treatment.—Overdoses are dangerous, and sometimes fatal. They cause in *dogs* twitching of the head muscles, rolling of the eyes, grinding of the teeth, rotation of the head, and epileptiform convulsions, followed by clonic spasms. During the spasms the respiration is disturbed, and asphyxia may occur (Cushny). Santonin imparts a blood-red colour to the urine. It has been known to cause temporary blindness in the dog.

Poisoning is to be treated by emetics and purgatives. The convulsions are prevented by chloroform, chloral, or bromides, and artificial respiration is used if necessary.

Chemical Diagnosis.—(1) Santonin in solution in a little alcohol is heated in a dish at 100° C. with 1 or 2 drops of 2 per cent. alcoholic furfural solution, and 2 to 3 c.c. of strong sulphuric acid. The liquid becomes purple-red, blue-violet, dark blue, and after several hours deposits a black precipitate (delicacy $\frac{1}{8000}$ grain, Thæter). (2) Santonin is heated with a mixture of 2 of strong sulphuric acid to 1 of water until the liquid is yellow, and after cooling a trace of ferric chloride is added. This gives, as a rule, a turbidity, whilst on again heating there is a violet coloration (delicacy $\frac{1}{8000}$, Lindo).

When a suspicion arises that harm has been caused by overdosage of santonin tablets, the recognition, and, if possible, weighing of santonin, removed from organs, becomes a very important matter from the medico-legal point of view.

TURPENTINE, CAMPHOR, AND ESSENTIAL OILS.

Occurrence.—Under this heading may be conveniently collected those cases of poisoning arising either from oils, essences, or the plants which contain them. The general effect of this class is irritant, and after absorption narcotic or paralysant.

Turpentine is the hydrocarbon, or mixture of hydrocarbons, distilled from the oleoresin of the pine, *Pinus sylvestris*, etc., of the order *Coniferæ*. Turpentines are optically active, rotating the plane of polarised light, and this affords an excellent qualitative guide. Thus, in examining a tar-oil preparation the obtaining of an active distillate indicates turpentine or wood tar, as distinguished from coal tar. *Oil of savin* is chemically a turpentine, and is contained to the extent of about 3 per cent. in the tops of the common savin (*Juniperus sabina—Coniferæ*), a cultivated evergreen shrub, commonly credited with abortive properties. Savin possesses a very characteristic acrid taste and smell, and probably contains other principles than turpentine, for its activity is certainly greater. Savin, American red, or pencil, cedar (*Juniperus virginiana*), and *Wellingtonia sequoia*, are conifers not indigenous to Britain, but cultivated in our gardens, and must be held liable to be possible causes of poisoning. Like savin, the two last-named species contain essential turpentine oils.

The *rue* (*Ruta graveolens*) is an exotic member of the *Geraniaceæ*, cultivated in this country, and which contains, according to Cornevin, an essential oil, and an acid *rutinic acid*.

Camphor is a neutral, crystalline, volatile solid of the class of ketones allied to turpentine, and is obtained from the evergreen camphor laurel, native to East Asia. Artificial camphor is prepared by the action of dry hydrochloric acid gas on turpentine.

The common *tansy* (*Tanacetum vulgare* of the *Compositæ*), allied to the *Artemisia*, is a herb often used in this country

to make tansy tea. It contains a volatile ketone tanacetone, and has been credited with causing poisoning.

In detailing the nature of the poisoning caused by the above-named active principles, or the corresponding plants, it may be again remarked that they present the same general features of local irritant and remote paralysant action. And, further, that the ill-judged exhibition by inexpert persons of all varieties of turpentine, such as eucalyptus, is liable to cause serious results.

Poisonous Effects.—The symptoms, lesions, treatment, and chemistry of these agents are here shortly summarised.

Turpentine.—Gamgee (1868) grouped turpentine with oil of tar and naphtha as an active irritant, and pointed out that as an antispasmodic it is a dangerous drug, often aggravating the disease it is intended to cure. Large doses cause irritation and sometimes ulceration of the bowels. Turpentine is quickly absorbed, and exercises paralysant effects in the same order as those of alcohol.

Elimination occurs by the lungs and kidneys, and the urine acquires the characteristic odour. Repeated small doses are more likely to cause renal inflammation than one large dose, which mostly passes off in the fæces.

On examining the alimentary organs after overdosage of turpentine, one observes marked congestion and fluidity of the contents. In the horse the stomach is devoid of solid, and contains brownish-yellow liquid, on which turpentine may be floating in large quantities. Such a result leaves little room for doubt that poisoning has occurred.

Turpentine is easily recovered from organic matter by distillation in a current of steam, and its identification presents little difficulty.

Savin.—Gamgee * records a case observed in 1855 in which the abortion of foals had been secured by repeated dosage of savin. The foals were dropped dead, and from the state of the membranes it was thought that they had died some ten to twelve days previously to abortion. The condition of the mares was poor, and there was a mucous

* 'Veterinarian's Vade-Mecum,' 1868, p. 200.

discharge of an irritant nature from the anus. The urine and fæces smelt strongly of savin. The mares recovered under treatment. The poisonous dose is uncertain. Hertwig gave half a pound daily for six or eight days to horses, without effect. With large doses there is in general—diarrhœa, thirst, accelerated pulse and respiration, and great prostration.

A. Fuller* observed in *horses* poisoned by savin—heavy appearance, tucked-up flank, difficulty in swallowing, salivation, and thirst; respiration quick and laboured, pulse quick and weak; fæces hard and covered with mucus, urine dark and scanty; temperature variable, and patches of cold perspiration. The symptoms lasted four to five days, when there was great prostration and death.

C. Moir† recorded savin poisoning of an eight or nine year old bay horse. He observed—staring coat, sunken eyes, mouth clammy, and viscid saliva; fæces slimy, great urination, corded pulse, watery discharge from eyes and nostrils. The respiration was not much increased.

On post-mortem both these observers found inflammation and mucous discharge of the mouth, gullet, stomach, and intestines. In Fuller's cases the cæcum was full of yellow liquid, but in some instances empty and contracted to about one-fourth. The colon was full of undigested food, rectum thickened and inflamed, and bladder full of offensive urine. In Moir's case, he found the stomach full of oily liquid, in which savin was detected.

The treatment of savin poisoning is by means of opium to allay pain, followed by mild aperients, demulcents, and stimulants.

Rue.—Cornevin states that the leaves of rue are sometimes used, occasionally with fatal results, to procure abortion. He characterises its effects as those of a gastro-irritant causing a period of excitement, followed by depression, weakened heart action, lowering of surface temperature, abundant salivation, and swelling of the tongue.

The lesions are those of gastro-enteritis, the posterior

* *Veterinarian*, 1860, p. 135. † *Ibid.*, 1862, p. 643.

parts of the alimentary canal being often normal. In females there is congestion of the uterus, with a violet colour if abortion has occurred.

Gamgee similarly remarks that in large doses the plant acts as a narcoto-irritant. Well-authenticated cases of poisoning by this plant are hard to find, and not very likely to occur in this country.

Tansy.—Poisoning by tansy has been placed on record recently in the German literature.* The subjects were *cattle*, and the symptoms included—refusal of food, rumination slow, dung hard, dark, dry, and covered with slime; shaking movements of head and neck, pulse strong—64, temperature 38·6° C.; eyelids half-closed, pupils contracted, and globe of eye flickering; dulness, staggering gait, and weakness.

Kobert of Rostock gave it as his opinion that death had resulted from tansy.

Camphor.—Hertwig states that from ½ to ½ an ounce of camphor proves fatal to the dog, whilst 2 to 4 ounce doses to horses and cattle, and 2 to 4 drachms to sheep accelerate respiration, heighten sensibility, and occasionally cause convulsions.

The general effects of camphor recall those of turpentine, causing preliminary stimulation, with subsequent paralysis of the central nervous system.

Camphor poisoning is rare, and not very likely to be encountered. Diagnosis is made easy by the elimination of the poison in the exhaled air, to which the familiar odour of the substance is imparted.

OXALIC ACID.

Occurrence.—Salts of oxalic acid are found in many plants, notably in rhubarb and sorrel, which contain the *acid potassium oxalate*. Many plants also contain *calcium oxalate* deposited in the microscopic quadratic, or envelope-

* See abstract, *Vet. Jl.*, 1908, p. 375.

shaped crystals, in which the same salt is so generally observed in urinary deposits of the herbivorous animals.

Oxalic acid is used on a fairly large scale commercially as a straw-cleaning agent, and under the name of salts of sorrel or salts of lemon, oxalic acid is used for domestic purposes, such as the cleaning of straw hats and brass-work, and the removal of ink stains. It resembles Epsom salt, and confusion with it has caused accidents. Oxalic acid is a common poison in the human subject, but cases of the poisoning of large animals by it are very unusual. Dogs may, however, be poisoned accidentally by this agent in doses of about 15 grains, and cats by about 3 grains.

Symptoms.—Concentrated oxalic acid causes, in *dogs*, nausea and vomiting of black or brown acid material. Difficulty in swallowing, thirst, diarrhœa, and colic are alimentary symptoms common to irritant poisoning. Gamgee * indicates labouring and spasmodic respiration, injection of conjunctivæ, and dilatation of pupil; small and irregular pulse; and with advancing stupor and prostration tetanic twitchings of the muscles.

Oxalic acid and oxalates are absorbed slowly and excreted by the kidneys as calcium oxalate, which, being insoluble, may cause calculi.

Post-Mortem Appearances.—These are—a blanched appearance of the membranes of the mouth, fauces and gullet. The stomach contains much gelatinous mucus, and is rarely perforated. More or less intestinal inflammation is observed, and the blood is dark and fluid. It will be remembered that oxalates prevent the coagulation of blood by removing the soluble lime in the form of calcium oxalate.

Treatment.—Burnt magnesia or chalk are better than carbonate of potash or soda, since the former render oxalic acid insoluble. Lime water and oil and demulcents are valuable, with stimulants as indicated.

Chemical Diagnosis.—Oxalic acid may be extracted from organic matters by feebly acidified water, but the best

* 'Veterinarian's Vade-Mecum,' 1868, p. 137.

method is that of dialysis through parchment as practised for salt, nitre, and mineral acids. The diffusate may be purified by neutralising and adding lead acetate, which precipitates insoluble lead oxalate. This is collected, washed, suspended in water, and sulphuretted hydrogen passed into the turbid fluid. Lead sulphide is thus precipitated, and after filtering the solution on evaporation deposits oxalic acid.

Two tests sufficiently characterise oxalic acid, if sufficient material is available. (1) Warmed with strong sulphuric acid, oxalic acid (and its salts) gives carbon monoxide and carbon dioxide, and does not char. (2) Calcium chloride precipitates calcium oxalate from a neutral solution. The calcium oxalate is insoluble in ammonia and in acetic acid (distinction from tartaric, citric, malic, and succinic acids).

In medico-legal work oxalic acid or a salt must be recovered from alimentary contents or vomit. The detection of calcium oxalate in the urine is not evidence, especially with the herbivoræ, in which a diet of sorrel will cause increased excretion of this salt.

ALCOHOL.

Occurrence.—Alcohol results from the fermentation of sugar by yeast, and thus enters into the composition of ale, wine, and distilled liquors. The latter rarely exceed a strength of 50 per cent. by weight of alcohol.

Acute alcohol poisoning is very occasionally observed in animals, and, as in man, follows the consumption of a large dose of pure spirit or of spirituous beverage. The chronic alcoholism of man is not observed in animals, possibly from the inaccessibility of alcoholics, for goat and sheep are stated to quickly acquire a liking and tolerance for spirits, taking 6 or 8 ounces of brandy without serious effect (Hertwig). Ducks, fowls, and parrots also take alcohol readily after having had it given a few times.

Alcoholic intoxication is stated to occur amongst stock fed on brewery and distillery residues, but the statement must be taken with reserve, for in such residues the dilution is very great, and this appears to be a most important factor. A dose of alcohol which, when highly diluted and slowly consumed causes no harm, would, if concentrated and given in one dose, cause grave intoxication, or even death. Cases of death in man after taking a few ounces of undiluted spirit in one draught are common, and the poisoning is often fatal in a very short time.

Toxic Doses.—According to Hertwig, 8 ounces of concentrated alcohol caused the death of an old but sound horse in about ten minutes. Four to five ounces of whisky (of about 45 per cent. alcoholic strength), if retained, kill a 20-pound dog in a few minutes (Finlay Dun). Eight grammes (about 120 grains) per kilogramme (about 2 pounds) body weight has been stated as the toxic dose of alcohol. The higher alcohols, propyl, butyl, and amyl, are more toxic and more irritant than ethyl alcohol, or common alcohol. Crude spirit, however, undoubtedly owes a part of its noxious qualities to the presence of aldehydes, which are the first oxidation products of the alcohols, and which are only slowly eliminated from raw spirit in the process of maturation.

Symptoms.—Alcohol in large doses paralyses the nerve-centres in the order of their development, the higher cerebral functions being first affected, the cardiac and respiratory last. In animals the motor paralysis thus declares itself as a prominent feature, as compared with the mental derangement of man.

In acute poisoning of animals there is a period of great excitement, during which the patient exhibits brightness of the eye, contraction of the pupils, and irregular movements. The horse prances and strikes out with its feet. Very soon there is collapse, with a small, weak pulse, coldness, coma, and death (Gamgee, Finlay Dun).

Post-Mortem Appearances.—The digestive organs show irritation after concentrated doses. The blood is dark,

and clots are found in the heart and large vessels. There is congestion of the meninges of the brain, of the lungs, and other organs.

Treatment.—Antidotes are tea, coffee, or caffeine. Strychnine is a physiological antagonist, and may be injected. Ammonia as a stimulant by the mouth, and purgatives, are indicated.

Chemical Diagnosis.—The separation of alcohol from tissues is easy, but its exact identification is surrounded by pitfalls. Distillation of the parts from a neutral solution will yield the alcohol in the first part of the distillate. If the quantity permits, the concentration may be increased by redistillation and dehydration with quicklime. If the alcohol can thus be got free of water, the boiling point (78° C.) may be observed even with very small quantities, and taken along with the iodoform test, is sufficient to absolutely identify. The iodoform test depends on the formation of the very characteristic iodoform on gently warming a dilute solution of alcohol with sodium carbonate and a scrap of iodine. It is, however, given by other compounds—*e.g.*, aldehyde and acetone—and therefore taken alone is not characteristic. In medico-legal work it is valuable evidence to show that alcohol is present in the blood and in the brain.

CANTHARIDES.

Occurrence and Uses.—The Spanish blister-fly, *Cantharis vesicatoria*, is found in Southern Europe, Germany, and Russia, and contains a powerful vesicant active principle, *cantharidine*. The powdered insects form the ordinary cantharides of pharmacy, and are sometimes adulterated with euphorbium and the China blister-fly (*Mylabris*). Characteristic of Spanish fly is the brilliant coppery-green of the wing sheaths. The Chinese fly is larger, has two orange-coloured bands and spots on the wing sheaths. Cantharides, besides its use as a vesicant, is employed

against excessive urination, and in large (dangerous) doses as an aphrodisiac.

Poisonous doses of cantharides, according to Gamgee,* are, for the horse or ox, ½ ounce and upwards; for the sheep, 1 drachm; and for the dog, ½ drachm. It is dangerous to employ too large or too extensive applications of cantharides blisters, for poisoning may result from absorption. Dogs lick the blistered parts, and this leads to swelling and engorgement of the tongue.

Symptoms.—Large doses of cantharides cause strangury, frequent passage of small quantities of urine, or its total suppression, a small and rapid pulse, quickened breathing, and excitement, followed by coma and collapse.

An interesting case of poisoning of a horse is recorded by H. King.† The animal had 8 ounces of a mixture composed of 5 ounces each of linseed and turpentine oils, and 14 drachms of powdered cantharides in mistake for linseed oil.

Next day the mouth and lips were seen to be blistered, the horse was blowing slightly, and passing large quantities of urine. The pulse was 90, very quick and feeble, and temperature 100° F., soon becoming subnormal. The whole of the mucosa of lips and mouth became blistered, and subsequently destroyed, the mouth, throat, and neck being very painful, causing much dribbling. There was constipation and refusal of all food for five days, when the patient took some oatmeal gruel and soft food. Belladonna and nutrient enemata were given by the rectum.

On the sixth day the urine contained blood, and there was slight abdominal pain. Injections of ether were given thrice daily, but death occurred on the tenth day.

On **Post-Mortem** the kidneys were found to be much inflamed, and each contained large abscesses. There were large hæmorrhagic spots on the bladder, and an ulcerated patch the size of a crown piece in the stomach. The in-

* 'Veterinarian's Vade-Mecum,' 1868, p. 209.
† *Vet. Jl.*, 1907, p. 270.

testines and lungs were much congested, and the endocardium of an intensely deep purple hue.

In the **Treatment** of cantharides poisoning mucilages and albuminous draughts, such as linseed tea, white of egg and the like, are indicated. Oils are to be avoided, as they favour the solution and absorption of the poison.

Cantharidine is present in all organs after death, but is especially to be sought in the urine. It is insoluble in water, but is dissolved by caustic alkalis. From urine, or an alkaline extract of organs, cantharidine, is extracted by chloroform after acidification. The only *test* of any value is the observation of the blistering effect which is produced by $\frac{1}{100}$ grain.

POISONOUS PLANTS

It will very readily be admitted that anything approaching to a full enumeration of the plants which are, or are suspected to be, poisonous to animals, still more a reliable account of their effects, would be a task of extreme difficulty. Moreover, a mere catalogue has little value. The attempt has therefore been made to collect primarily well-substantiated information relating to common poisonous plants. This naturally involves a preferential treatment of British and European genera, since there have been for a longer time opportunities of exact study. At the same time, no doubt can be entertained that many poisonous species cause harm in the East, in the Tropics, in America, and in the Colonies, and that probably in time our knowledge of such poisonings will be very extensive. But the author's Colonial correspondents agree in regarding the state of our present knowledge as too slender and inexact to warrant, or even to make possible, extensive descriptions, either botanical or clinical. Valuable work has been done, and is still going on, in America and the Colonies, and the departmental publications and agricultural journals already contain precise information on many important poisonous plants. Thus the Report for 1898 of the United States Bureau of Animal Industry contains a summary of numerous American species, by V. K. Chesnut; several important papers have appeared in the Transvaal, Cape, and later United South Africa agricultural journals; the home literature contains a few papers by Colonial veterinarians; and in South Africa L. H. Walsh has collected the available facts in a short but exceedingly valuable brochure.

All these sources of information have been freely drawn upon. As to the East, F. N. Windsor, in a useful little book on 'Indian Toxicology,' remarks, after having described a few well-known plant poisonings: 'So far as is known, there are no other commonly used poisons. Undoubtedly there are numerous other poisonous plants in India which are occasionally used for criminal purposes. However, very little has been recorded as to their effects and toxicology generally.' A similar remark seems applicable to Australia. But here, again, the labours of the tropical disease and agricultural institutes—such as that of Ceylon—will no doubt slowly unfold a wide field of knowledge. Many tropical plants have been well known for a long time as the sources of valuable drugs, many of which have received treatment in the preceding section. They are not repeated in the present connection, although it is possible that the plant may act as a vehicle of poisoning to animals.

As to Europe, the work of Cornevin, 'Des Plantes Vénéneuses,' 1893, is an invaluable standard manual, to which the author is greatly indebted. In our literature there is a fairly large number of papers and abstracts on vegetable poisoning, many of which have been drawn upon for information.

After considering possible alternatives, it finally appeared most satisfactory to deal with the poisonous plants in the sequence of their natural orders, although it is arguable that a more rational procedure would be to classify according to the pharmacological subdivisions of the active principles. But such a classification is not satisfactory because not precise, and it seemed better to follow the line of least resistance—or, it is hoped, of least controversy.

The writer would be most grateful to any foreign or Colonial veterinarians, or others interested who may chance to be readers, for any information relating to cases of animal poisoning which they may have encountered, and in particular those cases in which the plant implicated has been identified botanically.

CONIFERÆ.

The chief poisonous species of the **Coniferæ**, or pine family, found in Great Britain and Europe, are *Taxus baccata*, or yew, and the shrubs of the juniperus species, such as savin. Poisoning by turpentine and savin have been described under a previous heading, and therefore a description of yew poisoning only will be given at this point.

FIG. 1.—TAXUS BACCATA (COMMON YEW).
(From Smith's 'Veterinary Hygiene.')

Yew.—The leaves of the common yew, or *Taxus baccata*, and its varieties, such as the Irish yew (*Taxus fastigiata*), and yellow yew, have long been known to be poisonous, and contain as active principle the alkaloid taxine. The same active principle is probably contained in the American species, *Taxus minor*, found in the North-Eastern United States, and known there as common yew, ground hemlock, or poison hemlock.

This alkaloid occurs in the leaves of all species, but only in small proportion in the berries. According to Thorpe and Stubbs* the undried leaves yield on extraction from

* Transactions of the Chemical Society, 1902, p. 874.

0·1 to 0·18 per cent. of taxine. It seems likely, further, that the leaves of the male contain slightly more alkaloid than those of the female tree, but the difference is trifling.

Much controversy formerly existed as to the poisonous effects of yew, it having been held that the poisonous qualities vary with the season, with the freshness of the leaves, and with the species of the animal. But there can no longer be any doubt that the leaves at all times may be poisonous. The alkaloid may be easily separated and detected from dried or undried leaves. Well-authenticated examples of poisoning among the domesticated animals are very numerous, and the definite toxicity of the alkaloid extracted chemically from the leaves may be readily observed upon experimental animals. Such variations as have been observed are readily comprehensible in consideration of the known variability in the action of any poison according to the condition of the alimentary system and individuality of the subject. When a few sprigs of yew are eaten by an animal on a full stomach, it is quite to be expected that dangerous results may not ensue.

Action and Toxic Doses.—The action of yew, as of so many plants, is twofold. The sap is acrid, containing a volatile oil, or oil of yew, and the plant therefore produces irritation. The specific poison, or taxine, is non-irritant. It acts as a narcotic, producing, according to Borchero (1876), depression of the heart, paralysis of the respiratory functions, and death by suffocation. It has been alleged that the guinea-pig is not susceptible.

According to Cornevin,* the poisonous doses are—

	Grains per 1 Pound Body Weight.
Horse	15
Ox	75
Sheep	75
Goat	90
Pig	22

* Cornevin, 'Des Plantes Vénéneuses.'

The doses refer to ingestion of autumn and winter leaves, and show, as is usual, a relatively great degree of resistance on the part of the ruminants.

There are several recorded instances of yew poisoning in man, generally of lunatics, from which it appears that the poisonous dose of leaves for the human subject is small. In these cases, quoted by Taylor 'On Poisons,' it is noteworthy that the narcotic effects predominate, and it is also observable that, as with animals, a comparatively short period of time elapses between dosage and the onset of symptoms and death.

Symptoms.—A remarkable feature of yew poisoning is its rapidity.[6,8] Often the effects only appear in cattle when chewing the cud. Whilst quietly chewing, they drop as if shot (Wallis Hoare).[4] In some examples recorded in the literature the animal died whilst in the act of eating the plant,[7] or was found to have fallen and died suddenly and without evidence of a struggle. The animal will stop suddenly whilst working, start blowing and trembling, stagger, fall on haunches, then on side, and die quietly. Death occurs in about five minutes, with symptoms like apoplexy.[13] A case of death after sixteen to seventeen hours of a colt is recorded by Jarvis,[5] who points out that the plant was taken on a full stomach, but that paralysis of the alimentary system, with stoppage of digestion, immediately ensued.

Post-Mortem Appearances.—The stomach is generally distended with gas, owing to fermentation following the arrest of digestion. Intense inflammation is almost invariably observed, but does not appear to be a salient feature in the cases amongst the human subject quoted by Taylor. The stomach is found to contain dark green ingesta,[3] and sprigs, berries, or leaves of yew may be easily recognised, but if dry sprigs were eaten they may not be recognised. The inflammation rarely extends to the intestines,[10,11,12] which also rarely contain fragments of yew. The liver, spleen, and lungs are engorged with dark blood. The right heart is empty, and the left heart contains more

or less of dark, tarry looking blood. Similar observations are recorded by Gillam[1] in the case of the pig.

Treatment.—The prognosis of yew poisoning is grave. Cases of recovery are few, but one is recorded by M'Phail.[2] An emulsion in sodium bicarbonate solution of 1 pint linseed oil, with 2 ounces each of chlorodyne and nitrous ether, was given, followed after some hours by whisky and linseed oil. Stimulants and chlorodyne were repeated next day. After a purge and further dose of opiate, recovery ensued after the fourth day.

Another similar case is that recorded by Stanley,[9] in which the remedial measures were bleeding, sodium carbonate, and turpentine, followed by drenches and stimulants.

When possible, purgatives and demulcents are indicated, and stimulants, caffeine, alcohol, etc., to combat the narcotic action of the taxine. But so long as much yew remains in the rumen, medicinal treatment is of little use. If the diagnosis is certain, rumenotomy offers the best chance of success (Wallis Hoare).

Chemical Diagnosis.—The alkaloid taxine is extracted, though not without loss, in the general scheme of search for vegetable poisons. Risk of decomposition is minimised by operating at a low temperature, as is done by evaporating extracts in a partial vacuum. Taxine is not well defined chemically, it being not absolutely established that it is a single substance. But the substance extracted in the usual way is characterised by giving a pure rose-pink colour with strong sulphuric acid, which is fairly permanent and withstands dilution. Laboratory experiments with the rabbit have shown that taxine yielding the above test can be easily recovered from the stomach contents.

In practice, however, the finding of yew fragments offers an amply sufficient proof of poisoning, taken along with the clinical and post-mortem observations, and it is also to be noted, as a caution, that many other substances give a red colour with sulphuric acid. But of these many are not basic, and can therefore be distinguished from taxine, which

is; whilst the veratrine and hellebore principles do not give the red colour in the cold, and, further, also, give other characteristic tests. Finally, a physiological test on a mouse or rabbit should be used in additional confirmation.

REFERENCES TO YEW.

[1] W. Graham Gillam, *Vet. Record*, 1906, p. 88.
[2] J. M'Phail, *Vet. Jl.*, 1900, p. 27.
[3] G. E. Nash, *Vet. Record*, 1900, p. 271.
[4] E. Wallis Hoare, *Vet. Record*, 1893, p. 588.
[5] H. Jarvis, *Vet. Record*, 1893, p. 398.
[6] F. Earl, *Veterinarian*, 1875, p. 183.
[7] B. H. Russell, *Veterinarian*, 1875, p. 326.
[8] C. Stephenson, *Veterinarian*, 1859, p. 381.
[9] F. T. Stanley, *Veterinarian*, 1859, p. 450.
[10] J. E. Cornelius, *Veterinarian*, 1859, p. 697.
[11] J. Chapman, *Veterinarian*, 1858, p. 123.
[12] G. Waters, *Veterinarian*, 1854, p. 386.
[13] R. Read, *Veterinarian*, 1844, p. 255.

ARACEÆ.

The only member of the **Araceæ**, or arum family, found wild in Britain, is the *Arum maculatum*, known under the common names cuckoo-pint, lords-and-ladies, wild arum, water robin, Portland sago, etc. Cornevin further names *A. italicum*, *A. dracunculus*, and the marsh plant *Calla palustris*, as being similar to *A. maculatum* in effects.

The **Cuckoo-Pint** (Fig. 2) is a familiar hedgerow plant having a tuberous root-stock, flowering in May, and having clustered scarlet berries in August. The leaves are glossy, halberd-shaped, and spotted. Cases of the poisoning of animals by it are rare, although several cases, chiefly of children, are on record in human toxicology. Numerous other species of the arums have similar toxic properties, but they do not appear to have caused poisoning of animals to a notable extent.

Active Principle and Effects.—Like the allied species, this plant contains an acrid juice of unknown chemical nature, and has the effect of a powerful irritant. The juice

is found in all parts, and drying or boiling to a great extent deprives the plant of its activity. Formerly starch for laundry purposes, and a kind of sago used to be made from

Fig. 2.—Arum Maculatum (Cuckoo-Pint).

the plant, and preparations of the root also enjoyed some repute as cosmetics. The old herbalists used to recommend the juice as a purgative, a practice which must have been attended with grave risk.

Animals do not eat the plant readily, even if kept from other foods. If a dangerous quantity is taken, there is intense irritation, purgation, and vomiting when possible, with the after-effects of convulsions, exhaustion, and possibly death from shock.

In the **Treatment** of a case of poisoning demulcents and stimulants are indicated.

IRIDACEÆ.

The **Iridaceæ**, or iris family, includes the numerous cultivated species of our gardens, and there are found in the wild state, *Iris pseudacorus*—yellow iris, yellow flag, or water flag; and *Iris fœtidissima*—stinking iris, stinking gladwyn, or glader. They contain as active principle *iridin*, a glucoside belonging to the group of vegetable purgatives. Poisoning of animals is rare, and the effects are those of a drastic purgative. It will thus be sufficient to signalise the possibility of danger through the giving of parts of such plants to animals, especially to pigs.

The American *Gyrotheca capitata*, red-root, or paint-root, of the Atlantic coast and Cuba, belongs to the related order of *Hæmodoraceæ*, and has a red sap. It is supposed to be dangerous to pigs.

AMARYLLIDACEÆ.

The **Amaryllidaceæ**, or amaryllis family, includes the numerous species of narcissus or daffodil, so well-known as garden plants, and of which some are frequently found wild, probably having established themselves from garden culture. The same order also includes the *galanthus*, or snowdrop. None of these is likely to be the cause of poisoning, as animals refuse the leaves. They all contain an essential oil, and have powerful emetic and purgative properties. The atamasco lily, *Atamosco atamasco*, of the South Eastern United States, has been alleged to cause staggers in horses.

DIOSCORIDACEÆ.

The **Dioscoridaceæ**, or yam family, includes one poisonous British species, namely *Tamus communis*—black bryony, ladies' seal, or Isle of Wight vine (Fig. 3). This plant is

FIG. 3.—TAMUS COMMUNIS (BLACK BRYONY).

a climber, twining over hedges, known by its heart-shaped, shining leaves, with a tapering point turning blackish in autumn. The berries are scarlet It is found extensively in England, not in Scotland, and in Ireland only on the banks of Lough Gill in Sligo.*

The active principle probably resembles *bryonin*, the purgative glucoside of the white bryony. Cornevin states that the black bryony acts as a narcoto-irritant when the fruit is taken, but that the leaves are eaten by goats and sheep without ill effect.

COLCHICACEÆ.

This order comprises some important medicinal and poisonous plants. The veratrums include *Veratrum album* and *Veratrum viride*, neither found wild in England, whilst in America, in addition, *Veratrum californicum* is noted as poisonous, as also is the officinal Mexican *Schœnocaulon officinale*. *V. viride* is known in the Northern and Eastern United States as swamp hellebore, American white hellebore, false hellebore, or Indian poke. Sheep are said to eat the young leaves and shoots with apparent relish, but the seeds are poisonous to fowls. For an account of veratrum poisoning reference may be made to veratrine. Other dangerous American species of this order are *Chrosperma muscætoxicum*, fly poison, or crow poison, the bulbs of which, mashed with molasses, are used to stupefy flies ; *Zygadenus venenosus*, or death camas, in distinction from the true edible camas (*Quamasia quamash*) ; and *Z. elegans*, or alkali grass. The most important and best known from the point of view of toxicology of this order is *Colchicum autumnale*, or meadow saffron, and the European literature contains several records of the poisoning of animals by it.

Colchicum autumnale—the common colchicum, meadow crocus, or meadow saffron (Fig. 4). 'At the time of flowering, in August, there are no leaves, the brown bulb ending

* Bentham and Hooker, 'British Flora,' 1908, p. 455.

in a sheath of brown scales, enclosing the base of the flower, whose long tube rises to 3 or 4 inches above ground, with six oblong segments of a reddish-purple or rarely white,

FIG. 4.—COLCHICUM AUTUMNALE (MEADOW SAFFRON).

and nearly $1\frac{1}{2}$ inches long. Soon afterwards the leaves appear, and attain in spring a length of 8 or 10 inches, by about 1 or $1\frac{1}{2}$ inches in breadth. The capsule is then

raised to the surface of the ground by the lengthening of the peduncle, soon after which the leaves wither away.'* The habitat is in moist meadows and pastures. It is rare in Ireland, and naturalised in Scotland.

Toxic Principle and Doses.—Colchicum contains in all parts the active alkaloids *colchicine* and *colchiceine* (about 0·05 per cent.), which is not destroyed on drying or on boiling, passing into the water. Poisoning of animals may result in the spring from the eating of the young leaves, or in autumn through the flowers in pastures.

Cornevin estimates the toxic dose of green leaves per pound body weight of the ox at 60 to 75 grains, and of the bulb for pigs at 3 grains per pound.

Colchicine, being absorbed slowly, only exercises its effects after a comparatively long period, and is gradually eliminated, mainly by the urine and milk, so that there is danger of a cumulative effect. Its most marked effect is as a violent purgative, animals suffering from it passing fœtid, green or black evacuations. The nervous symptoms of stupor, coma, and paralysis, are probably referable to the general collapse, rather than to a specific action. Death occurs from respiratory failure.

Symptoms.—The general toxic symptoms occur after several hours, and death up to several days, according to the amount ingested. Nausea, abdominal pain, violent purgation, sometimes prolapses of the rectum, cessation of urination, and lactation, tympanites, gritting of teeth, weak pulse, coldness, progressive loss of power from posterior onwards, are features of this poisoning ([1, 2, 3, 4]).

Barret and Remlinger[4] observed sudden illness of thirty-one out of fifty-one cattle, and five deaths. Calves showed slight affection through the secretion of the poison into the milk.

Post-Mortem Appearances are those of acute gastro-enteritis, the rumen distended, and probably containing leaves or seeds of the plant. The other organs do not show notable or characteristic appearances.

* Bentham and Hooker.

Treatment should consist of oily and mucilaginous drinks; there is no satisfactory chemical or physiological antidote. Barret and Remlinger used milk drenches, with egg-white and black coffee, injections of caffein, and external applications of mustard.

Chemical Diagnosis.—The alkaloids of colchicum are feebly acid, and are separated in the course of the general search for vegetable poisons. The residues have an acrid bitter taste, are coloured yellowish-brown by concentrated sulphuric acid, and blue passing to olive-green and yellow by nitric acid.

REFERENCES TO COLCHICUM.

[1] Whitemore, *Veterinarian*, 1861, p. 455.
[2] Guilmot, *Veterinarian*, 1861, p. 738.
[3] W. Litt, *Veterinarian*, 1860, p. 429.
[4] Barret and Remlinger, *Vet. Jl.*, 1912, p. 306.

LILIACEÆ.

There are many plants of this order more or less certainly known to be poisonous, and very widely distributed. The European species found in Britain include—

Paris quadrifolia—Herb Paris, or four-leaved grass—is a rare plant, widely diffused over the temperate zones, but local in England, and not found in Ireland. It grows in woods and shady places, has a whorl of four ovate leaves from 2 to 4 inches long, and bears bluish-black berries. All parts are stated to be toxic, and to contain an active glucoside, *paradin*.

Convallaria majalis—the well-known lily of the valley—contains the glucoside *convallamarin*, which belongs to the digitalis class as regards its physiological effect. The plant and its extracts are thus very dangerous, though few cases of poisoning are to be found. According to Cornevin, 4 drops of the extract injected into the veins killed a dog in ten minutes. For the effects of poisonous doses reference may be made to digitalis, and it is perhaps desirable

that attention should be drawn to the possible danger of this common plant.

Fritillaria meleagris—snake's head, fritillary, drooping tulip, or chequered daffodil—a bulbous herb, having lanceolate leaves, and bearing single red or pink flowers, grows in meadows and moist places in a few localities in the South and East of England, but not in Scotland. Poisoning by it is not common. The plant is stated to contain a glucoside, *imperialin*, but little is known of its effects, and there are no cases of accidental poisoning on record. There can be little doubt that this plant resembles the allied species, *F. imperialis*, in its actions.

No record of poisoning by the British squills—*Scilla verna*, the spring, and *S. autumnalis*, the autumnal squill—is to be found, although related plants are officinal and poisonous, The onion, *Allium cepa*, has been observed to be poisonous (see under Mustard).

In the United States *Leucocrinum montanum* has been reported to be very fatal to sheep in Montana, especially after the fruit is developed, and *Nothoscordum bivalve*, crow poison, or yellow false garlic, was reported as very dangerous to cattle in Texas in the spring of 1898.

Amongst Indian poisonous plants of this order mention may be made of *Gloriosa superba*, which contains a glucoside, *superbim*, allied to *scillain*, the active principle of squills.

Urginea.—The officinal *Urginea maritima*, medicinal squill, or sea onion, is a maritime plant common on the Mediterranean littoral, and in the Cape, and is very abundant in Algeria, where the poisoning of pigs and of young animals has been observed (Cornevin). The lanceolate leaves grow from the base of the flowering stem, and die before flowering. The greenish flowers are numerous on long pedicles in an erect raceme. The root is a bulb covered with scales, and about 6 inches in size. The plant reaches 1½ to 2 feet in height.

All parts are poisonous, but chiefly the bulb, and the active principles are *scillain* and *scillitoxin*, acting in an analogous manner to digitalis. According to Cornevin, the probable

toxic doses by the mouth per kilogramme body weight are, for the horse, 20 centigrammes; for the ruminants, 50 centigrammes; and for the pig, 25 centigrammes.

Symptoms.—When taken in a large dose, or in repeated moderate doses, the diuresis, obscured with small doses, gives place to anuria and hæmaturia; there is nausea and diarrhœa with colic in ruminants. When possible, vomition occurs. The respiration is laboured and pulse quickened, and there is agitation and convulsions, followed by a phase of prostration and death. Cornevin states that, in spite of the smell of the plants, pigs in Algeria have died through eating the plant eradicated from pastures and thrown on to the roadside, whilst young animals have succumbed to the leaves in pastures infected by the plant. The *lesions* are those of alimentary and renal irritation.

South African Slangkop.

Two plants are incriminated, on well-authenticated evidence, as the causes of the poisoning, known as slangkop poisoning in the Transvaal and the Cape—viz., *Urginea Burkei*, the Transvaal slangkop, and *Ornithoglossum glaucum*, the Cape slangkop. No research appears to have been made on the active principles involved, but it is possible that these plants owe their poisonous properties to glucosides allied to scillain, but Cape slangkop is said to resemble *colchicum* in its poisonous properties.

Botanical Characters.—The Transvaal slangkop has a reddish-brown bulb from 3 to 4 inches in diameter. The outer scales are easily removed, and have a blood-like colour when held up to the light. With the first rains it puts up a succulent stalk bearing the green flower-buds, and resembling a snake's head, whence the vernacular name, slangkop. The bluish-green leaves form a spike of from five to seven leaves about 6 to 9 inches long, $\frac{1}{2}$ inch wide, with curved edges, and tapering to the point. The flower-spike and leaves may thus each cause poisoning at different seasons.

The Cape slangkop has a flowering stem of from 6 to 9 inches, scentless green flowers edged with purplish-brown. The corm is small, egg-shaped, and has a thin underground neck 2 or 3 inches long. The leaves are shiny green, long, and lanceolate.

Symptoms. — Poisoning by slangkop is marked by diarrhœa, stiffening, and paralysis of the limbs, a fixed and staring look, coma, and death.

The effects of slangkop on sheep were studied by J. T. Dumphy.* He states that the toxicity may vary in different districts, that three flowering heads may kill, and that two to three days may elapse before the onset of symptoms. These are not very characteristic. The animal becomes dull and weak, leaves the flock, and lies down by itself. The head hangs, the conjunctivæ are injected, and heart action irregular. Diarrhœa is excessive. In aggravated cases the patient lies on its side, throwing its head about.

Post-Mortem Appearances.—These are not characteristic. There is patchy inflammation of the intestines and fourth stomach, and the brain is congested.

Treatment.—The treatment is directed against the inflammation by means of oils and lime-water or laudanum. Dumphy recommends a rapid purge of Epsom salt, or $\frac{1}{8}$ grain arecoline in $\frac{1}{2}$ ounce hypodermically for sheep, followed by stimulants, such as ether and brandy.

Chinkerinchee.

Botanical Characters.—The South African *Ornithogalum thyrsoides*, chinkerinchee, or viooltje, has a white bulb of $1\frac{1}{2}$ inches in diameter, and a round, green, succulent stem of $1\frac{1}{2}$ to 2 feet. The leaves are green and fleshy, and 1 to $1\frac{1}{2}$ inches wide. The flowers are white with a brown centre and yellow stamens, and form a cluster on short stalks at the top of the stems. The fruit is a capsule bearing many seeds.

Walsh (*loc. cit.*) states that the plant is a common forage

* *Transvaal Agricultural Journal*, 1905-06, p. 315.

pest in the western province of the Cape, and that, being dangerous in the withered condition, it gives rise to accidents through becoming mixed with dry forage—*e.g.*, at Kimberley.

Active Principle.—The chinkerinchee has been studied by Power and Rogerson,* who succeeded in associating the toxic action with a dark green resin. This substance is unfortunately indefinite from the chemical point of view. No alkaloid was detected. Experiments by the Agricultural Department of the Cape proved that all parts are poisonous, and that the plant is very dangerous.

Poisoning.—Walsh records dulness, depression, and loss of appetite, followed by severe purgation, with severe abdominal pain, or a drugged appearance. Horses go off their feed, but drink copiously. The purging continues, the evacuations becoming fluid. The eyes are staring and glassy, and heart affected, the beating being abnormally loud. Violent struggling, kicking, and flow of foam and mucus from the nostrils may precede death.

On *post-mortem* there is found much gastro-enteritis, the blood thick and dark, and alimentary contents fluid and stinking.

In *treatment*, sedatives (laudanum) and oily purges are given with ammonia, spirits of nitre or brandy against weakness. Alkali, such as bicarbonate ($\frac{1}{2}$ ounce), is stated to be valuable.

Tulp.

This order includes, according to L. H. Walsh, the South African 'tulps,' distributed throughout the country, some having been introduced from Australia, where their dangerous character had been noted. They thrive in various localities—in sandy soil, in vlei lands, and on hillsides. Tulp is frequently a prominent plant after the first rains on burnt veldt. Three species are specially noted—viz., Groot tulp (*Homeria collinia*), Klein tulp (*Moræa sp.*), and Blauw tulp (*Moræa sp.*).

* *Pharmaceutical Journal*, 1910, p. 326.

Botanical Characters.—*Groot tulp* (greater tulp) reaches about 2 feet, with long, very narrow, tapering leaves. The root is a small round bulb. The flowers are pale yellow or reddish, 1½ inches in diameter, with orange-yellow stamens, and six petals. The seeds are contained in long, compact, pod-like structures (Walsh).

Klein tulp (lesser tulp) is 8 to 9 inches high, and has six petals, more spread out than in groot tulp, whitish-mauve in colour, with dark mauve spots on the upper surface. The flowering stalk is light brown or brownish-green (Walsh).

Blauw tulp (blue tulp) reaches about 15 inches in height, and has long, very narrow, flexible leaves, which clasp the stem at the base. The flowers resemble those of klein tulp, but are somewhat stouter, and blue in colour (Walsh).

Poisoning.—The dangerous character of the tulps seems established beyond all doubt, although little is known as to the active principle. It is not unlikely that it resembles the glucoside *iridin* found in other species of the *Irideæ*.

Bowhill * has described tulip-grass poisoning of horses in South Africa, and remarks as the symptoms—Temperature, 102° F., rising to 104° F.; pulse thready and strong; loss of appetite, dulness, and drooping of head and ears; the mouth is burnt, there is some frothing, gritting of teeth, and in some cases arching of neck, regurgitation of gas, and attempted vomition; the colic, slight at first, increases rapidly in severity, and in about two hours there is well-marked tympanites; finally, there is coma, in some cases convulsions, and death in five to ten hours.

A. J. Williams † distinguishes *subacute* cases with tympanites and the usual symptoms of flatulent colic, and *acute* cases, in which there is extreme tympanites, the animal dashes about with staggering gait, pupils dilated, convulsive twitchings, lips retracted, and symptoms of asphyxia. The patient falls and dies immediately.

The plant incriminated by Williams is described by him as having a plant stalk about 6 inches long, a yellow

* *Record*, 1900, p. 229. † *Ibid.*, 1902, p. 421.

tulip-like flower, a grass-coloured leaf growing to about 12 inches along the ground, and a bulb about the size of a filbert.

From the accounts given, it is not unlikely that there are some variations in the character of tulp-poisoning according to species and locality.

In the *treatment* Bowhill gave large doses of turpentine, linseed oil, and carbolic acid, with hypodermic injection of 1 grain of eserine, and tapping of the distended bowel. Williams advised puncture with trocar and canula on both sides, if necessary, and 2 ounces each of turpentine and tincture of opium, with a pint of linseed oil.

The *lesions* are those of acute gastro-enteritis, with large ecchymosed patches at the entrance to the pylorus (Bowhill).

Addendum.

A. J. Williams, in the same paper (*loc. cit.*) also refers to two plants only met with in the Karoo district—namely, the sterkos, or pepper-bush, and the ink-bush. The sterkos he describes as a small bush, with the very hot flavour of pepper. It causes acute diarrhœa and abdominal pain, which are treated with chlorodyne and linseed oil.

The ink-bush causes acute abdominal pain, diarrhœa, intensely injected mucous membranes, quickened pulse and respiration, elevated temperature, and death in six to twelve hours. Acute inflammation extends from the stomach to the rectum, and there is no treatment.

GRAMINEÆ.

Of the very large family of the grasses, **Lolium temulentum**, or darnel, is the only species found wild and native to Great Britain which is dangerous. Of exotic grasses, *Zea mais*, or Indian corn, is grown occasionally, and the young shoots are poisonous. Poisoning by ergotised or diseased rye or other fodders is referable to the parasite, and not to the grass.

L. temulentum (Fig. 5) is closely allied to *L. perenne* (Fig. 6), the rye-grass, known in two varieties—the English and Italian—of which both are cultivated, the former being the more preferred as a fodder.

Fig. 5.—Lolium Temulentum (Darnel).
(From Smith's 'Veterinary Hygiene.')

The distinguishing points between the species *temulentum* and *perenne* are—

Temulentum.—Outer glume as long as, or longer, than the spikelet. Some of the glumes with awns as long as themselves.

FIG. 6. — LOLIUM PERENNE (PERENNIAL RYE-GRASS).

(From Smith's 'Veterinary Hygiene.')

Perenne. — Outer glume shorter than the spikelet. Awns short or none.*

L. temulentum is not very common in Great Britain, but is found in South Africa, where it is known as 'drabok,' cheat, or bearded darnel, and is an introduced plant specially abundant in the Pacific slope. The poisonous properties are confined to the grains, which have a yellowish-green colour, in distinction to the violet seeds of *bromus*. The flour is colourless and tasteless, and the starch grains have a dimension of 4 to 8 μ.

Accidents due to *Lolium temulentum* commonly arise from the admixture of the grains with barley or other cereals, or from the addition of the flour to ordinary flour.

The *detection* of *L. temulentum* flour in the ordinary material may be effected by means of a microscopic examination of the starch granules; ether extracts an olive-coloured fat, which may be shown to be poisonous on a small animal; or, a flour, shaken with alcohol, will give a yellowish-green colour if *L. temulentum* is present in important amounts.

* Bentham and Hooker, 'British Flora,' 1908, p. 530.

Toxicity.—According to Cornevin, the lethal doses in grammes per kilogramme body weight are—

Horse, 7 (equivalent to about 50 grains per pound).
Dog, 18 (equivalent to about 130 grains per pound).

Ruminants and birds are less susceptible.

Symptoms.—In the horse there is dilatation of the pupils, vertigo, uncertain gait, and trembling. The subject falls, the body is cold and extremities stiff, respiration laboured, pulse slow and small, and there are convulsive movements of the head and limbs. There is rapid enfeeblement, and death within thirty hours. No special *lesions* beyond a little intestinal irritation are seen. This account is abridged from experiments quoted by Cornevin, in which 4 pounds of the grains were given to a horse.

According to the same authority, the yellow ether extract of the grain appears to act as a hyperæsthetic, causing salivation and vomition, trembling, convulsions, and tetanic rigidity. The water extractive displays anæsthetic and narcotic properties, causing drowsiness, coma, prostration, and lack of co-ordination in movements, as well as salivation and vomition.

A case of the poisoning of pigs through the admixture of *L. temulentum* grains with barley dressing was noted by Tait.* There were foaming, convulsions, and paralysis; the stomach and intestines were inflamed, and the lungs congested.

As regards *maize*, which is but rarely cultivated in Great Britain, the dangerous properties seem to be confined to the male flowers, and result in urinary disturbance, a fine yellow powder being passed or concreting to calculi. Disease from this cause is very unlikely to be met with in ordinary practice, but in South Africa the male flowers are reputed to produce poisoning, though it is also suggested that the results may be due to fatal tympanites, following an excessive feed. But it should be remembered that young maize is capable of generating prussic acid. In consideration

* *Veterinarian*, 1842, p. 212.

of the large toxic dose of nitre, there is very little plausibility in ascribing injury by maize to the presence of that salt.

Millet (*q.v.*), or sorghum, contains a cyanogenetic glucoside, *durrin*, and owes its dangerous qualities to the formation therefrom of hydrocyanic acid by enzyme action.

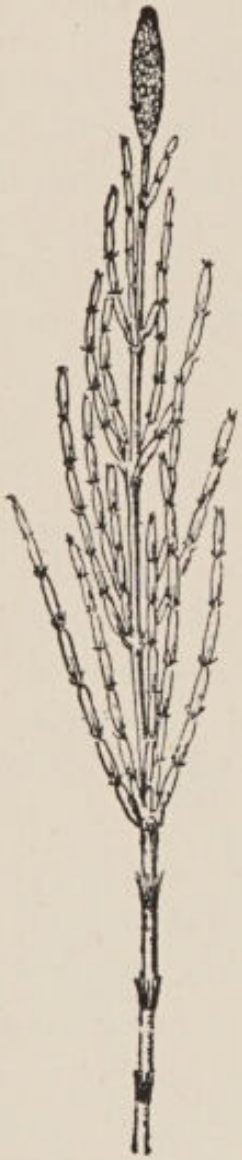

FIG. 7.—EQUISETUM PALUSTRE (HORSE TAIL).

(From Smith's 'Veterinary Hygiene.')

In South Africa and the United States poisoning by so-called 'sleepy grass,' or dronk grass, is well recognised. It is attributed to certain of the *Gramineæ*, and to varieties of the horse-tail, or *Equisetum* (*Equisetaceæ*), which, for convenience, are included at this point. The common European horse-tail, or *E. palustre* (Fig. 7), is classed as poisonous, though, unless given in forage, there is not much likelihood of mishap from it. In Connecticut, in 1871, *E. arvense*, the field horse-tail, was reported as poisonous to horses, but such cases are rare, and it has been suggested that the physical nature of the food was to blame. In South Africa *E. ramosissimum* is found in damp localities of the Transvaal and Cape. Amongst true grasses *Stipa robusta* is noted as a narcotic, sleepy grass in Arizona and New Mexico, and *Melica decumbens* is similarly noted in the Cape. Doubt as to the ætiology of these poisonings still exists, though Walsh quotes Matz and Ludwig to the effect that European *Equisetaceæ* contain aconitic acid.

The **Symptoms** are of narcosis, recalling drunkenness, during which horses and cattle stagger and wander aimlessly. A fatal end is apparently rare, removal from the locality and careful dieting, along with the ordinary measures of elimination, usually securing recovery.

POISONING DUE TO DISEASED FORAGE AND MOULDS.

The fungi apt to affect forage are *Ustilago carbo*, or smut, attacking grasses and grains; *Ustilago maydis*, affecting

FIG. 8.—SMUT OF OATS.

A, panicle of oats attacked from below upwards; *B*, spikelet with the fungus in an early stage of growth; *C*, free spores of *Ustilago carbo*; *D*, spores germinating and producing yeast-like buds.

(From Smith's 'Veterinary Hygiene.')

maize; *Puccinia graminis*, causing rust and mildew in grains; *Claviceps purpurea*, or ergot of rye; and *Tilletia caries*—all of which cause disease of the growing grain. Moulds affect damp, badly harvested or stored forage, and cause fermen-

tation, loss of colour and aroma, and possibly poison, it being, perhaps, more likely that the moulds themselves act as poisons. The commonest are *Botrytis*, *Penicillium*, *Aspergillus*, and *Oïdium*.

As regards active principles, the only member of this group, concerning which there is at present definite knowledge, is *claviceps*, or ergot. The physiological activity,

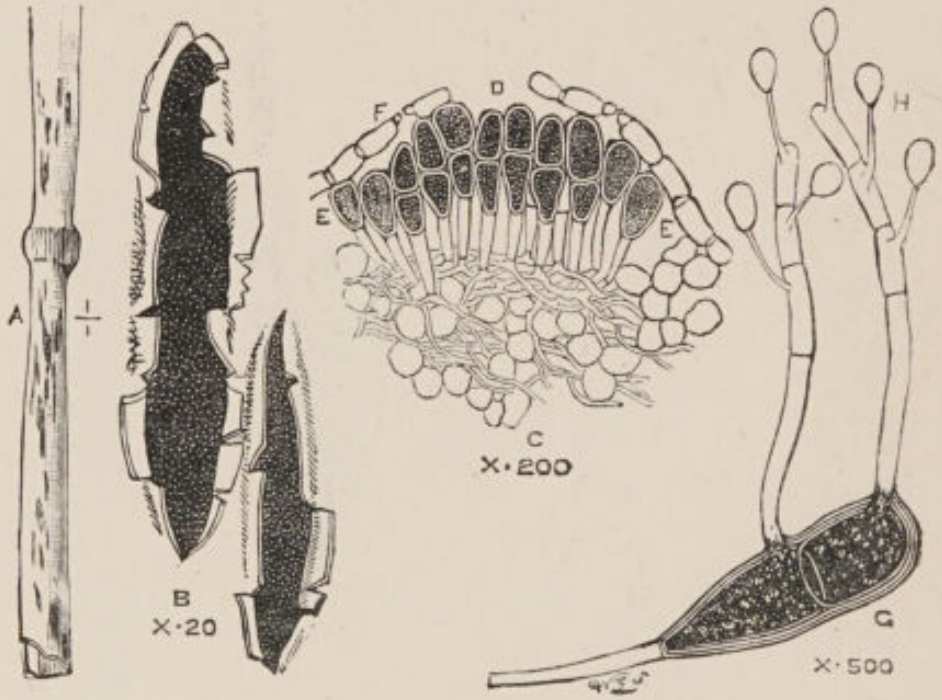

FIG. 9.—RUST AND MILDEW.

A, part of stem of oat-plant attacked by *Puccinia graminis*; *B*, two of the blotches from *A* enlarged 20 diameters; *C*, *P. graminis* within the stem, but near the surface, bursting the cuticle at *D*, beneath which are seen the teleutospores; *E*, *E*, spores of *Uredo linearis*, which sometimes surround the teleutospores of *P. graminis*; *G*, teleutospores germinating and producing sporidia at *H*. These sporidia, on germinating, give rise to *Æcidium berberidis*.

(From Smith's 'Veterinary Hygiene.')

giving rise to the well-defined ergotism of man, is attributable, according to Barger and Dale, to an alkaloid 'ergotoxine,' isolated in 1907 by Barger and Carr.* Along with it is the inactive 'ergotoxine,' and the two together do not form much more than 0·1 per cent. of the drug.

Ergot does not appear to exercise much action on rumi-

* Transactions of the Chemical Society, 1907, p. 337.

nants. W. Watson,* in one of his invaluable pioneer articles on botany as applied to veterinary science, refers

Fig. 10.—Spike of Ergotised Rye.

(From Smith's 'Veterinary Hygiene.')

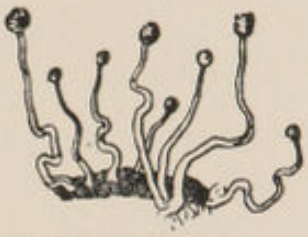

Fig. 11.—Ergots Germinating.

(From Smith's 'Veterinary Hygiene.')

Fig. 12.—Botrytis Grisea.

(From Smith's 'Veterinary Hygiene.')

Fig. 13.—Penicillium Glaucum.

(From Smith's 'Veterinary Hygiene.')

to the popular belief that cows, grazing in a field containing ergotised grasses, aborted their calves, but sheep and cows

Veterinarian, 1859, p. 574.

have been fed on quantities of ergot without dangerous effects.* According to the Continental authorities,† however, although very great doses would be required to induce acute poisoning, prolonged administration of small doses causes chronic ergotism, when besides gastro-intestinal irritation, coldness, anæsthesia, and dry gangrene of the feet, ears, and tail, or comb, tongue, and beak, of birds, ensue. The parts drop off without pain, and the disease closely resembles ergotism in man, death resulting from asthenia.

According to Kaufmann, the post-mortem appearances are gastro-enteritis, flaccid and soft viscera, muscles semi-

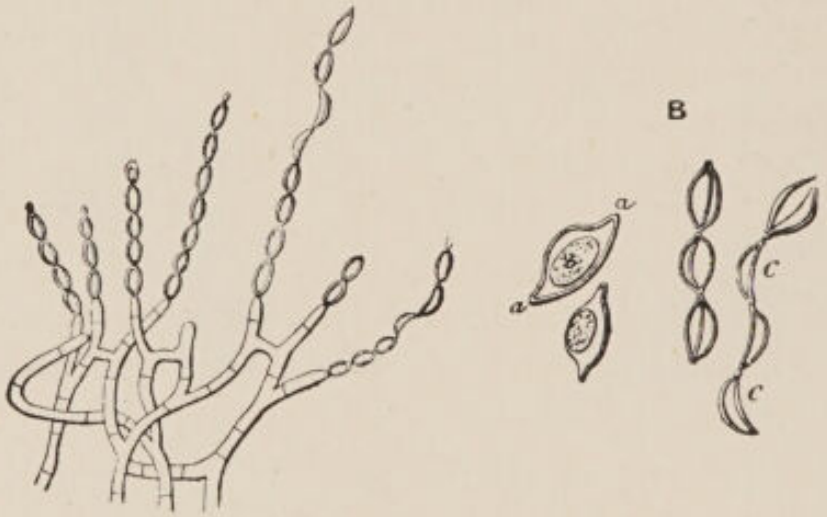

FIG. 14.—OÏDIUM AUREUM.

B, spores further magnified.

(From Smith's 'Veterinary Hygiene.')

gelatinous, blood fluid and dark, and the interior of the vessels red.

The symptoms of poisoning by MOULDS appear capable of distinction into two phases—the production of alimentary and urinary disturbances, and action on the central nervous system. Thus there are shown colic and tympany, and frequently excessive and constant urination, and also dejection, staggering gait, dilatation of the pupil, blindness, and paralysis, especially of the hind extremities.

* See F. Smith, *Hygiene*, 1905, p. 170.

† See Kaufmann, 'Traité de Materia Medica.'

Varnell* gave an old but healthy mare twelve feeds of oats, infected with *aspergillus*, during four days, and observed the above indicated nervous disorders. Death was painless on the sixth day. He found on autopsy the stomach and intestines pale and flaccid, liver paler than normal, and spleen very small.

The effects of the mould *oïdium* (on bread) on horses have been recorded by Perrin.† There were internal rumblings, frequent defæcation and passage of small quantities of urine, difficulty in moving, membranes injected, pulse 60, full and hard. Later the animal fell, the pulse was weak, there was coma followed by vertigo, and death after repeated seizures.

On post-mortem he found congestion of the mucous membranes; the liver yellow and friable, and the brain congested.

Müller‡ has described extensive disease amongst horses, cattle, and sheep, in Alsace-Lorraine, due to grain infected by *puccinia*. It was characterised by myopathic paresis or paralysis, and excessive salivation. Acute cases terminated fatally in one or two hours; subacute in from ten to forty-eight hours; and in chronic cases there was death from inanition. The post-mortem was negative.

Bansse§ signalises the effects of mouldy clover on the horse, observing delirium, with staring eyes, perspiration, and foaming, followed by weakness, loss of power of hind legs, and muscular tremors of elbow and thigh.

Aspergillus fumigatus or *glaucus* gives rise to *Aspergillosis* in all mammals and birds, causing a pneumonia with many of the symptoms of tuberculosis. It is frequently transmitted by food, or may be inhaled, and is communicable from animals to man.

* *Veterinarian*, 1862, p. 65.
† *Vet. Jl.*, 1907, p. 248.
‡ *Jl. Comp. Path.*, 1909, p. 66.
§ *Vet. Jl.*, 1903, p. 80.

RANUNCULACEÆ.

This order contains the following poisonous genera: *Clematis, Thalictrum, Anemone, Adonis, Ranunculus, Caltha, Helleborus, Aquilegia, Delphinium, Aconitum,* and *Actæa,* members of which are found wild, or are cultivated in Britain, and are widely distributed, especially over northern temperate regions. Only *clematis* is tropical. Of these the most important, from the standpoint of toxicology, are *aconitum, helleborus, delphinium,* and *ranunculus,* and these will therefore be first described.

Aconitum and Aconitine.

Botanical Characters.—*Aconitum napellus* (Fig. 15), monk's-hood or wolf's-bane, is the chief species of the genus *aconitum* found in Great Britain. 'Stem firm and erect, 1½ feet to 2 feet high. Leaves stalked, or the upper ones nearly sessile, of a dark green, glabrous or slightly downy, divided to the base into five or seven deeply cut, linear, pointed segments. Flowers large, dark blue, on erect pedicles, forming a handsome, dense, terminal raceme. The upper helmet-shaped sepal at first conceals the lateral ones, but is ultimately thrown back. Spur of the small upper petals short, conical, and more or less bent downwards. Carpels three, often slightly united at the base. Habitat moist pastures, thickets, and waste places in mountainous districts, In Britain wild in the West of England and South Wales.'*

The plant is exceedingly dangerous, owing its poisonous properties to the alkaloid *aconitine,* which is present in the root to the extent of about 2 to 4 per cent., and in less proportions in the leaves, flowers, and seeds. The maximum content of alkaloid is attained just before flowering, and the proportion is less in the plant growing in higher than in lower latitudes, and also after several generations of culture as an ornamental plant. In the Himalayas the species *Aconitum laciniatum* and *A. spicatum*—Bish, Indian

* Bentham and Hooker.

or Nepaul aconite—contain more poisonous alkaloids than the European plant.

FIG. 15.—ACONITUM NAPELLUS (MONK'S-HOOD).
(From Smith's 'Veterinary Hygiene.')

In America *A. napellus* is a garden plant, and *A. columbianum* is native to the North-West, where it sometimes poisons sheep.

Toxic Doses.—Kaufmann gives the poisonous doses of powdered root by the mouth as 13 to 14 ounces for the horse and $\frac{1}{6}$ ounce for the dog. Of the preparations of aconite the B.P. tincture is 1 in 20, and Fleming's tincture 1 in $1\frac{1}{2}$. Of the latter 120 to 150 minims are stated as poisonous to the horse, and 50 to 60 minims to a dog of 40 pounds (Finlay Dun). The alkaloid aconitine is exceedingly poisonous, $\frac{1}{2}$ grain killing a dog of 30 pounds in sixty-five minutes (Finlay Dun). The toxic dose on injection is estimated at $\frac{1}{6}$ grain for the horse and $\frac{1}{30}$ grain for the dog.

Effects.—Aconitine is speedily absorbed, and is eliminated slowly chiefly by the kidneys. Its local effect is irritant, producing tingling and twitching, followed by numbness. It acts as a gastro-intestinal irritant, causing diarrhœa. The general effect is exercised upon the medullary vagus centre, producing cardiac depression and fall of blood-pressure, and on the respiration, the breathing becoming slow.

Symptoms.—In the *horse* [1, 2, 3] there are noticed champing and copious salivation, with choking movements of the œsophagus, eructation of frothy matter, and continued attempts to vomit. The gait is staggering and pulse weak. Intense colic, purgation, and spasmodic contractions of the diaphragm, are observed. Paralysis follows, the respiration being difficult and heart and pulse weak, the pupils are dilated, membranes blanched, and temperature low; there is loss of power and sensation, convulsions, and death by asphyxia.

In the *dog* aconite poisoning is marked by salivation, nausea, violent vomiting, and purgation. The jaws are champed, and the dog rubs its nose with its paws. The heart action and respiration become progressively more feeble.

Post-Mortem Appearances.—Notable gastro-enteritis is not found in cases of rapid poisoning. The lungs contain little blood and are collapsed, the passages containing frothy mucus. The lungs are extensively studded with

patches of extravasated blood, the right heart is engorged, and the left nearly empty.

Treatment.—If possible, the stomach is to be emptied by emetics or the pump. Tannin or iodine in potassium iodide may be exhibited as alkaloid precipitants, but with doubtful value. As physiological antidotes to the depressant action on the heart, digitalis, ether, or atropine may be given. Warmth and friction will assist in stimulating the heart and lungs.

Chemical Diagnosis.—Aconitine and pseudo-aconitine (contained in Indian aconite root) are isolated in an impure condition in the routine alkaloid separation. Chemical tests are very unreliable, though when pseudo-aconitine is present Vitali's test (see Atropine) is given. Concentrated sulphuric acid or phosphoric acid, warmed with aconitine, both eventually give a reddish-violet colour. But the test is absolutely unreliable, for almost always there are traces of organic bases, or ptomaines, which behave similarly.

Aconitine induces a burning, tingling effect, followed by numbness, on the tongue or lips, and this observation has value (use with caution !). The only satisfactory proof is by an observation of the physiological effects on a small animal.

REFERENCES TO ACONITUM.

[1] R. C. Moore, *Vet. Jl.*, 1909, p. 136.
[2] W. Graham Gillam, *Vet. Record*, 1906, p. 88.
[3] C. Morgan, *Veterinarian*, 1882, p. 457.

Helleborus.

The three species of helleborus found in Great Britain are *Helleborus niger* (Fig. 16), Christmas rose or black hellebore, native to South Eastern Europe, and a garden plant in this country, *H. viridis*, green hellebore or bear's-foot, a European plant sometimes found in American gardens, and *H. fœtidus*, or setter-wort. Care must be exercised in distinguishing these from the so-called white hellebore

or *Veratrum album*, an Alpine plant belonging to the Colchicaceæ.

Botanical Characters.—Only *H. viridis* and *H. fœtidus*, which grow wild, need be described here.

Fig. 16.—Helleborus Niger (Black Hellebore).

H. viridis (Fig. 17), green hellebore or bear's-foot: 'Radical leaves large, on large stalks, divided into seven to eleven oblong, acute, toothed segments, 3 to 4 inches long, the central ones free, the lateral ones on each side connected together at the base, so as to form a pedate leaf. Stem

scarcely exceeding the leaves, bearing usually two, three, or four large drooping flowers of a pale yellowish-green, and at each ramification a sessile leaf, much less divided than the radical ones, and the segment usually digitate.'*

Fig. 17.—Helleborus Viridis (Green Hellebore).

H. fœtidus (Fig. 18), or setter-wort: 'Lower leaves not all radical, but mostly raised on the short perennial base of the stems, forming a larger and thicker tuft than in *H. viridis*, their segments narrower, less toothed, stiffer, darker green,

* Bentham and Hooker.

and more shining, their outer lobes at a less distance from the central ones. Flower-stem about a foot high, with a large close panicle of drooping flowers, of a pale green, tinged at the apex with purple, the concave sepals giving them a

Fig. 18.—Helleborus Fœtidus (Setter-Wort).

globular form. Bracts at the ramification of the panicle ovate and entire, or shortly lobed at the summit.' *

Habitat of both species chiefly South-Eastern England.

All parts of the hellebores are poisonous, particularly the root, whence the extract is made. The tincture at one

* Bentham and Hooker.

time had repute as an abortive, but is not now used, and *H. fœtidus* used to be given for quarter-evil. The *active principle* is the glucoside *helleborein*, along with a little of the less powerful *helleborin*. The action is similar to that of digitalis (*q.v.*).

Toxic Doses.—Cornevin gives 9 ounces of the fresh root as poisonous to the horse. The dried root he states as toxic in 2½-ounce doses to the horse, and 120 to 150 grains to the sheep.

Mayer records* that a horse had in all 5 half-pints of the chopped leaves of *H. fœtidus* in a bran mash during two days, and was fatally poisoned.

Symptoms.—A full dose of hellebore causes in the horse and ox bloody purgation, salivation, attempts to vomit, and excessive urination. Mayer similarly found violent straining and the discharge of frothy mucus, but no effort to vomit. The heart action resembles that observed in digitalis poisoning, showing periodic intervals of arrest in systole.

Post-Mortem Appearances. — Hellebore acts as an irritant, and congestion of the fourth stomach and small intestine have been recorded. The rumen is full, but the fourth stomach and intestines may be empty, the inflammation of the pylorus preventing the passage of food.

Treatment.—This consists of purgatives, mucilaginous draughts, stimulants, and in general measures similar to those employed against digitalis.

Chemical Diagnosis.—The glucosides of hellebore are yielded to solvents in the systematic extraction of the acid liquid in the search for organic poisons. There are no satisfactory tests. Sulphuric acid on warming causes a coloration passing from pink to red-violet, but a similar reaction is given by many other substances.

Delphinium.

Delphinium Staphysagria (Fig. 19), or stavesacre, a native of the South of Europe, is not found wild in Great Britain. The

* *Veterinarian*, 1847, p. 5.

seeds of the plant contain four alkaloids—*delphinine*, *delphisine*, *delphinoidine*, and *staphysagrine*, of which del-

Fig. 19.—Delphinium Staphysagria (Stavesacre).

phinine is the most poisonous, resembling veratrine and aconitine.

The powdered seeds are used as a valuable agent against lice.

In addition to *D. Staphysagria*, the species *Requienii*, *pictum*, and *consolida* occur in Central and Southern Europe, whilst in the United States *D. tricorne*, *consolidum*, *menziesii*, *geyeri*, *recurvatum*, *scopulorum*, and *trolliifolium*, have been suspected of being poisonous. S. B. Nelson* fed as much as 24¾ pounds of the fresh leaves of *D. menziesii* to sheep within five days without ill effect; but, on the other hand, E. V. Wilcox,† of Montana, killed a yearling lamb in two hours by the extract from less than an ounce of the dried leaves.

Symptoms. — Poisoning by stavesacre very closely resembles that by aconite, and, moreover, being rare, need not receive detailed treatment here. A. Macgregor‡ observed a case of poisoning of a horse, which showed dulness, excessive salivation, deglutition, and attempts to vomit. These symptoms, along with the weak pulse, display the likeness to aconite. In this case recovery followed the exhibition of a pint of whisky in a pint of linseed oil.

The *lesions* are similar to those of aconite.

Chemical Diagnosis. — The delphinium alkaloids are obtained by the extraction from alkaline solution in systematic work. Delphinoidine gives definite tests which, therefore, serve to characterise the nature of the poisoning. The other alkaloids do not interfere. (1) Concentrated sulphuric or phosphoric acid gives a brown colour, slowly passing to red-brown. (2) When a drop of sugar solution is added, and then concentrated sulphuric acid, a brown and eventually deep green colour is given. (3) A trace of bromine water added to the sulphuric acid solution gives a violet, slowly changing to a cherry-red and blood-red colour. None of these tests is satisfactory. The first may be confused with aconite, the second with veratrine, and the third with digitalis, and ptomaine bases and biliary pigments often confuse and mark these tests. A physiological test is therefore to be preferred.

* Report of Bureau of Animal Industry, 1898, p. 421.
† *Ibid.*, p. 479. ‡ *Vet. Jl.*, 1908, p. 502.

Ranunculus.

Description.—The species of this genus which are poisonous include—*Ranunculus sceleratus*, or celery-leaved crowfoot; *R. acris*, or upright meadow crowfoot; *R. bulbosus*, or common buttercup; *R. arvensis*, or corn crowfoot; *R. repens*, or creeping ranunculus; *R. Lingua*, or great spearwort; *R. Flammula*, or lesser spearwort; and *R. Ficaria*, or lesser celandine. These plants are well known, and detailed descriptions are superfluous, but attention may profitably be drawn to some of the particular characteristics of the several examples. The *general characters* are—annual or perennial, leaves divided or entire, flowers generally yellow, with double perianth, five sepals and five petals, numerous uniovular carpels.

Those having divided leaves—

R. acris (Fig. 20): Leaves hairy; calyx spreading, but not reflexed; stems erect, without runners; lower leaves palmately divided; carpels in a globular head. Flowers early summer till late autumn.

R. repens: Runners creeping and rooting.

R. arvensis: Leaves glabrous; segments narrow: carpels very prickly; plant erect. Abundant in slovenly farms in South of England. Flowers and ripens seed with the corn.

R. bulbosus: Calyx closely reflected on the peduncle; rootstock or thickened base of stem, forming a kind of bulb; carpels perfectly smooth. Flowers early summer.

R. sceleratus (Fig. 21): Petals very small; carpels small, numerous, in an ovate or oblong head.

Those having undivided leaves—

R. Lingua (Fig. 22): Flowers large, plants 3 feet high; and *R. Flammula*, about 1 foot high, flowers small, having leaves long and lanceolate, growing in marshes and wet places.

R. Ficaria (Fig. 23): Leaves cordate, smooth and shining. Flowers early spring.

All the above have yellow flowers.

Active Principle.—The *Ranunculi* all contain acrid juices whose chemical nature is not well understood. A volatile acid—ficaric acid—has been isolated, and also a substance, ficarin, probably a glucoside resembling saponin. Dragen-

Fig. 20.—Ranunculus Acris (Upright Meadow Crowfoot).

dorff further cites the existence of a substance named by him 'ranunculol.' *R. sceleratus* and *R. Ficaria* are also stated to contain anemonin (the active principle of anemone).

Symptoms.—The first symptoms induced by *ranunculus* are those of gastro-enteritis, colic, nausea, vomiting if

possible, salivation, emission of black fæces, and sometimes hæmaturia. To these are added nervous symptoms, retardation of pulse, slow and stertorous respiration, weakness of the posterior parts, difficulty in mastication and drinking, and blindness. With large quantities there may be con-

Fig. 21.—Ranunculus Sceleratus (Celery-leaved Crowfoot).

vulsions, with the eyes retracted in the orbits, an exaggeration or absolute arrest of defæcation, and death usually within twelve hours after the appearance of convulsions.

J. Gerrard * observed the effects of common buttercups on

* *Veterinarian*, 1874, p. 654.

the horse. The symptoms accorded with those above named. A change of diet and mild aperient effected a cure.

A case of the poisoning of sheep is described in the *Veterinarian* of 1844, p. 488. The cause was *Ranunculus*

Fig. 22.—Ranunculus Lingua (Great Spearwort).

repens. A few hours after the sheep had been in the field several suddenly fell, the eyes rolled, and some showed dizziness, and died with the head inclined over the left

flank. The shepherd bled them, which probably hastened the deaths of eleven.

Post-Mortem Appearances.—These consist of inflammatory lesions of the alimentary tract, particularly of the

Fig. 23.—Ranunculus Ficaria (Lesser Celandine).

intestines. The kidneys may also be inflamed. Valuable evidence may be forthcoming from the finding of fragments of the plant in the ingesta.

Treatment.—Mild purgatives, demulcents, and stimulants are indicated. Gerrard (*loc. cit.*) gave nitrous ether, aromatic ammonia, extract of hyoscyamus, peppermint water, tincture of opium, and a 4-drachm ball in the case of a horse. There was purgation and gradual recovery.

Chemical Diagnosis.—In the absence of precise knowledge chemical tests for ranunculus poison are impossible—indeed are non-existent. The exact study of the physiological and chemical properties of this, as of other indefinite 'acrid juices,' is greatly needed.

Other Genera of the Ranunculaceæ.

Clematis.—The *Clematis Vitalba*, traveller's joy or old man's beard, is a well-known climbing plant, the white flowers of which are common on hedge-rows in June and July. All parts of the mature plant are dangerous, but cases of poisoning are rare. It contains as active principle *clematin*, resembling anemonin, and acting as an irritant purgative and diuretic, causing enteritis, and, in quantity, fatal dysentery. Externally it is a powerful irritant and vesicant.

Thalictrum.—The exotic *Thalictrum macrocarpum* has been shown to contain in the root an alkaloid *thalictrine*, whose effects are like those of aconitine. In Great Britain the species *T. flavum*, yellow thalictrum or meadow rue, is sometimes found in moist places and along ditches. It is not common, nor does it appear to be so dangerous as the cultivated *T. macrocarpum*. In poisoning the general character of the symptoms would probably recall those of aconite, but no cases are recorded.

Anemone.—The anemones have a perennial rootstock, leaves radical, flower-stem naked, excepting an involucre of three leaves, usually at a considerable distance from the flowers. Sepals five or more, frequently six, coloured and petal-like, longer than the stamens. No petals; stamens numerous; carpels numerous, one-seeded, pointed, or ending in a long feathery awn.

A. Pulsatilla has purple flowers, silky outside, and carpels ending in feathery awns.

A. nemorosa has white or pink glabrous flowers, and carpels ending in a point.

The anemones contain oil of anemone and anemonin, volatile with steam, and extractible from the distillate by chloroform. The effect resembles that of cantharides, and anemonin is chemically allied to cantharidin. As with cantharides, there is local irritant and blistering action, and internally the anemones give rise to gastro-enteritis, convulsions, and paralysis.

Accidents sometimes occur to animals owing to the prevalence of the plant in woods in spring-time, when fresh green food is most greedily eaten.

If an alcoholic solution of anemonin is treated with a little sodium nitro-prusside, and then with caustic soda, an intense blood-red colour is produced. This test may be applied to the material separated by steam distillation, and extracted from the distillate by chloroform, after having allowed the chloroform to evaporate at a low temperature.

Adonis.—The *Adonis autumnalis*, or pheasant's eye, having five to eight bright scarlet petals, with a dark spot at the base, is rarely found in the warmer counties of England and Ireland. *A. vernalis*, or ox-eye, has a yellow flower, and is cultivated in Britain.

These plants contain a glucoside adonidin, which is credited with abortive properties. In large doses death is caused by superpurgation and cardiac disturbance, but cases are not common.

Caltha.—*Caltha palustris*, marsh marigold or king-cup, with bright yellow flowers, is abundant in marshy places and on brook-sides. It flowers early in the spring. The nature and symptoms of poisoning by it are like those of Ranunculus.

Aquilegia is represented by the species *Aquilegia vulgaris*, or columbine, which grows wild in chalky woods and pastures. According to Cornevin, poisoning by it resembles that of aconite as regards symptoms and lesions.

Actæa.—This genus is represented by the rare *Actæa spicata*, baneberry, or herb Christopher, very local in Britain, and only found in the northern counties. It is found in mountain woods, and has white flowers and black berries. The active principle is apparently an essential oil. When eaten in sufficient quantities, the plant causes violent gastro-enteritis, purgation, and vomition, followed by drunkenness and delirium. The nature of the toxic substance and the effects merit further study.

In America there occur *A. alba*, white baneberry, and *A. rubra*, red baneberry, but animals refuse the plants, so poisoning is unlikely.

PAPAVERACEÆ.

Poisonous members of this order belong to the genera *Papaver*, *Rœmeria*, *Chelidonium*, and *Glaucium*.

Papaver.

The opium poppy, *Papaver somniferum*, occasionally assumes the wild state in England in cornfields and in the fens. Poisoning by it is most unlikely, and, should it occur, will recall that of opium or morphine (*q.v.*).

The common cornfield red poppy, *P. Rhœas*, is so well known as not to require description in this place.

Active Principle.—The red leaves are sometimes used to make coloured syrup for medicines (syrupus rhœados), and are harmless. The plant does not contain the opium alkaloids, but yields a sparingly soluble alkaloid, *rhœadine*, decomposed by warm diluted acids with formation of a blood-red colour.

Symptoms.—Fatal poisoning by the common poppy is rare, but its possibility ought to be kept in mind in those conditions where animals might get it along with fodder on account of its relative abundance.

The plant causes in the ox arrest of digestion, following a period of excitement. Immobility, coma, low tempera-

ture, slowed respiration, convulsive movements, and death in asphyxia, are to be anticipated. The *lesions* are as in opium poisoning, with more pronounced alimentary disorder.

P. dubium, the long-headed poppy, is distinguished from *P. Rhœas* chiefly by the capsule, which is oblong, about twice as long as broad, and narrow at the base, whilst that of common poppy is globular or slightly top-shaped.*

It is less common than *P. Rhœas* in England and Ireland, but more frequent in Scotland. Poisoning by it is even less likely than with *P. Rhœas*.

The exotic species, *Rœmeria hybrida*, violet-horned poppy, of the genus *rœmeria*, having purple flowers, red at the base, has established itself very locally in cornfields in the eastern counties. It also contains rhœadine.

Chelidonium.

Botanical Description.—*Chelidonium majus* (Fig. 24), the greater celandine. Rootstock perennial. Stems erect, slender, branching, 1 or 2 feet high, full of a yellow fœtid juice, and generally bearing a few spreading leaves. Leaves thin, glaucous underneath, once or twice pinnate, the segments ovate, coarsely toothed or lobed, the stalks often dilated into a kind of false stipules. Flowers small and yellow, three or six together, in a loose umbel, on a long peduncle. Pod nearly cylindrical, glabrous, 1½ inches long.

Common on roadsides, near houses, more in England and Ireland than in Scotland.

Active Principles.—Celandine contains two chief alkaloids, *chelidonine* and *sanguinarine*, produced probably chiefly on fruition.† Chelidonine resembles morphine, acting on the central nervous system with less stimulant effect, and sanguinarine promotes peristalsis and salivation, and causes tetanus and excitement.‡

* Bentham and Hooker.
† H. Caulton Reeks, *Jl. Comp. Path.*, 1902.
‡ Cushny, *Pharmacology*, 1905, p. 229.

Symptoms.—The juice of the plant acts as an irritant, and internally causes nausea, vomiting, and violent purgation.

Caulton Reeks (*loc. cit.*) describes a most interesting case

FIG. 24.—CHELIDONIUM MAJUS (GREATER CELANDINE).

of the poisoning of cows by celandine, in which the effects due to chelidonine predominate. He observed drowsiness, salivation, thirst, uncertain gait, torpid bowels, kidneys active. On attempting to touch the cow she fell in convulsions, limbs stretched out and quivering, moaning, and

beating about with head, eyes retracted in orbits. Death occurred within two hours.

Some of the animals recovered under oleaginous purgatives, but one which had not responded, showed drowsiness, was given castor oil, and displayed symptoms of gastro-enteritis and kidney irritation. Offensive purgation set in, with death from exhaustion after a few days.

Chemical Diagnosis.—The reactions of the alkaloids of celandine are not very characteristic. Chelidonine is coloured greenish-yellow, brown, cherry-red, and finally violet by concentrated sulphuric acid. Sanguinarine, which is only present in traces, is characterised by giving blood-red-coloured salts.

Glaucium.

Glaucium luteum, the well-known horned or sea poppy, with large yellow flowers, and pods from 10 to 12 inches long, is alleged to cause poisoning similar to that of celandine; but cases are not to hand, and the point needs revision. It is a very abundant maritime plant in the South, but is rarer in Scotland. Animals do not eat it, but there is a chance of its accidental inclusion in forage.

CRUCIFERÆ.

The genera of this order found wild or cultivated in the temperate parts of the Old World, and credited with poisonous or objectionable properties, are *Brassica*, *Cochlearia*, *Raphanus*, and *Sisymbrium*.

Active Principles.—If to them we add onion, belonging to the *Liliaceæ*, all the plants implicated owe their activity to volatile or essential oils of the type of mustard oil (allyl sulphocyanide). In the black mustard seed is a glucoside, myronic acid, which is resolved by ferments or acids into glucose, potassium acid sulphate, and mustard oil, or allyl sulphocyanide. White mustard contains sinalbin and yields oxybenzyl sulphocyanide.

Botanical Characters.—The plants likely to cause harm

are all well known, and it will therefore suffice to name those which are stated to have caused poisoning. They are—

Brassica alba (*Sinapis alba* Linn.), cultivated, or white, mustard; *Brassica Sinapis* (*Sinapis arvensis* Linn.), charlock or wild mustard; *Brassica nigra* (*Sinapis nigra* Linn.), or black mustard; *Cochlearia Armoracia*, or horse-radish; and onion (*Allium sativum*).

Wild radish (*Raphanus Raphanistrum*), according to Cornevin, probably contains oils like mustard, whilst *Sisymbrium alliaria* taints milk like onion.

Symptoms.—In the horse black mustard produces bronchial symptoms, marked by difficulty in breathing and the discharge of great quantities of yellowish frothy matter from the nose, the post-mortem showing pulmonary congestion and bronchi injected, dark red, and full of frothy yellow liquid.

Black mustard seeds* are sometimes found in cake, such as that of rape seed, and J. Gerrard[7] records the effects of such a cake on cattle. The uneasiness, restlessness, and intense colic, with frantic rushing about and mania, ending in exhaustion, falling, struggles and collapse, were prominent features, and Gerrard gave the case as a good example of the action of a purely irritant poison.

J. W. Anderton[8] similarly records a case in which cows were made fatally ill after about 1 pound each of an oil-cake, shown by Tuson to be composed chiefly of mustard seed. Salivation, colic, respiratory distress, and accelerated pulse were noticed.

The more recent case recorded by F. J. Roub,[6] in which cattle ate mustard on pasture, appears very different, for he noted dulness, coldness, some tympany, laboured respiration, staggering, and falling. In fatal cases there was immobility and a semi-comatose condition.

Cozetta[5] records cattle poisoning by a colza cake of Indian origin containing various species of mustard seed,

* Whole seeds pass through unaltered in the fæces. They may be eaten by the pound without any effect. In cake, however, they may be in the crushed condition.

and remarks that steeping in boiling water withdraws the essential oil, and renders the cake harmless.

Cochlearia armorica, or horse-radish, has been held responsible for poisoning in England,[2,3,4] but is not reckoned amongst the poisonous plants by the Continental authorities. Since it also yields allyl sulphocyanide, there seems little doubt that it might equally with mustard prove dangerous.

W. E. Litt[4] observed in cattle: wildness, lowing, excitement, and rushing about, recalling the symptoms noted by the earlier observers on mustard. Thereafter, in conformity with Roub's observations, he noted collapse and coldness, with low pulse and staring eyes.

It appears most likely that the nervous symptoms here, as in so many cases, are due to exhaustion, and not to any specific action of the active principles.

Comparable with the poisoning by mustard is that observed by Goldsmith,[1] in which onion was the cause. He remarked that cows, after eating freely of onions in a cart, some of which were sprouting, and others decayed, displayed severe colic. Some were constipated, others slightly purged, and in one case there was vomition. In the worst case there was severe constipation, staggering, tenderness in the loins, temperature 103° F., and the dark-coloured urine smelt of onions.

Post-Mortem Appearances.—The prominent lesions in these cases were inflammation of the œsophagus and trachea, and less characteristic inflammation of the rumen, and patchy inflammation of the intestines. Roub (*loc. cit.*) found the abdominal cavity and bladder to contain abundant yellow fluid, smelling of mustard. In the onion case the viscera all smelt strongly of onions.

Treatment.—Roub gave 1½ pounds Glauber's salt, followed by 1 pound every twelve hours till purgation resulted, and as stimulants nux vomica and spirits of nitre. Change of diet, oleaginous purgatives, and stimulants are indicated.

Chemical Diagnosis. — Mustard oil is volatile from neutral or acid media in a current of steam. It is thus

separated in the search for volatile poisons, and its characteristic odour suffices for its recognition. When in sufficient quantity it may be got pure and definitely identified by its physical properties.

REFERENCES TO CRUCIFERÆ AND ONION.

[1] W. W. Goldsmith, *Jl. Comp. Path.*, 1909, p. 151.
[2] C. E. Dayus, *Vet. Record*, 1906, p. 155.
[3] D. Fairbank, *Vet. Record*, 1906, p. 117.
[4] W. E. Litt, *Vet. Record*, 1894, p. 546.
[5] Cozetta, *Vet. Jl.*, 1905, p. 95.
[6] F. J. Roub, *Vet. Jl.*, 1902, p. 166.
[7] J. Gerrard, *Veterinarian*, 1875, p. 396.
[8] J. W. Anderton, *Veterinarian*, 1861, p. 265.

VIOLACEÆ.

The members of this family, well known in *Viola odorata*, the common violet, contain the active principle *iridin* of the *iridaceæ* (*q.v.*). The poison is contained in the roots, and when these are eaten symptoms of nausea, vomiting, nervous, respiratory, and cardiac disturbances are set up. Poisoning is, however, very rare, and the possibility of its occurrence alone warrants its inclusion here.

CARYOPHYLLACEÆ.

This family includes the genera *Saponaria*, *Lychnis*, *Arenaria*, and *Stellaria*, members of which are poisonous.

Botanical Characters.—Saponaria is represented by *S. officinalis*, soap-wort, hedge-pink, or crow-soap. It frequents hedge-banks and waste places, attains a height of about 2 feet, and is a perennial. The leaves are ovate, lanceolate, and opposite ; calyx cylindrical, petals pink, and flowers in August.

Lychnis is represented by *L. Githago* Scop. (Fig. 25) (*Agrostemma Githago* Linn.), the corn-cockle, in cornfields. It attains 2 to 3 feet, is covered with silky hairs, and bears purple flowers in July. The seeds are small, dark coloured, and wrinkled, and number thirty to forty in a capsule.

Occasionally the grain gets mixed with cereals, and thus enters flour or forage. The starch is small, having the dimension 1 to 2 μ, as compared with wheat, which has 15 to 35 μ.

FIG. 25.—LYCHNIS GITHAGO (CORN-COCKLE).

Arenaria includes *A. serpyllifolia*, or the thyme-leaved sandwort, and is common on walls, dry sands, and waste places. 'A very much branched, slender, and slightly downy annual, seldom attaining 6 inches. Leaves very

small, ovate, and pointed. Pedicles from the upper axils or forks of the stem, 2 or 3 lines long, and slender. Sepals pointed, about 1½ lines long. Petals usually much shorter, but variable in size, obovate. Capsule opening in six narrow valves.'*

Stellaria.—This genus (the star-worts), represented by several British species, was noted by Cobbold† as being responsible for the poisoning of horses in Russia, and as having occasioned losses in the Crimea campaign.

Active Principle.—All these plants owe their poisonous properties to glucosides of the saponin type, which are widely diffused in the plant kingdom, and which present minor differences to one another. The officinal *Quillaja Saponaria* (Rosaceæ) is a chief source of saponin. It is native to South America, and is known as Chili soap bark, or Panama root. The saponins possess certain interesting properties which throw light on their physiological effects. They do not form true solutions in water, but give colloid suspensions, and impart remarkable and permanent frothing qualities to the liquid. For this reason saponin is used as an adulterant to such beverages as lemonade. The saponins are not diffusible, being colloidal, and are therefore not easily absorbed. When taken by the mouth it is probable that absorption only occurs when inflammatory lesions are also caused, as happens with some of the plants. When introduced into the blood stream the saponins cause hæmolysis, or dissolution of the red cells, and also similarly act on other cells—*e.g.*, of ganglia. To these effects are due the nervous symptoms of stupefaction and paralysis. Saponin is taken up from water by the gills of fishes into the blood stream, and thus produces poisoning, even in a dilution of 1 to 200,000. The hæmolytic action is stopped by cholesterol, which thus directly neutralises or antagonises saponin poisoning.

It has been stated that animals develop tolerance (or immunity?) after continual feeding on small doses of corn-cockle meal.

* Bentham and Hooker. † *Veterinarian*, 1880, p. 453.

Symptoms.—Animals refuse to eat the corn-cockle plant, and generally poisoning is the result of the ignorant feeding of the grains, or fraudulent admixture of the flour with meal. Cornevin gives the approximate lethal doses of the flour as 18 grains and 7 grains per pound body weight for the ox and pig respectively, about twice that proportion of the whole grain being needed. Pigs reject large doses by vomiting, but small doses repeated over a long interval give rise to chronic poisoning.

In the acute poisoning of *horses* there is copious salivation, colic, pallor of the mucous membranes, pulse small and rapid, elevated temperature, and accelerated respiration. The fæces are diarrhœic and fœtid. Muscular tremors and rigidity set in, followed by collapse, coma, and death, without convulsions.

Cattle display similar gastric disturbances. A period of coma is also reached, during which there is continual passage of diarrhœa, gradual loss of motor and sensory powers, and death.

According to Cornevin, *Arenaria* does not poison, but causes great salivation.

Post-Mortem Appearances.—After subcutaneous injection of saponin, there is no gastro-enteritis, although the alimentary canal is emptied by reason of the purgation and vomiting. But after the poison has been taken by the mouth, there is inflammation, sometimes extending throughout the alimentary tract. The contents are always fœtid, and mixed with bloody mucus. There is congestion of the cerebral meninges and lungs, the kidneys may be a little inflamed, and the liver normal.

Treatment.—The treatment of poisoning should consist of removal of the cause, opiates against pain, and stimulants as indicated.

Chemical Diagnosis.—There is little prospect of a satisfactory recovery of saponin from ingesta or organs after poisoning. Search should be directed to the discovery of the starch grains of corn-cockle in the food. The starch of corn-cockle requires more iodine to induce the blue starch

iodine coloration than wheat, or other cereal, or potato starches. Roughly, about seven to ten times as much iodine is required (Tabourin). A meal of corn-cockle flour (like that of lolium) gives a full orange-yellow solution when 10 grammes are warmed with 30 to 40 c.c. of 70 per cent. alcohol, with 5 per cent. of dilute hydrochloric acid (Vogl's test). Under the same conditions various other flours give to the acid alcohol the following tints: fine wheat and rye, colourless; coarse wheat and rye, pale yellow; barley and oats, straw yellow; pea meal, full yellow; vetch and bean meal, purple-red; ergot, blood-red; meals containing rhinanthin, green. Rhinanthin is a glucoside contained in certain of the *Scrophulariaceæ—e.g.*, *Melampyrum*, *Rhinanthus*, etc. (*q.v.*).

HYPERICACEÆ.

Botanical Characters.—The only member of this family reputed to be dangerous is *Hypericum perforatum*, or St. John's-wort, which is abundant in woods, thickets, and hedges, and found in Europe and America. It reaches about 1 to 1½ feet, and bears a yellow flower. As cases of poisoning by it are rare, a detailed description is unnecessary.

Active Principle.—The active principle of the St. John's-wort has not been accurately studied. It appears to contain a resin and a volatile oil. The resin is probably allied to the drastic gamboge, obtained from species of the related order, *Guttiferæ*.

Effects.—The plant is not taken by animals save when mixed with such forage as lucerne. Cornevin relates that a mare under the influence of the plant became semi-comatose, the head drooping between the outstretched forelegs. The pulse was full and slow, respiration deep and slow, and the appetite lost. The pupils were dilated, conjunctivæ injected, and the unpigmented skin of the nose coloured wine red, as in purpura. In about twelve hours the condition passed off.

MELIACEÆ.

This order is tropical, and comprises *Melia Azedarach*, or the Chinese umbrella-tree, grown in Central Europe and the United States, and escaped from cultivation in the South. Hogs are stated to have been poisoned in Arizona by ignorant feeding of the seeds. T. J. Symonds* refers to *Azadirachta Indica* as a drastic purgative, the juice of the leaves being used as an anthelmintic, emmenagogue, and diuretic.

Poisoning, which specially affects *pigs*, is marked by nausea, vomition, violent colic, and tympanites, followed by diarrhœa, sweating, convulsions, uncertain gait, and intense thirst. The *lesions* are those of intestinal inflammation.

CELASTRACEÆ.

Botanical Characters.—This order is represented in Britain by the *Euonymus europæus*, spindle-tree or skewer-wood (Fig. 26). A glabrous shrub, about 3 to 5 feet high. Leaves shortly stalked, ovate-oblong, or lanceolate, pointed, and minutely toothed. Peduncles shorter than the leaves, with seldom more than three or five flowers, of a yellowish-green colour. Petals four, obovate, about 2 lines long, the stamens half that length. Fruit quadrangular, red when ripe, opening at the angles so as to show the seeds enclosed in a brilliant orange-coloured aril. The shrub is frequent in parts of England in hedges. Animals eat the young leaves in early summer, when the poisonous effects are greater than in the autumn.

Active Principle.—The *euonymus* contains a glucoside called euonymin, belonging to the group of purgatives.

Effects.—The effects, lesions, and treatment of poisoning by euonymus are like those of other vegetable purgatives, and need not therefore be again detailed, especially as recorded cases are very few.

* *Quarterly Jl. of Veterinary Science in India*, 1886, p. 77.

FIG. 26.—EUONYMUS EUROPÆUS (SPINDLE-TREE).

The leaves of the American *Celastrus scandens*, climbing bitter-sweet or staff-vine, are reported to have seriously, though not fatally, poisoned a horse.

RHAMNACEÆ.

Botanical Characters.—Two species of the Rhamnus, or buckthorn, genus are poisonous—viz., *Rhamnus catharticus*, the common buckthorn, and *R. Frangula*, the alder buckthorn. Neither species is very common in Britain, the former less so than the latter, and both grow in hedges or bushy places. The flowers are small and green, and the fruit about the size of a pea, that of *R. catharticus* being black, and that of *R. Frangula* dark purple. *R. cartharticus* usually grows on calcareous, and *R. Frangula* on peaty or leafy, soil.

Active Principle.—The fruit contains a glucoside or glucosides, cathartin and frangulin of the anthraquinone group of vegetable purgatives, which embraces the aloes. The bark of *Rhamnus Purshianus* (America) yields cascara sagrada.

Effects.—The berries are purgatives, and dangerous effects, which are rarely likely, take the form of super-purgation, and need no elaborate detail in this connection.

Cornevin states, on the authority of an Italian physician (Prota-Giurlœo), that the leaves of *R. alaternus* (not found in Britain), and also those of *Ligustrum vulgare*, or privet (*q.v.*), arrest lactation in the female, and may find useful application on account of this property.

LEGUMINOSÆ.

This family comprises many important poisonous species, few of which are native to Britain, though several are cultivated. Amongst British wild plants are found *Cytisus Scoparius*, the common and very abundant broom, which resembles the cultivated *Spartium junceum*, or Spanish broom, in containing the alkaloid sparteine, and *Lathyrus aphaca*, the yellow vetchling, which contains a cyanogenetic glucoside like the exotic *Phaseolus lunatus*, or Java bean (see under Cyanides).

Of exotic species cultivated in Britain, mention must be made of *Cytisus Laburnum*, the laburnum-tree; varieties of *Lupines* grown as ornamental plants and also sometimes for forage; and *Trifolium hybridum*, or alsike clover, grown for forage. Definite cases of poisoning by these are well established. The cultivated *Coronilla* and *Wistaria*, are reputed to be dangerous, but are not important.

Exotic species, the seeds of which have been used as forage, and which are definitely and seriously poisonous, are *Lathyrus sativus*, the Indian pea, and *Phaseolus lunatus* (see under Cyanides).

Laburnum.

Cases of poisoning by *laburnum* are not numerous, nor are the records of our literature satisfactory. Cornevin, however, has submitted the question to experimental test and established the toxicity of the plant beyond doubt. Moreover, poisoning in the human subject, usually by eating the flowers, is well recognised.

Active Principle.—The active principle, possibly not the only agent, is the alkaloid *cytisine*. All parts of the plant are poisonous, the greater part of the poison being, however, found in the seeds. Desiccation does not destroy the activity, and the poison is not entirely removed from the seeds by boiling.

Effects.—Cornevin found it impossible to kill the dog or cat by feeding (on account of vomition); or the sheep, goat, or ox by reason of refusal, after a certain point, of the food. The horse and ass could be killed by feeding, and all animals by injection of the poison. Taylor records, similarly, failure to poison the dog and cat.

Vomition, excitement, followed by clonic contractions and coma, appear to be produced in carnivores.

In the *horse* Cornevin observed general and sexual excitement, muscular tremors, followed by contractions, beginning in the posterior parts. In general the poisoning is in three stages—(1) excitement; (2) coma and inco-ordination of movements; (3) convulsions.

The *lesions* are not characteristic.

Chemical Diagnosis.—The alkaloid cytisine is separated in the routine method of search for alkaloids. It is not very easily recognised, since confusion with other alkaloids may arise. It gives a yellow, brown, and finally green colour on solution in strong sulphuric acid, with nitric or chromic acids. Van der Moer's reaction is characteristic, but difficult to apply, since the substances ought to be in fairly definite proportions (one molecule of each), and free acid absent. It consists in the production by ferric chloride of a blood-red colour, destroyed by adding hydrogen peroxide, and then passing on warming to blue.

Brooms.

Spartium junceum, Spanish broom, and *Cytisus Scoparius*, common broom, merit mention as a precaution, for they contain the volatile alkaloid *sparteine*, allied chemically to conine, and showing almost identical physiological effects, causing central nervous paralysis. The therapeutic dose of sparteine sulphate for the horse is 15 to 75 grains (Kaufmann), and the plant yields about 0·3 per cent. of active principle, whence it appears that from about 25 pounds of the plant about 1 ounce would result, and this would probably be an average poisonous dose (ten times the average therapeutic). Poisoning by spartium is therefore only likely in exceptional cases.

Lupines.

Of the lupines, only *Lupinus luteus*, or the yellow lupine, appears to be very dangerous, and has been responsible for poisoning of sheep on the Continent, especially in Germany, but in Montana *L. leucophyllus* has caused the deaths of many sheep. Other animals are susceptible, but do not receive so much ordinarily as do sheep. According to the German authorities, a daily ration of 1 pound of the whole plant, $\frac{3}{5}$ pound of empty pods, or

$\frac{1}{3}$ pound of seeds, will produce poisoning. The poisoning in Germany was most common about 1880, when nearly 6 per cent. of the sheep in Pomerania died of lupinosis.

Symptoms.—Acute lupinosis is marked by inappetence, dyspnœa, and fever, the temperature rising by 1° to 2°, hæmaturia, circulatory, and digestive derangements; trembling, spasms, and vertigo. Jaundice is characteristic. In the chronic form the interstitial hepatitis predominates.

Post-Mortem Appearances. — The liver shows fatty degeneration, kidneys inflamed and urine icteric, spleen soft and tumefied. The first stomachs are inflamed, and there is effusion of blood in the intestine, peritoneum, and on the skin. Œdema of the lungs, larynx, and pia mater is constant.

Treatment.—The treatment of lupinosis consists in the exhibition of acidified water to hinder absorption, owing to the insolubility of lupinotoxine in acids. Change of diet is necessary, and removal of the cause by oily purgatives.

Active Principle.—An active principle is extracted by means of 2·5 per cent. soda from lupines, and has been named *lupinotoxine* (Arnold), or *icterogene* (Kuhne), from the jaundice characteristic of lupinosis. It is possible that the poison is formed by the agency of moulds after storage, as lupine is not constant in its actions, but the point cannot be regarded as settled. Little is known of the chemistry of icterogene, which is not soluble in water or acids, withstands three hours dry heating of 190° F., but loses its toxicity on moist heating at 2·5 atmospheres pressure. Detection of lupine poisoning by analysis is therefore impossible, but a diagnosis is certain if jaundice and the general symptoms follow the use of the food.

Trifolium hybridum.

The well-known "alsike clover" differs from white or Dutch clover (*Trifolium repens*) in having a pale pink flower, and stem erect and branched, without roots at

the nodes, the stem of *T. repens* being creeping and rooting at the nodes.*

There seems to be an agreement of opinion that prolonged feeding of sheep and other animals on alsike clover may prove harmful, and it is remarked that the plant is not eaten readily, particularly by the horse (Cornevin).

Symptoms.—In the *horse* the same authority notes, as the results of the derangement, excessive salivation and stomatitis; and as general symptoms sweating, convulsive masticatory movements, and œdema of the upper lip. At the same time there is chronic intestinal irritation and colic. In severe cases the conjunctival and buccal membranes are infiltrated, and display a marked yellow tint.

W. M. Scott† describes the poisoning of tup *lambs* by alsike clover on the point of seeding. Ten out of fifty were very ill, staggering and gritting teeth continuously. Some of the worst emitted short, sharp, painful grunts, and two died.

Treatment.—This consisted, in Scott's cases, of removal to bare pasture land, and exhibition of ether, morphine hydrochloride, hydrocyanic acid, chloroform, and sodium bicarbonate, with 2 tablespoonfuls of linseed oil, and ½ pint of linseed tea—night and morning.

Post-Mortem Appearances.—The rumen full, reticulum empty, abomasum containing watery herbage, membranes much inflamed. The intestines alternately contracted and dilated; the contracted parts pale and bloodless, the dilated parts red or purple. Yellow seeds were found in the ingesta. Lungs and right kidney slightly congested. Bronchial tubes and trachea contained slightly sanguinaceous froth.

The cause of poisoning by this plant is unknown.

Lathyrism.

Several varieties of the genus *Lathyrus* (such as dog-tooth, Indian, or mutter, pea) have proved poisonous. *L. sativus*, *L. cicera*, and *L. clymenum* are typical examples.

* Bentham and Hooker. † *Vet. Record*, 1900, p. 101.

The Indian pea was first brought into Europe with cereals of Eastern origin; but peas of these varieties have long been known to be poisonous, having been mentioned by Hippocrates and Pliny.

Cases of poisoning amongst horses appear to have been first noticed in England in the eighties.

In the earlier stages of growth the plant is harmless, and may be used as forage, but from the time of the formation of seed and onward it becomes toxic, the seeds being the most dangerous part. The poison is not lost by drying, nor by boiling for a short time, though possibly prolonged boiling might destroy it. When boiled the poison seems to pass into the water. Cornevin extracted 5 litres of peas, concentrated the water, and injected the extract into a dog of average weight, which died in twenty-four hours.

Pathological changes are more speedily produced when meal rather than the whole pea is fed.

Symptoms.—The effects of Lathyrus are very characteristic, and the condition known as 'lathyrism' has been observed in man and all the domesticated animals. Lathyrism is only produced when considerable proportions of the pea enter into the rations, and over a prolonged period of time; in man generally in the fourth month; in the horse fed exclusively on the pea, the tenth day; but when 1 or 2 quarts are given daily only towards about the eightieth day. Moreover, the malady may declare itself so long as fifty days after the cessation of the pea feeding.

In man a constant sign is paralysis of the lower extremities; speech, intelligence, and power over the upper extremities are preserved. The symptoms resemble those of dorsal tabes, and lathyrism has further been compared with beri-beri.

The first complete observations on the *horse* were made in this country by McCall in 1886.[7] An animal was feeding well, but thick in his wind. After going about 200 yards with an empty lorry it stood with the forelegs forward, neck stretched, elbows out, and laboured breathing.

It was kept on its feet, until unyoked, with difficulty; each breath gave a loud sound from the larynx. There was profuse sweating; quick, irregular, and intermittent pulse; increased impulse of the heart; venous pulsations; normal temperature; and a vibration over the region of the larynx. After about five minutes the symptoms disappeared, the animal seemed well, and began to eat hay.

The horses had been having a mixture containing 20 per cent. of Indian pea meal. Some had it boiled, others wetted, and McCall noticed that only those which had had unboiled meal were affected.

McCall experimentally fed an old but sound cart mare, whose pulse rose in eleven days from 52 to 84, temperature normal; but after exercise the pulse was 130, and there was vibration over the larynx. Later vesicles and inflamed spots formed in the mouth, and there was extensive loss of hair, not caused by parasites.

G. E. King[6] observes similar symptoms with gritting of teeth, frothing, and convulsive movements of the eyes, recalling epilepsy, in cart horses.

J. P. Slidders[5] and Joseph Abson[4] record the effects of the dog-tooth pea on horses and ponies. The dog-tooth pea is much larger and whiter than the mutter pea. The symptoms observed were thick wind, a staggering gait, weakness of hind quarters, and general signs of intoxication were noticed, and sudden violent attacks of laryngeal paralysis and dyspnœa, during which there was palpitation, frothing, tongue protruded, eyes staring, bluish tint of buccal membranes, and palpitations. The paroxysms sometimes proved fatal, otherwise there would be a speedy temporary recovery. Change of diet is ineffectual in arresting the malady, at any rate at first.

F. Meachem[3] noticed no ill-effect in horses until after twelve months' feeding.

Cases of disease in *cattle* have been observed with *L. clymenum*[2] and with the Indian vetch,[1] in both cases in France. Cows had green vetches containing *L. clymenum* for ten days. Three weeks later they were ill; they lay on

sternum with neck muscles on non-affected side contracted, the mouth closed, thick viscid saliva, suspended rumination, constipation, paralysis of the limbs, loss of sensibility in the skin, pulse small and weak.

In the Indian vetch case the cause was a cake containing *lathyrus*, and there was stiffness of the lower joints, staggering, blindness, and other symptoms, as in the preceding case.

With *sheep* and *pigs* paralysis of the hind extremities is notable.

Cornevin's researches on the *dog*, after hypodermic injection, show trembling and spasmodic movements, beginning in the hind extremities, and propagated eventually to the muscles of the neck and front legs; later nausea and vomition, profound depression, and loss of power of the hind limbs; eventually complete motor paralysis and death without convulsions from the twenty-fourth to fortieth hour.

Post-Mortem Appearances.—In chronic lathyrism of the horse there are thickened congested patches in the stomach and intestines; lungs engorged, and bronchi showing signs of catarrh and congestion. The larynx shows irregular congested patches, especially round the glottis. The intrinsic muscles are paler and smaller on the left than on the right, and show fatty degeneration. The bronchi, trachea, and nasal passages are filled with bloody spume and froth.

In the case of cattle[2] the blood was thick and dark, the cranium and anterior portions of the spinal canal containing a large amount of bloody serum, the meninges, deeply congested, forming a well-marked network over both hemispheres, with black hæmorrhagic patches, extending a considerable depth into the tissue.

The only effective *treatment* beyond removal of the cause, and rest, would appear to be tracheotomy.

The chemical study of *lathyrus* has not succeeded in associating its effects with any definite simple active principle. Evidence as to the destruction or otherwise of the poison by boiling is conflicting, but the point is of considerable importance, for it is not impossible that further research

may show that we have here to deal with toxine poisoning like that of the castor bean. Or it may possibly be found that lathyrus lacks certain essential components, present, perhaps, only in traces, but whose absence leads to disease, as has been shown to be the case with polished rice in giving rise to beri-beri.

REFERENCES TO LATHYRISM.

[1] Abstract, *Vet. Record*, 1901, p. 323.
[2] Lucet, *Vet. Record*, 1898, p. 249.
[3] F. Meachem, *Vet. Jl.*, 1896, p. 77.
[4] J. Abson, *Vet. Record*, 1894, p. 159.
[5] J. P. Slidders, *Vet. Record*, 1894, p. 90.
[6] G. E. King, *Jl. Comp. Path.*, 1892, p. 371.
[7] McCall, *Veterinarian*, 1886, p. 789.

Locoism.

The disease of sheep and horses, well known in America as *loco disease*, or *locoism* (Spanish 'loco,' mad), is very common, and causes great loss, particularly in Colorado and Montana. The ætiology is obscure, but no doubt remains that the disease is caused by leguminous plants, mostly of the *Astragalus* species. The chief loco-weeds incriminated are *Astragalus mollissimus*, woolly loco-weed, especially abundant in Colorado; *Aragallus spicatus*, white loco-weed (Montana); *Aragallus lambertii*, stemless loco-weed, in the Western States; and *Astragalus splendens*, *lagopus*, and *besseyi*. The disease has not been associated with any chemical component of the plants implicated.

Symptoms.—Locoism assumes the *acute* and *chronic* forms. The acute disease in *sheep* is marked by the animal becoming unmanageable, completely blind, and dizzy, walking in long circles to the right, and then standing in a stupor for a few moments. At the beginning of an attack the head is elevated and drawn to the right. The attacks become more frequent as the malady progresses. The pupil is not dilated, and the expression and pulse are nearly normal. Trembling fits are characteristic. Locoed

animals are hard to manage, tending to bolt in an erratic fashion.

In *chronic locoism* there is progressive emaciation and craziness. The animal is unable to take care of itself, and may fall into the water when drinking. Horses may remain standing, unable to walk, in the same place and without water for as long as two weeks. Sheep shed the fleece in patches or as a whole. Trembling fits are frequent, and death is from exhaustion and mal-nutrition. Isolation of locoed sheep is necessary, for the 'loco habit' may be picked up by imitation.

The *lesions* are not characteristic, sheep and horses always showing slight cerebral congestion.

The only effective *treatment* appears to be segregation, confinement, and careful feeding. But on liberation the habit declares itself, and locoed horses after an apparent recovery are always dangerous.

Other Leguminosæ.

The European *Ervum Ervilia*, or bastard lentil, produces in pigs symptoms of somnolence, passing into coma, interrupted by muscular tremors, and occasionally with nausea and vomition. Sheep and cattle appear to be tolerant, and the pig also to acquire tolerance (Cornevin). It seems possible that mishaps are due rather to mal-nutrition than to a definite toxic substance.

Numerous other species of this order are held responsible for poisonings, of which mention may be made of the following:

Erythrophleum guineense and *E. Couminga* have caused poisoning in Guinea and the Seychelles. *E. guineense*, and probably also other species, contains a glucoside, *erythrophlein*, of the digitalis class.

The North American *Gymnocladus dioïca*, or coffee-tree, is stated to contain a saponin, but cases of animal poisoning are not recorded.

In South Africa *Crotalaria Burkeana*, or 'stijfziekte

bosje,' according to Burtt-Davy, causes paralysis or stiffening of the limbs of cattle, due to laminitis. After about five days the animal becomes very stiff in the joints, and frequently unable to stand. The hoofs may grow until they break off. According to Theiler, the plant is only dangerous when fresh. Related to this is the *Crotalaria sagittalis*, or rattle-box, of the Eastern and Central United States, which causes 'crotalism,' or Missouri-bottom disease, so-called from its prevalence along the Missouri river-bed, and marked by loss of flesh and decline in vigour.

The Texan *Sophora secundiflora*, frijolillo, or coral bean, and *S. sericea*, or silky sophora, of the Southern Great Plains, are named as causing locoism.

Species of *Lessertia*, notably *L. annularis*, are believed to cause the South African C'Nenta disease (*q.v.*).

A consideration of the poisonings attributed to plants of this order gives the impression of their great diversity in character, and especially of the vagueness in our knowledge of the causation of such important and well-defined diseases as lathyrism and locoism. It emphasises the need for careful classification of the plants implicated, and for more exact experimental, clinical, and chemical study.

ROSACEÆ.

The *Amygdalus* varieties contain cyanogenetic glucosides, and an account of poisoning by them has been given under Cyanides (*q.v.*). The exotic species, *Quillaja Saponaria*, native to South America, is a source of saponin, and for an account of poisoning by this agent reference may therefore be made to the description of the Caryophyllaceæ.

CUCURBITACEÆ.

The *Cucurbitaceæ*, or gourd family, is represented in the wild flora of Britain by *Bryonia dioïca*, or white bryony. The squirting cucumber, *Ecbalium Elaterium*, and bitter

apple, or colocynth, *Cucumis Colocynthis*, are found in Southern and Central Europe.

Fig. 27.—Bryonia Dioïca (White Bryony).

Botanical Characters.—Only the wild white bryony need be described here. It must not be confused with the

black bryony or *Tamus communis*, which belongs to the *Dioscoridaceæ*, and which probably contains the same or a similar active principle.

Bryonia dioïca (Fig. 27) is a perennial climber common in hedgerows in England, though not found in Scotland and Ireland. It differs from *Tamus* in having leaves divided into five or seven broad deep lobes, as compared with the entire leaves of *Tamus*. The flower is green and the berry red.

Active Principles.—The white bryony contains the glucoside *bryonin*, the squirting cucumber contains *elaterin*, and the colocynth contains *colocynthin*. These glucosides belong to the jalap or colocynth group of drastic purgatives, which also includes jalap, gamboge, podophyllum, leptandrin, and euonymin. Some, such as jalap and colocynth, are irritants to the mucous membranes of the eyes, nose, and throat.

Symptoms.—Our literature is not rich in examples of poisoning by bryony or by the allied purgatives. Gamgee* quotes Orfila on the effects of the root of white bryony on animals, stating that dogs show great dulness after ½ ounce, and die within twenty-four hours, not showing other symptoms. According to Hertwig, 2 pounds of the fresh or 6 to 8 ounces of the dried root given to horses did not cause purging, but abdominal pain, loss of appetite, accelerated breathing, fever, dulness, and copious urination. Cornevin states that bryony promotes sweating, and causes a livid hue, nausea, diuresis, and abundant painless watery defæcation, to which are added in case of poisoning nervous symptoms of stupor and tetanic convulsions. There may be superpurgation or a suppression of defæcation.

The *lesions* are not significant, and Cornevin concludes that the purgative effect is not primitive but secondary, and of reflex origin. The active principle is found in the alimentary tract, urine, blood, and bile.

As regards other observations on bryony, J. E. King, in 1855, noted hæmaturia in horses to which white bryony had been given to improve the condition. J. S. Auger †

* Veterinarian's Vade-Mecum, 1868, p. 190.

† *Vet. Record*, 1899, p. 254.

encountered a case in which a horse ate garden clippings containing white bryony, and observed no abnormal symptoms save stiffness of the muscles of the loins, which passed off on application of mustard and embrocation.

Chemical Diagnosis.—Bryonin and the glucosides of allied nature are separated by organic solvents (*e.g.*, chloroform) from the acid liquid in seeking for vegetable poisons. Sulphuric acid colours bryonin orange-yellow, then red, and, on warming, violet. Colocynth similarly gives a yellowish-red and then brown colour, and elaterin a yellowish-brown and then dark-red coloration.

The chemical tests are not satisfactory or clearly diagnostic. Taken in conjunction with the finding of parts of the suspected plants, such results of analysis are, however, valuable confirmatory evidence.

CRASSULACEÆ.

The only species of this family native to Britain, and to which poisonous properties are commonly assigned, belong to the stone-crops—viz., *Sedum acre*, common stone-crop, wall-pepper, creeping jack, or gold-dust; and *Sedum album*, the white stone-crop. Both are found on walls and rocks, the former having yellow and the latter white flowers.

They are stated to contain an acrid juice, and in *S. acre* an alkaloid *sedine*, allied to piperine of pepper, but nothing of a precise nature is known as to its composition. Poisoning is not likely to occur, save, perhaps, among birds (Cornevin).

Cornevin injected juice corresponding to 105 grains per 2 pounds body weight into a dog, and noted salivation and muscular tremors, developing into choreic movements, more marked in the posterior than the anterior members. The respiration was deep and accelerated, and sense impaired. To this succeeded somnolence and coma, lasting about twelve hours, when the normal condition was regained. There was abundant urination and diarrhœa.

Amongst the South African plants belonging to this

order, varieties of *Cotyledon*—viz., *C. ventricosa, Eckloniana,* and *orbiculata*—are quoted as poisonous, and causing the C'Nenta disease (*q.v.*).

UMBELLIFERÆ.

The umbel family contain important poisonous plants, of which those belonging to the genera *Conium, Cicuta, Œnanthe, Æthusa,* and *Chærophyllum* (*Anthriscus*) are the commonest.

Conium.

The only member of this genus is the very common and well-known *Conium maculatum,* or spotted hemlock, the poisonous nature of which has been known for centuries. Infusion of the hemlock was a favourite poison among the Greeks, and was the agent of the official suicide of Socrates.

Botanical Characters.—*Conium maculatum* (Fig. 28), or spotted hemlock. An erect, branching annual or biennial, 3 to 5 feet high, or sometimes more, glabrous, and emitting a nauseous smell when bruised. Leaves large and much divided into numerous small ovate or lanceolate deeply cut segments; the upper leaves gradually smaller and less divided. Umbels terminal, not large for the size of the plant, of 10 to 15 rays. Bracts short and lanceolated; those of the general involucre variable in number; those of the partial ones turned to the outside of the umbel. Fruit about 2 lines long. The stem is often conspicuously marked with purplish-red spots. The plant is common on the banks of streams, in hedgerows, and the borders of fields.

Active Principle and Doses.—In the green state all the parts are poisonous, less so in northerly than in southerly latitudes, the root containing only small quantities of the active volatile alkaloid conine. Slow drying or boiling results in the loss of most of this volatile alkaloid. The plant is liable to be eaten more particularly in early spring;

sheep have been stated to be comparatively insusceptible, but several cases of sheep poisoning by it are on record.

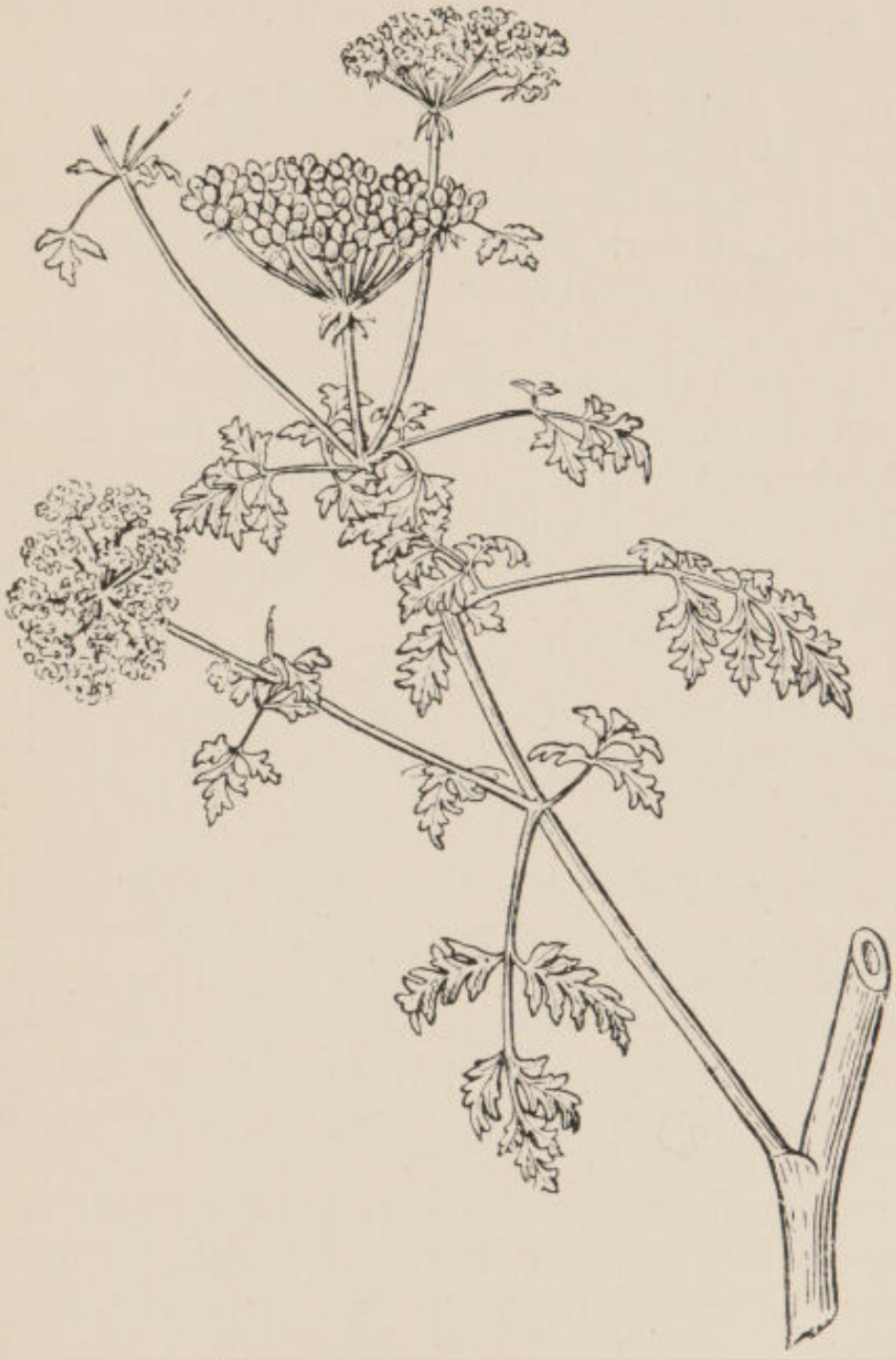

Fig. 28.—Conium Maculatum (Spotted Hemlock).

(From Smith's 'Veterinary Hygiene.')

According to Cornevin from 4 to 5 pounds of the fresh plant will kill a horse, and from 8 to 10 pounds an ox.

Poisonous Effects and Symptoms.—The general effect of conine is the production of paralysis of the motor nerve-endings, dyspnœa resulting from that of the pectoral nerves, and acceleration of the heart from that of the inhibitory fibres of the pneumogastric.

In the *horse* there is nausea, gritting of the teeth, accelerated respiration and dyspnœa, muscular trembling, beginning first in the posterior members, difficulty in walking, and paralysis; loss of sensibility, low temperature, rapid pulse, and death by arrest of respiration.

With the *ox* there is salivation, arrest of digestion, constipation, and profound stupor[7]; the respiration is rapid. In some instances bloody evacuations have been noticed.[1,5]

In *sheep*[2,3,4] the abdomen is tucked up, there is a dazed appearance, dilatation of pupils, unsteady gait, the hind limbs being dragged, coldness, and death after a few convulsive movements.

In the *pig*[6] there have been noticed prostration and inability to move, coldness, slow breathing, livid mucous membranes, eyes amaurotic, imperceptible pulse, paralysis, particularly of the posterior members, and no convulsions.

Conine is *eliminated* by the urine and by the lungs, imparting its peculiar odour to the exhaled air.

Post-Mortem Appearances.—As regards the alimentary tract, these are not characteristic, the poison not being an irritant, but some congestion may be noticed. The organs are engorged, blood black and tarry; the right heart filled, the left almost empty.

Treatment should consist in the evacuation of the stomach and purgation; tannic acid, to remove the alkaloid; warmth and stimulants—*e.g.*, strychnine, atropine, and alcohol.

Chemical Diagnosis.—Conine, being volatile, is separated by steam distillation from the alkaline material. The odour of the pure alkaloid is very characteristic, but is generally masked by the smell natural to the organic matters. If much conine is present, oily drops are seen

in the distillate. In the absence of definite chemical tests the effect on a mouse or frog should be observed.

REFERENCES TO HEMLOCK.

[1] C. Aggio, *Vet. Jl.*, 1907, p. 599.
[2] W. Graham Gillam, *Vet. Record*, 1906, p. 88.
[3] W. Graham Gillam, *Vet. Record*, 1897, p. 703.
[4] B. Freer, *Vet. Record*, 1893, p. 3.
[5] L. T. Barker, *Veterinarian*, 1873, p. 601.
[6] J. Gerrard, *Veterinarian*, 1873, p. 107.
[7] Holford, *Veterinarian*, 1841, p. 600.

Cicuta.

Botanical Characters.—*Cicuta virosa* (Fig. 29), cow-bane or water hemlock. Stem hollow, somewhat branched, attaining 3 or 4 feet. Leaves twice or thrice pinnate or ternate, with narrow lanceolate acute segments, 1 to 1¼ inches long, bordered with a few unequal acute teeth. General umbels of from ten to fifteen or even more rays. Bracts of the partial involucres subulate, not quite so long as the pedicels. The habitat is in wet ditches and on the edges of lakes, and it is very local in England, Ireland, and southern Scotland.

The roots are hollow and septate in structure, from 2 to 4 inches long, and about 1½ inches in diameter, and marked by transverse scars of leaf bases.

The *Cicuta* species are confined to the Northern Hemisphere. In America, *C. maculata*, water hemlock or beaver poison, is abundant in the United States. *C. vagans* has killed cattle in Oregon and Washington, and *C. bolanderi* in marshy land in California has also been reported.

Toxic Principle.—The poisonous principle is not known with certainty; the roots contain a yellowish acrid juice of peculiar smell, and a small quantity of a terpene, but no volatile alkaloid. According to Dragendorff there is an active principle, which has been called *cicutoxin*, classed by Cushny in the picrotoxin group. E. M. Holmes, F.L.S.,

in an article* on the *Cicuta*, states that the North American species—*Cicuta maculata*, the spotted cowbane, or beaver

FIG. 29.—CICUTA VIROSA (COWBANE).
(From Smith's 'Veterinary Hygiene.')

poison—has in its seeds a volatile alkaloid, resembling conine.

* *Pharmaceutical Journal*, 1911, p. 430.

Symptoms.—*Cicuta virosa* ranks as one of the most dangerous of the *Umbelliferæ*, the root having frequently caused poisoning. Cobbold* noted a case in Brittany when eleven beasts died with violent symptoms of vertigo, which set in within two hours of eating, the first death occurring within six hours. C. Skirrow Addison † observed a case of poisoning of cattle by eating the roots which had drifted to the side of a lake at Clones, in Ireland. The animals were lying on their right sides, head extended, and respiration very hurried. Froth had collected at the mouth and nostrils, and there was tympanites, which greatly increased shortly after death. The limbs were extended, and alternately stiffened and relaxed. Some animals appeared to have died without a struggle. The post-mortem revealed nothing; there was no gastric irritation, and there were no extravasations of blood.

Cowbane appears to be a most powerful narcotic poison, the extreme rapidity of death recalling the action of yew.

Chemical Diagnosis. — Cicutoxin does not possess characteristic chemical reactions. Diagnosis must therefore depend on the discovery of parts of the plant, especially of the roots, in the ingesta. Like many *Umbelliferæ*, the plant contains the substance, *umbelliferone*. A water extract of the plant shows a blue fluorescence due to this substance, but this does not definitely establish *cicuta*, merely pointing to the presence of an umbelliferous plant.

Œnanthe.

Botanical Characters — *Œnanthe crocata, or Water Dropwort* (Fig. 30).—A stout, branched species, attaining 3 to 5 feet; the root-fibres forming thick, spindle-shaped tubers close to the stock; the juice both of the stem and roots becoming yellow when exposed to the air. Leaves twice or thrice pinnate; the segments much larger than in the other species, always above ½ inch long, broadly cuneate or

* *Veterinarian*, 1877, p. 572. † *Vet. News*, 1911, p. 83.

rounded, and deeply cut into three or five lobes. Umbels on long terminal peduncles, with fifteen to twenty rays, 2 inches long or more; the bracts of the involucres small and linear, several in the partial ones, few or none under the general umbel. The pedicellate flowers at the circumference of the partial umbels are mostly, but not always, barren, the central fertile ones almost sessile. Fruit cylindrical, with long erect styles, the ribs broad and scarcely prominent. The habitat is similar to that of *Cicuta virosa*, and the plant is common in Great Britain.

The root of *Œnanthe crocata* has been the cause of several poisonings, as, for instance, of a gang of convicts at Woolwich in 1835; and, like the *Cicuta*, has an extremely rapid paralysant effect. *Œnanthotoxin*, the active principle, resembles cicutoxin.

Symptoms. — According to W. Graham Gillam,* the symptoms recall hemlock poisoning, with the addition of green fœtid diarrhœa.

Cornevin gives the toxic quantities of the root for the horse 0·1, the ox 0·125, the sheep 0·2, the pig 0·15, the rabbit 2·0, per cent. of the body weight.

The juice of *Œnanthe* has a powerfully irritant effect on the skin.

In the *ox* there is foaming, distended nostrils, shivering, rapid and laboured respiration, spasmodic contractions of the limbs; the subject reels in a circle for several minutes, falls and dies.† Should death not occur, the paralysis persists. Wallis Hoare‡ saw cows poisoned by the roots left in a field after a flood, and observed well-marked delirium, succeeded by rapid death.

With the *horse* the onset of symptoms is rapid, the nervous predominating; and with the *pig* large doses fail to cause vomition, and death occurs with the rapidity of cyanide poisoning.

Post-Mortem Appearances.—In acute poisoning the thoracic and abdominal viscera are normal, there is some

* *Vet. Record*, 1906, p. 88.
† See *Veterinarian*, 1873, p. 695. ‡ *Vet. Jl.*, 1887.

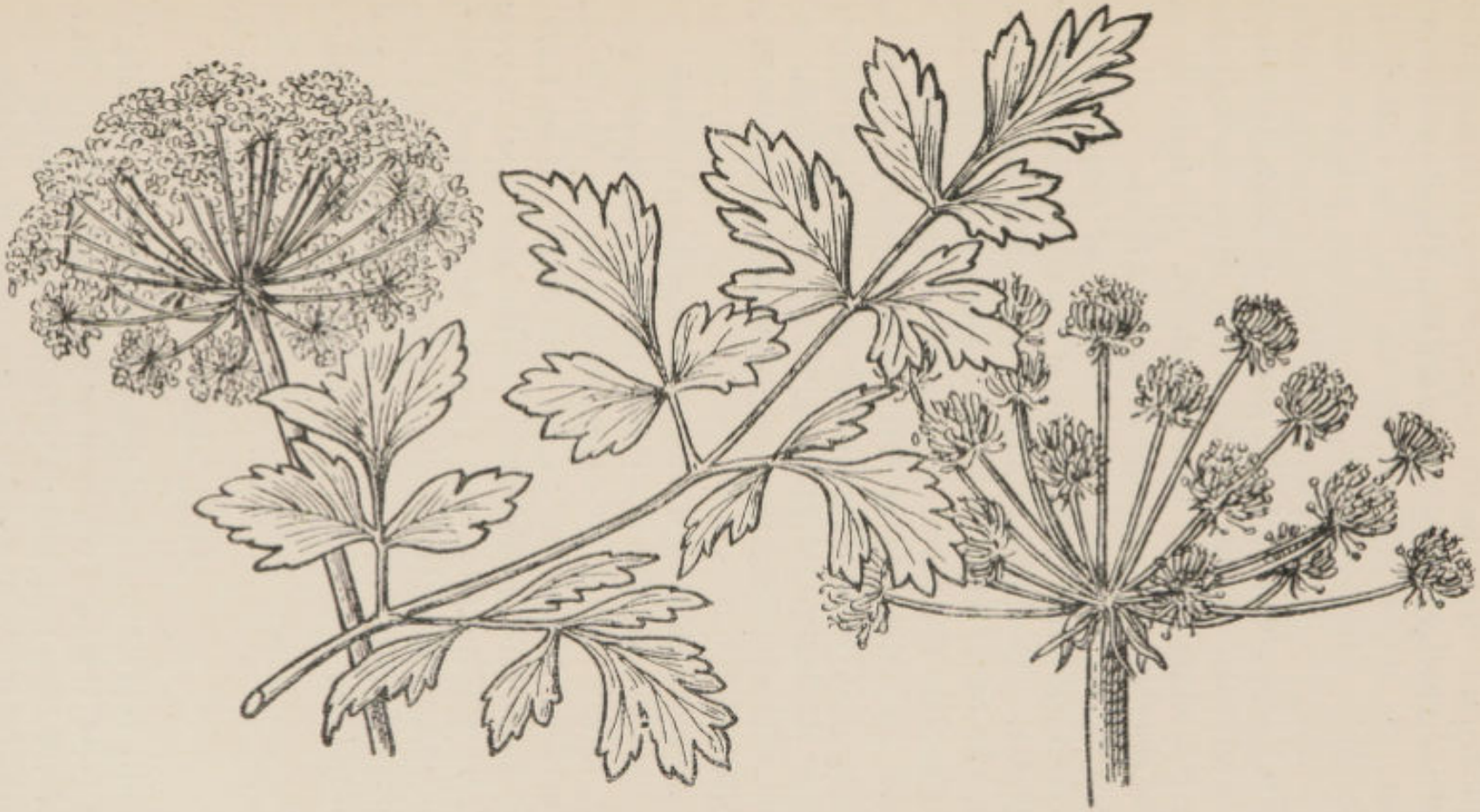

FIG. 30.—ŒNANTHE CROCATA (WATER DROPWORT).

(From Smith's 'Veterinary Hygiene.')

congestion of the nervous centres, the veins of the *pia mater* distended, and there are apoplectic foci. In more protracted cases ecchymosed patches are found in the abdominal viscera.

Other Genera.

Æthusa cynapium, or fool's parsley, is not a dangerous plant to animals. From confusion with edible parsley it has led to poisoning of the human subject. It contains an alkaloid *cynapine*, also present in *Æthusa fatua*.

Chærophyllum sylvestre, wild chervil or asses' parsley (*Anthriscus sylvestris* Hoff), is one of the commonest British *Umbelliferæ*. In spite of its odour and acrid taste the ass eats it, and other animals also take the plant, apparently without serious results, for it is a common food for tame rabbits. According to a German observer quoted by Cornevin, pigs, having eaten the green plant, displayed paralysis, dilatation of the pupils, refusal of food, and enteritis. The post-mortem revealed acute gastro-intestinal inflammation.

Other species have been named by various authorities as dangerous or objectionable; but further evidence being wanted, it will suffice to name them here as suspected. They include: *Daucus carota*, the wild carrot, which forms a bitter acrid root in distinction to the cultivated carrot; *Heracleum Sphondylium*, cow parsnip or hog weed, which appears under certain conditions to develop an irritant juice; *Sium angustifolium*, cow-cress or fool's water-cress; and *S. cicutæfolium*, the hemlock water-parsnip of the United States.

ARALIACEÆ.

The only representative of this family found in Britain is *Hedera helix*, the very common and well-known ivy. The berries have been long known to be poisonous, having been mentioned by Pliny, and cases of the poisoning of children by them have been recorded. Over and above an emetic

and purgative action, the berries produce nervous effects like drunkenness, involving excitement, then coma, laboured respiration, and the like symptoms. Cases of the poisoning of animals are not on record. The leaves are eaten by cattle without effect; indeed, are sometimes given to sick cows by country people as a dainty (Wallis Hoare). Birds and small animals might possibly be poisoned by the berries.

CAPRIFOLIACEÆ.

Belonging to this family are the elders, *Sambucus nigra*, the common elder, and *Sambucus Ebulus*, the dwarf elder. The former is common in hedges, attains 8 to 15 feet, has ovate leaves, white flower, and black berries; whilst the latter is found on waste ground, reaches 2 to 3 feet, has lanceolate leaves, pink flowers, and black berries. All parts of these plants exhale a strong and repulsive odour, and have been found to contain an emetic and purgative oil, a resin, and traces of valeric acid. Animals do not eat the plant spontaneously, and in the very rare cases of poisoning the symptoms and lesions are those of superpurgation.

VALERIANACEÆ.

Similar remarks to those above made apply to *Valeriana officinalis*, common valerian, cat's valerian, or all-heal, and *V. dioïca*, or marsh valerian, which contain an essential oil, or mixture of oils, and valeric acid. It is most unlikely that sufficient of these plants would be eaten by animals to cause serious functional disorder.

DIPSACEÆ.

A case of injury by *Scabiosa succisa*, or devil's-bit, has been placed on record by J. Moir.* Bullocks and heifers

* *Vet. Record*, 1899, p. 523.

which had broken into a plantation and eaten the herb, showed salivation, champing, gritting of the teeth, and twitching of the facial muscles. The tongue, which was slightly protruded, had an abraded patch about as large as a crown-piece, about 3 inches from the tip, and was very swollen and sensitive.

In a test experiment on cattle the plant was found by Moir to produce violent inflammation of the tongue and mouth.

COMPOSITÆ.

The very large and widely-distributed order of *Compositæ* fortunately embraces comparatively few poisonous plants, but in all parts of the world poisoning has been more or less definitely attributed to some species.

In Europe the possibility of poisoning by *Artemisia* on account of the volatile oil of absinth has been noted (*q.v.*), whilst *Lactuca virosa*, closely allied to *L. scariola*, the wild or prickly lettuce, has a whitish latex of disagreeable odour. It is not eaten willingly by animals, and no doubt large quantities would be required to cause harm. Its effects recall those of opium, the general action being one of narcosis.

In Algeria the roots of *Atractylis gummifera* are noted by Cornevin as being sometimes eaten by stock in times of scarcity, and to exert narcoto-irritant, conjoined to cardiac effects, resembling those of colchicum.

In the United States *Helenium autumnale*, the sneeze-wort, stagger-weed, or false sunflower, is alleged to kill sheep, cattle, and horses, unfamiliar with it in the Southern and Eastern States. Though generally avoided by stock, it is further alleged that a taste for it is sometimes developed. Species of *Solidago*, or golden-rod, have been blamed for the poisoning of horses in Wisconsin, but the damage may be due to a parasitic growth on the plant.

In Texas seedlings of *Xanthium canadense*, or cocklebur, have been reported as rapidly fatal to pigs, and *X. spinosum*

and *X. strumarium*, which are occasionally found in Britain, have also been suspected of being poisonous.

In South Africa several plants are described as 'bietouw.' They appear to cause fatal tympanites, unless kept down by grazing. When allowed to grow up, deaths may occur on the return of stock. The species implicated under this head, given by Walsh, are *Haplocarpha lyrata*, *Dimorphotheca cuneata* and *D. nudicaulis*, *Lasiospermum radiatum*, and *Osteospermum moniliferum*.

Two African stock diseases caused by plants of the *Compositæ* appear to be more definitely known, and therefore merit more detailed description—viz., 'vomeerziekte,' or vomiting sickness, and senecio poisoning, or 'Molteno cattle disease.'

Vomeerziekte.

This complaint is caused by the small-branched shrub *Geigeria passerinoides*, or vomeerbosje, found in the North Eastern parts of the Cape Colony. The flower is yellow, about $\frac{3}{4}$ inch in diameter, and resembles the sunflower in structure.

Poisoning by this plant affects chiefly goats and sheep. Walsh states that when fed experimentally to sheep the results are negative, and suggests that it may be that the plant must be eaten in large quantities, or that it is only dangerous at certain seasons, or that it does not generate any poison when well nourished.

Symptoms.—Sheep and goats vomit continually, the vomiting being accompanied by a husky cough, and attempts to swallow the vomit. There is great loss of condition and attendant weakness, and death follows exhaustion.

The *lesions* are inflammation of the fourth stomach, and inflammation of the lungs and bronchi, caused no doubt by passage of vomitus into the trachea.

In *treatment* alkalis and sedatives are advised, since the irritant seems to be of an acid character. After vomition has been checked, purgatives may be given.

Senecio.

The *Senecio Burchelli* and *Senecio latifolius* are the causes of the so-called Molteno cattle disease, or straining sickness, which affects both cattle and horses. These plants are members of the numerous genus *Senecio*, represented in Great Britain by the rag-worts (groundsel), which have not very definitely been proved to be injurious.

Active Principles.—The senecios have received considerable attention from the chemical and therapeutic points of view. Common groundsel (*S. vulgaris*) has been shown to contain in the underground parts two alkaloids, senecionine and senecine (Lutz and Grandval, and Lajoux, 1895). *S. vulgaris* has been used in dyspepsia (Dalché, 1904), and *S. jacobœus* in functional amenorrhœa. The latter plant is also the cause of the New Zealand 'Winton disease,' which is acute cirrhosis of the liver.

Debierre (1889) examined the Mexican *S. canicida*, or dog poison, and recognised stages of excitement, rest, and spasm in its action. It causes death from respiratory paralysis. The spasms simulate those of strychnine, but the reflex irritability is lowered.

H. E. Watt * examined *S. latifolius* from South Africa at the Imperial Institute. He isolated two alkaloids, senecifoline and in smaller quantity senecifolidine. The former examined by Cushny was found to exercise the same effects as the whole plant.

Toxic Doses.—According to experiments by the Cape Agricultural Department, quoted by Walsh, ½ pound daily of *S. Burchelli* for four days killed an ox on the fifth day, whilst 8 to 10 pounds of *S. latifolius* in daily feeds of 2 to 6 ounces killed in about six weeks. When the plant is common it is not eaten save in times of scarcity, though it is taken freely by strange stock.

Symptoms.—A good account of straining sickness is given by W. H. Chase,† who fully investigated the disease.

* Transactions of the Chemical Society, 1909, p. 466.
† *Record*, 1904, p. 425.

He observed persistent diarrhœa (in cattle diminished lactation), the coat dry and staring, no desire for food, and straining soon sets in. At first slight, the straining quickly increases in frequency (fifteen per minute) until just before death. As a result the rectum is often everted and blood-vessels ruptured. From the commencement of straining there is great pain, the animals sometimes lying down and groaning, with head outstretched, or sometimes standing and getting into a frenzied condition. Eventually there is unconsciousness, and death in two to four days after appearance of symptoms.

F. Chambers * has described the effect of senecio on horses leading to cirrhosis of the liver, and tentatively associates this poisoning with roaring.

Both authorities agree in the impossibility of a cure unless in very early stages. Chambers says that a horse may be kept alive for a few months by careful feeding, but when let out on the veldt the stomach becomes again engorged with grass, and the original symptoms re-appear.

Post-Mortem Appearances.—In cattle, according to Chase, these are most prominent in the liver, which has a leathery feel, and on cutting is very tough, with acute venous congestion in a few cases. The organ is reduced in size, slaty-blue in colour, and has lost the sharp edges. The gall-bladder is distended, and may contain as much as 32 ounces of bile, very viscid, and yellowish-black to black in colour. The interior of the gall-bladder sometimes has a number of red spots about the size of a pin's head, sometimes also seen on the interior of the urinary bladder and on both surfaces of the heart membrane. The first three stomachs are healthy, the fourth covered with hæmorrhagic spots. A thickening by a submucous gelatinous exudate of the folds of the mucous membrane is a constant lesion. There is generally inflammation of the small intestines, and the bloodvessels of the last part of the canal are congested on account of the straining. When the liver is not badly

* *Vet. News*, 1911, p. 318.

diseased there is more change in the fourth stomach, and *vice versâ*.

In the horse, Chambers observed in six of nine cases enormous distension of the stomach. In eight cases the heart and aorta were greatly enlarged.

CAMPANULACEÆ.

Lobelia urens, or acrid lobelia, is the only poisonous species of this family found wild in Britain, and is very rare, being found on moist heaths in Dorset and Cornwall.

The genus *Lobelia* is widely diffused, though scarce, in the greater part of Europe, and is found in America and Australasia. Of American lobelias, *L. inflata* (Indian tobacco), *kalmii*, *spicata*, and *syphilitica*, are regarded as suspicious, and are sometimes found in meadow hay.

Active Principle.—The lobelias contain an alkaloid, *lobeline*, analogous in many physiological respects to nicotine and conine. It is extracted for pharmaceutical purposes from the leaves of the American *Lobelia inflata*.

Symptoms.—G. Fleming,* in 1873, gave an account of poisoning by a lobelia in Australia. According to his observations, after eating plentifully of the plant, cattle show lassitude, and on being driven hard, or suddenly startled, drop in convulsions, death following in a few minutes.

According to Cornevin, lobelia causes some inflammation, and acts like belladonna.

Chemical Diagnosis.—The alkaloid lobeline, separated from organic matter, is not very well characterised by diagnostic tests. In a neutral solution potassium bichromate gives a yellow precipitate (*cf.* strychnine), and Fröhde's reagent colours violet, deepening within about two hours, and eventually becoming brown. The pure alkaloid does not give this reaction.

* *Veterinarian*, 1873, p. 451.

ERICACEÆ.

The poisonous members of this family likely to be found in Great Britain are the exotic shrubs, *Rhododendron*, *Azalea*, and *Kalmia*, or laurel ivy, widely cultivated for ornamental purposes.

Rhododendron appears to be often eaten freely by cattle and sheep, and several cases of poisoning by it are on record. In America *R. californicum* is native on the Pacific Slope from San Francisco to British Columbia, and has been reported as poisonous to sheep in Oregon, whilst *R. maximum*, native in the Allegheny Mountains, is also fatal to stock.

Active Principle.—Our chemical knowledge of rhododendron poison is not complete. The leaves contain a tannin, resolved by acids to the yellow-red rhodoxanthine, and it is noteworthy that a reddish tint has been observed [1] in the milk of a cow poisoned by this plant. The leaves also contain a bitter yellowish-brown resin, and contain the active principle *andromedotoxin*.

Symptoms.—After ingestion of rhododendron leaves, *cattle* and *sheep* manifest intense pain, diarrhœa, and discomfort, gritting the teeth, salivating, and frequently vomiting. There is suppression of lactation, trembling, and spasms, vertigo, loss of power, and death.

Recent cases of rhododendron poisoning of cattle are those noted in references 1, 2, and 4. They agree in all the salient points, serve to emphasise the generality of actual vomition under the influence of this poison, and also well display the nervous symptoms.

One of the earliest observations [7] is on *calves*, and is remarkable, in that no diarrhœa or actual vomition was observed, but there was the same disinclination to move, staggering, and reeling gait.

In *sheep* [3,5,6] there are similar symptoms, but actual vomition does not seem so general as with cattle.

Post-Mortem Appearances.—These are not well marked. The mucous membrane of the rumen may be easily detachable, but there is not extensive inflammation. Rhododendron leaves will probably be found in the stomachs.

Treatment.—Most of the cases referred to recovered under treatment by means of a brisk oleaginous purgative, followed by chlorodyne or counter-irritants to abdominal pain, and general stimulants and tonics, such as ammonium carbonate and spirits of nitrous ether.

Wallis Hoare[2] in the case of a young cow, gave brandy; chlorodyne, ℥ii.; sp. æth. nit., ℥iv.; ol. lini, O.iii.; and opiates, with recovery on the fifth day.

Chemical Diagnosis.—The history of the case and the detection of leaves of the plant in vomit or contents should render this superfluous. As, moreover, the chemical reactions of the active principles have not yet received precise study, a diagnosis by this means is at present of doubtful value.

In a case of a cow poisoned just after calving, Wallis Hoare had difficulty in differentiating from milk fever, until he observed the vomition of rhododendron leaves.

REFERENCES TO RHODODENDRON.

[1] H. B. Eve, *Vet. Record*, 1907, p. 4.
[2] E. Wallis Hoare, *Vet. Record*, 1906, p. 630.
[3] T. Slipper, *Vet. Jl.*, 1906, p. 439.
[4] C. H. Golledge, *Vet. Record*, 1900, p. 326.
[5] C. Williamson, *Veterinarian*, 1865, p. 305.
[6] W. C. Spooner, *Veterinarian*, 1865, p. 281.
[7] B. Kettle, *Veterinarian*, 1859, p. 435.

Cases of poisoning by *Azalea* are not numerous, but the California azalea (*A. occidentalis*) is dreaded by shepherds in the Southern Sierras. According to Cornevin, the symptoms approach those of *Lolium temulentum* (*q.v.*), whilst the same authority quotes Xenophon's narrative of the symptoms of delirium and prostration exhibited by those of his soldiers who partook of honey from azalea in Asia Minor.

Since the azalea is a common ornamental plant, and since it is very dangerous, caution must be exercised in regard to cuttings of the plant.

Kalmia angustifolia, or sheep laurel, is abundant in the North-Eastern United States, and *K. latifolia*, found throughout the greater part of the Eastern States, and known as laurel or ivy, is regarded as the most poisonous species of the *Ericaceæ*, killing scores of cattle and sheep annually.

Kalmia, or *laurel ivy*.—J. Young, of Braintree,* signalised the death of 20 out of 150 ewes, which all suffered from eating a species of kalmia. On the next day they were all lying down, and showing symptoms like those of gripes in horses. The bodies were full, but not swollen. Castor oil was swallowed with difficulty. Before death there was great stiffness, probably tetanic.

Amongst others of the *Ericaceæ*, *Ledum palustre* is noted by Cornevin as causing similar poisoning to rhododendron on the Continent, and *L. glandulosum* and *grœnlandicum* are suspected in America. *Leucothoë catesbœi*, the branch-ivy, hemlock, or calf-kill of the Alleghany Mountains, is known to be fatal to all kinds of stock in that district, and *L. racemosa* has been reported from New Jersey as especially fatal to calves.

OLEACEÆ.

This family is represented in Britain only by *Fraxinus*, the ash, and *Ligustrum vulgare*, the privet, which has been introduced into America. The common privet has been observed to cause poisoning by Turner.† It contains a glucoside, *ligustrin*, which has not received very close study.

Turner's observations were made on horses put into a field with an unclipped privet hedge.

The *symptoms* observed were loss of power in hind quarters, pulse 50, temperature 102° F., mucous membranes

* *Veterinarian*, 1877, p. 77.
† *Vet. Record*, 1904, p. 319.

slightly injected, and pupils dilated. Death occurred within from thirty-six to forty-eight hours.

PRIMULACEÆ.

This family includes *Cyclamen europæum*, the common cyclamen or sow-bread, and *Anagallis arvensis*, the common pimpernel or shepherd's weather-glass.

Cyclamen is not an indigenous plant, but has established itself locally in Kent and Sussex from garden culture. The pimpernel is a common weed of cultivation, and of corn-fields, gardens, and waste places.

Cyclamen contains in the roots the glucoside *cyclamin*, and pimpernel contains the glucoside *smilacin*, both of which are varieties of saponin.

The effects are thus similar to those of other saponin-containing plants, and reference may therefore be made to these (see *Caryophyllaceæ*).

As regards pimpernel, the plant is too small to make it likely that a large animal could eat enough to cause harm, but extracts have been proved to be poisonous to the horse (Cornevin).

APOCYNACEÆ.

This family is mainly tropical, and the common and harmless *Vinca*, or periwinkle, is the only representative in Britain. But many plants of this order are notable causes of poisoning throughout the tropics. As regards the active principles, the species involved all contain glucosides, which are classed by Cushny in the digitalis series.

The genus *Apocynum* is represented by *A. androsæmifolium*, found in Central Europe, Asia, and America, and *A. cannabinum*, or Canadian hemp, used in America as a fish poison. The acrid juices, which contain *apocynin*, provoke vomiting and diarrhœa, and, if in quantity, fatal superpurgation (Cornevin).

Strophanthus hispidus, or *Kombé*, is the West African Gaboon arrow-poison, but no data as to animal poisoning by it are on record, nor are there such regarding the Madagascar *Tanghinia venenifera*.

Oleanders.

Varieties of the oleanders are very important and well known, especially to tropical toxicology. The *Nerium oleander* is a commonly cultivated evergreen plant on the continent of Europe, grows out of doors in the Southern and Western United States, is a garden and hedge plant in South Africa, grows wild in Mexico, and is native to Asia. In India *Nerium odorum* is the sweet-scented oleander, with white or pink flowers (vernacular, *Kaner*); *Cerbera thevetia* is the bastard, or yellow oleander (vernacular, *Pila kaner*); and *Cerbera odallum* is closely related to them.

Active Principles.—As above stated, these are glucosides allied to digitalin. From *N. odorum* there have been separated *neriodorin*, *neriodorein*, and *karabin*, of which neriodorin and karabin stimulate the vagus and cause a slow, forcible heart-beat, and then, by exhaustion, a rapid feeble action. Karabin also exercises spinal effects like strychnine. *Thevetin*, from *thevetia*, resembles digitalis, with sometimes convulsant action. These poisons are found in the sap and leaves of the plants.

The *dose* is not large, a single growing top of oleander having been said to be fatal to cattle and horses, whilst men have been fatally poisoned by eating meat cooked on skewers of the wood (Walsh). Three seeds of *C. thevetia* would probably kill a man, since two have caused dangerous symptoms (Windsor).

Symptoms.—In the experimental poisoning of animals Cornevin distinguished a phase of stupor, succeeded by convulsions, insensibility, and then paralysis. Vomiting occurs when possible, and the retching continues after the stomach is empty.

The effects of *N. odorum* were studied in India, and

noted by 'Snipe' in the *Quarterly Journal of Veterinary Science in India*, 1887, p. 50. He gave a horse 2 ounces of the leaves night and morning for three days, and they were

Fig. 31.—Nerium Oleander.

readily taken with food. He observed dull abdominal pain, anorexia, yellowness and injection of the conjunctivæ, no narcosis, and temperature normal.

The *lesions* were chiefly intense congestion of the small intestines, particularly of the duodenum and jejunum. The cæcum and colon were similarly affected; contents liquid and sanguinaceous. There was patchy congestion of the stomach, especially at the cardiac end. The brain and spinal cord were normal.

There is no antidote for the *treatment* of oleander poisoning, which is symptomatic. Evacuation and stimulation are indicated, and chloroform or chloral against convulsions, if marked.

The *detection* is attended by uncertainty, unless the poisoning was clearly indicated at the outset. It is difficult to separate the glucosides, but thevetin gives a deep blue colour on boiling with 1 to 4 hydrochloric acid, which, according to Windsor, is characteristic.

ASCLEPIADACEÆ.

The order *Asclepiadaceæ*, or milkweed family, includes plants which have an abundance of milky, acrid, and poisonous sap, and comprise *Asclepias syriaca*, cultivated in Europe, but native to America, where it is known in the North-Eastern States as milkweed or silkweed; *A. tuberosa* is the Eastern United States 'pleurisy-root'; and *A. mexicana* and *A. eriocarpa* are milkweeds of California, Oregon, and Nevada.

In Europe *Vincetoxicum officinale* and *Cynanchum acutum*, belonging to the same order, are dangerous. All these species appear to act as powerful drastics, and cause poisoning in accordance therewith.

In South Africa the disease known as 'krempziekte,' or cramp sickness, is ascribed in the Western Cape to the *Cynanchum capense*, or klimop, which is a trailing creeper; and in the Eastern Cape to species of *Cotyledon*, belonging to the *Crassulaceæ* (*q.v.*), and known as C'Nenta.

The *causation* of the C'Nenta disease has been variously ascribed to atmospheric conditions, to the plants, or to

fungoid growths on them. Nothing is known as to the chemical nature of the active principle involved.

The animal mainly attacked is the sheep, though horses and cattle may be also affected.

In *poisoning* the animals affected tend to lag behind and stagger in their gait. They may fall and lie quiet, then rise and continue to feed. Convulsions are characteristic. The head is pushed down between the forefeet and then jerked upwards and backwards, or from side to side. The convulsions may occur at regular intervals or very frequently, leading to great exhaustion and a partially paralysed condition, from which there may be recovery.

The poisoning is *treated* by means of aperient (Epsom salt), and sedatives, such as chloral hydrate or potassium bromide, which is given to the ox in doses of 1 ounce thrice daily, and to sheep or goats in doses of 1 to 2 drachms. Distension in the case of cattle may be relieved by the trocar.

CONVOLVULACEÆ.

Convolvulus.

The genus *Convolvulus* includes *C. Scammonia* and *C. jalapa*, which contain the glucosides convolvulin and jalapin respectively. These substances belong to the group of drastic purgatives. The British species of *Convolvuli*, or bind-weeds, do not appear to have been definitely proved to contain these glucosides, though it is possible that they do so.

Symptoms.—Convolvulin acts as a local purgative, but in large doses, when it encounters insufficient bile it causes an astringent effect. The *convolvulus*, bind-weed or lap-love, is dangerous to pigs. A good example of its effects was recorded by Olver.* The animals had eaten profusely of convolvulus, and displayed loss of appetite and attempts to vomit. Before death the head hung down, and the animals had a sleepy appearance.

* *Veterinarian*, 1872, p. 727.

On **post-mortem** there was found serous effusion in the abdominal cavity, and the intestines were empty, save for a little fluid and gas. The stomach was full of green food containing convolvulus. There were a few petechial patches on the villous membrane of the stomach; the other viscera were healthy, but the brain was highly congested.

Cuscuta.

The genus *Cuscuta* includes *C. europæa*, the greater dodder, which has been held responsible for poisoning.

Botanical Characters.—The dodder is a greenish-yellow, tending to red, leafless, parasitic herb. The flowers form clusters, are small, sessile, and have broad and rounded sepals. It is not very common in England, and is confined to the South. A variety, *Cuscuta trifolii*, is found in clover-fields.

Symptoms.—Dodder is stated to cause enteritis, and to evolve also nervous symptoms in *pigs*.

Holterbach* observed illness of *cows* caused by a clover containing 50 per cent. of the *Cuscuta trifolii*. He noted trembling movements of the hind quarters and swelling over the hock-joints. The back was arched, head outstretched, anxious appearance, and quick breathing. There was violent shaking, in which the hind feet alternately took part, increasing until the animal was in a frenzy. The attack declined, leaving the patient exhausted and covered with sweat. Other attacks followed, but all the animals recovered quickly, and remained healthy.

SOLANACEÆ.

The four British genera of this family—viz., *Atropa*, *Hyoscyamus*, *Datura*, and *Solanum*—each contain poisonous plants, and to them we may add the exotic tobacco, or *Nicotiana tabacum*, which is cultivated to a limited extent, and is likely to be more widely grown for the sake of the

* *Vet. Jl.*, 1908, p. 632.

nicotine. The family thus includes many of our commonest and most dangerous poisonous plants. Each is represented in America, and *Cestrum nocturnum* in South Africa, whilst datura is a common Indian poison.

Active Principles.—Before proceeding to details regarding each species it will be convenient to describe the active principles of the plants involved, which from this point of view may be divided into three groups—(1) the atropine group; (2) the solanine group; (3) the nicotine group.

(1) The atropine group comprises the genera *Atropa*, *Hyoscyamus*, and *Datura*, and the species *Atropa Belladonna*, *Hyoscyamus niger*, and *Datura Stramonium*. They contain alkaloids, or mixtures of alkaloids, of the atropine group. These are atropine, hyoscyamine, hyoscine (also called scopolamine), and atroscine (also called *i.*-scopolamine). They are very closely related chemically and also physiologically, and form the mydriatic, or pupil-enlarging, group.

(2) The solanine group comprises the genus *Solanum*, and the species *S. dulcamara* and *S. nigrum*, found wild, and *S. tuberosum* (potato), *S. lycopersicum* (tomato), and *S. melongena* (egg-plant) cultivated. They all contain the glucosidal alkaloid solanin, allied to the saponins, but containing nitrogen, and resolvable into a sugar and an alkaloid solanidine. Like the saponins, solanin is colloidal, and not easily absorbed through the intact alimentary mucosa. But solanidine is easily absorbed.

(3) The nicotine group, represented by tobacco, contains the volatile alkaloid nicotine, allied chemically and physiologically to conine.

Atropa.

Botanical Characters.—*Atropa Belladonna* (Fig. 32), deadly nightshade or black cherry, reaches about 3 feet, has an herbaceous stem, ovate leaves, solitary dark purple flowers, and black berries. It frequents waste, stony places in chalky districts, the vicinity of old castles and ruins, and is rather local in the South of England, rare in the North, but common in the South of Europe.

All parts, especially the roots, contain mainly atropine (about 0·6 per cent.), with variable proportions of the allied

Fig. 32.—Atropa Belladonna (Deadly Nightshade).
(From Smith's 'Veterinary Hygiene.')

alkaloids. Atropine usually forms a larger proportion in the old than in the young plants.

Toxic Doses.—Poisoning by the plant is rare, in part by reason of its scarcity. Cats, dogs, and birds are sensitive,

the horse and ox less so, whilst the pig, goat, sheep, and rabbit cannot be poisoned by the plant or even the root. This is probably due to the rapid elimination of atropine by the kidneys, for when given intravenously the alkaloid causes typical symptoms.

According to Cornevin, 2 pounds of the green herb daily for three days produced no pathological disturbances in horses.

Hertwig states 6 ounces of the dried root to be a fatal dose for the horse, and that cattle are equally susceptible.

Dangerous symptoms result in dogs from the administration of 30 to 50 grains of the dry plant, and $\frac{3}{4}$ of a grain hypodermically kill dogs of from 15 to 16 pounds weight (Finlay Dun).

Symptoms.—When given in toxic doses, atropine causes in animals dryness of the mouth as a noticeable effect of the general inhibition of secretion, increased pulse and respiration frequencies, and elevation of temperature. There is dilatation of the pupil, with blindness, restlessness, nervousness, delirium, and muscular trembling. After this period of excitement there ensues fall of temperature, convulsions, motor and sensory paralysis, with staggering movements, feeble and slow respiration, relaxation of the sphincters, and death in convulsions.

In dogs the pulse-rate may be as high as 400; in horses it may be about doubled.

G. H. Livesey* recorded typical symptoms, excepting that there was no mydriasis, in a fox-terrier bitch, which had licked herself after rubbing with belladonna liniment.

The **Post-Mortem** appearances are not characteristic, being those of asphyxia.

Treatment.—The treatment of poisoning consists in elimination by emetics or purgatives, and treatment of the symptoms. Sedatives may be desirable in the early stages of excitement, but it must be remembered that toxic doses of atropine cause depressant effects. Stupor is combated by movement, and stimulants, such as alcohol, ammonia,

* *Jl. Comp. Path.*, 1904, p. 359.

or caffeine. The cautious use of the physiological antidotes, eserine against mydriasis and pilocarpine against the drying up of secretions, is advisable.

Chemical Diagnosis.—Atropine and the allied alkaloids are separated in the search for vegetable poisons in cases of poisoning by any of the plants of this group. A very valuable test is an observation of the mydriatic effect on the eye of a cat. A good chemical test is Vitali's. The residue of alkaloid is heated on the steam bath to dryness, with a few drops of concentrated nitric acid. On moistening the yellow residue with alcoholic potash, atropine and its allies give a red-violet colour. The test is exceedingly delicate.

Hyoscyamus.

Botanical Characters.—*Hyoscyamus niger* (Fig. 32), or black henbane, is a coarse, erect, branching annual, 1 to 2 feet high, more or less hairy and viscid, with a nauseous smell. Leaves rather large, sessile, the upper ones clasping the stem, ovate, and irregularly pinnatifid. Flowers very shortly stalked, the lower ones in the forks of the branches, the upper ones sessile, in one-sided leafy cymes, rolled back at the top before flowering. Calyx short when in flower, but persists round the fruit, and then an inch long, strongly veined, with five stiff, broad, almost prickly lobes. Corolla above an inch long, pale, dingy yellow, with purplish veins. Capsule opening transversely, with numerous small seeds.

The henbane is somewhat rare, and of similar habitat to that of the deadly nightshade. In the United States it occurs as a weed of European origin. The West African *H. falezlez*, according to Cornevin, is eminently poisonous.

Symptoms. — Henbane poisoning is infrequent, but the plant is sometimes cultivated for medicinal use, and J. R. Welsby* has placed upon record a case in which animals were poisoned in a field thus cultivated several years previously.

* *Vet. Record*, 1903, p. 181.

There were observed nervo-muscular exaltation, eyelids and irides much dilated, eyes amaurotic and very bright,

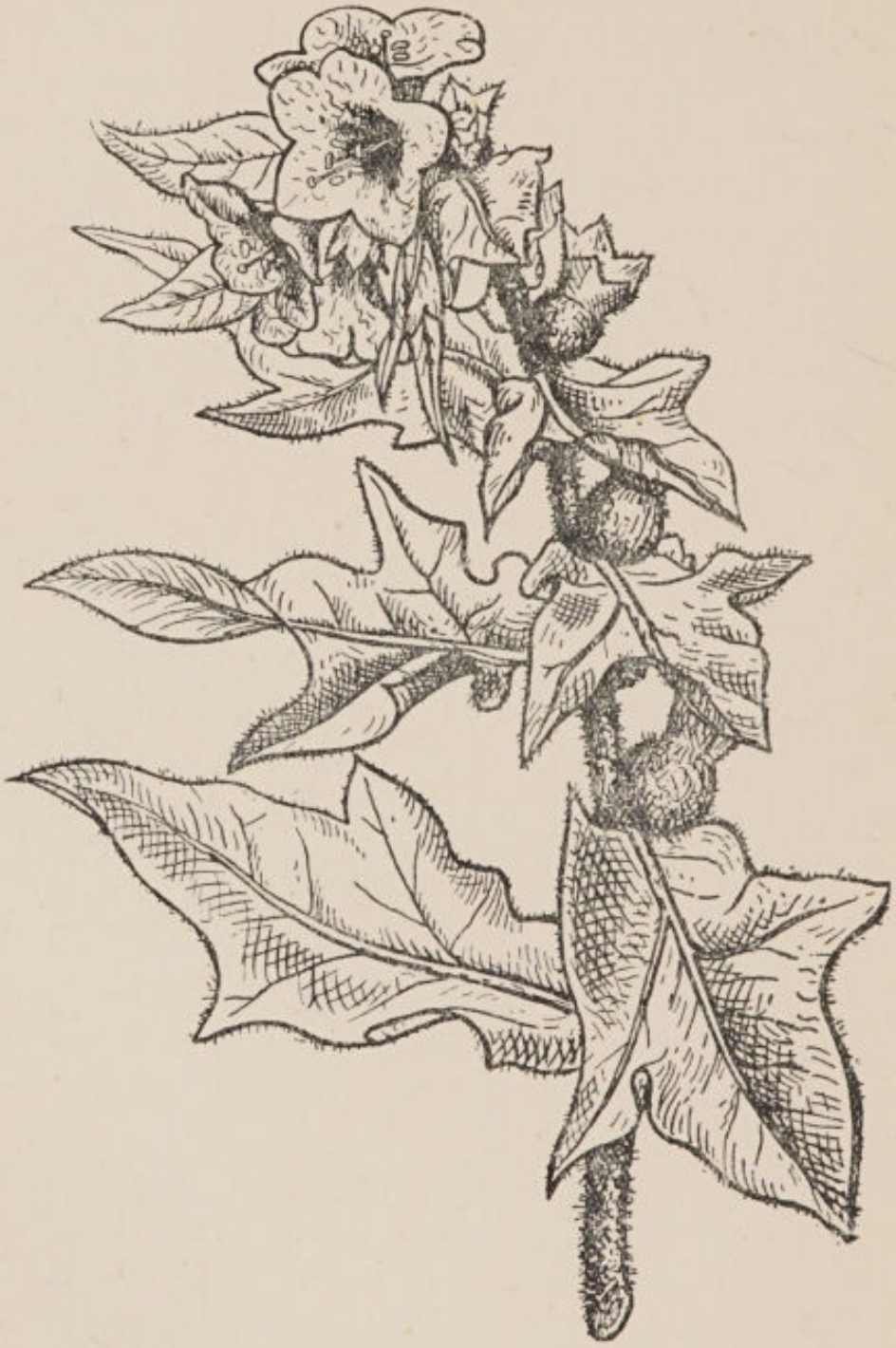

FIG. 33.—HYOSCYAMUS NIGER (BLACK HENBANE).
(From Smith's 'Veterinary Hygiene.')

pulse full, temperature normal, respiration difficult and hurried, profuse salivation, muscles of the neck and extremities in a state of tetanic rigidity, considerable

abdominal distension, stercoraceous and renal emunctories entirely suspended, death.

Creuzel* observed henbane symptoms in a cow two hours after eating the plant. The pupils were dilated, conjunctivæ injected, carotids beat violently. The animal attempted to rise, but fell again. There were general convulsions, loud respiration, salivation, and purgation.

Black henbane contains chiefly the two alkaloids, *hyoscyamine* and *hyoscine* (scopolamine). Hyoscyamine closely resembles atropine in its physiological effects, the mydriasis being less permanent and the depressant effects greater, whilst hyoscine is an extremely powerful depressant and hypnotic drug. As regards poisoning by henbane, it is interesting to remark that profuse salivation takes the place of the great dryness due to atropine.

Datura.

Botanical Characters.—*Datura Stramonium* (Fig. 34), or thorn-apple, is sometimes encountered in Southern England, being a plant originally of South American origin, now fairly widely diffused over Southern Europe.

It is a coarse, glabrous, or slightly downy annual, 1 to 2 feet high, with spreading, forked branches; leaves rather large, ovate, with irregular, angular, or pointed teeth or lobes. Flowers solitary, on short peduncles, in the forks, or at the ends of the branches. Calyx loosely tubular, about $1\frac{1}{2}$ inches long, and falls off after flowering, leaving a small rim under the capsule. Corolla about 3 inches long, bordered with five, narrow, distant teeth, usually white, but occasionally (especially in hot countries) purple. Capsule nearly globular, very prickly, with numerous wrinkled seeds. In the States it is a common weed known as 'Jimson' weed, and in South Africa it is called the 'stinkblaar.'

Symptoms.—A case of thorn-apple poisoning is on record by H. A. Sullivan.† A horse which had eaten

* *Veterinarian*, 1840, p. 661. † *Vet. Jl.*, 1905, p. 182.

FIG. 34.—DATURA STRAMONIUM (THORN-APPLE).

datura bush was down and unable to rise, pupil dilated, mouth partly open, tongue peculiarly dry, pulse quick and full, and visible mucous membranes slightly congested.

J. M. Sinclair* mentions the death of ostrich chicks, whose stomachs were found to contain the thorn-apple seeds. Ostrich poisoning by datura seeds is marked by staggering gait and spasmodic jerking of the neck, with unnatural contortions. Stupor and coma precede death (Walsh).

In Sullivan's case ½ pint of 1 per cent. potassium permanganate was given every three hours, followed by suitable doses of magnesium sulphate, and ordinary turpentine liniment was applied to the lumbar regions and extremities. There was recovery.

The thorn-apple contains a mixture of atropine, hyoscyamine, and a little hyoscine, which used to be considered a single substance, and called *daturine*. From the observations detailed it will be seen that datura operates similarly to atropine, producing paralysis, causing dilatation of the pupil, suspension of secretion, and of the inhibitory fibres of the vagus, leading to the rapid action of the heart.

Of the numerous exotic *Solanaceæ* which contain atropine, or the allied alkaloids, the *Mandragora officinalis*, or mandrake, contains *mandragorine*, which may be a mixture of the better known alkaloids, and African varieties of *Scopolia* appear to resemble hyoscyamine in effects.

Solanum.

Potato.—The unripe and green potato contain dangerous quantities of solanin, and old and rotten or sprouting tubers, which have been kept for a long time, are also dangerous; moreover, the alkaloid is most abundant in the 'eyes' and in the skin. Since, however, the potato is usually boiled, in which case the alkaloid passes into the water, poisoning in the human species is rare.

Symptoms.—Potato poisoning amongst animals occurs when green, old, or damaged tubers are fed in long-continued and large quantities. After about a week of such feeding the *horse*, as in a case noted by C. G. Saunders,† shows a small and weak pulse, normal temperature, and loss of

* *Vet. Record*, 1898, p. 367. † *Vet. Jl.*, 1907, p. 699.

co-ordination in movements; complete loss of appetite, excessive thirst, but inability to drink; mydriasis, stertorous breathing, suspension of peristalsis, and slight muscular tremors over the crural muscles.

In cases of horse poisoning encountered by G. T. Willows,* a very rapid and feeble pulse, temperature 103° F., intense congestion of the mucous membranes, and very fœtid diarrhœa, were observed, the cases terminating fatally. With *pigs* fed on steamed potatoes which were budding, and which had the buds on, Schneider† observed after a few days loss of appetite, dulness, exhaustion, imperceptible pulse, watery diarrhœa, low temperature, and comatose condition. According to Cornevin, when animals are fed with raw and entire potatoes there is depression, loss of appetite, cessation of lactation, gritting of the teeth, and profound prostration, with a remarkable somnolence, but no dilatation of the pupils. After a period of constipation there succeeds diarrhœa and, when possible, vomition. In the less acute forms the prostration is the dominant characteristic, to which is added the intestinal irritation, with rapid loss of flesh.

Post-Mortem Appearances.—The post-mortem lesions are those of acute or chronic enteritis according to the course of the poisoning. The other viscera are not abnormal, but there is congestion of the cerebral membranes.

Treatment.—In the potato poisoning of horses Saunders (*loc. cit.*) gave 1 grain of strychnine subcutaneously, and rectal injections of warm water. Next day there was purgation, and ½ grain of arecoline bromide speedily caused profuse salivation and sweating. Offensive black fæces were expelled. Eventually the horses recovered.

Some of the pigs treated by Schneider recovered after tannin and linseed tea.

Bitter-Sweet.—*Solanum dulcamara* (Fig. 35), or bitter-sweet: Stem shrubby at the base, with climbing or straggling branches, often many feet in length, but dying far back in winter. Leaves stalked, ovate or ovate-lanceolate, 2 or 3 inches long, usually broadly cordate at the base and entire,

* Private communication. † *Vet. Record*, 1902, p. 3.

but sometimes with an additional smaller lobe or segment on each side, either quite glabrous or downy on both sides, as well as the stem. Flowers rather small, blue, with yellow

Fig. 35.—Solanum dulcamara (Bitter-Sweet).
(From Smith's 'Veterinary Hygiene.')

anthers, in loose cymes, on lateral peduncles shorter than the leaves. Berries small, globular or ovoid, and red.

Symptoms.—W. Graham Gillam * observed in sheep

* *Vet. Record*, 1906, p. 88.

small intermittent pulse, temperature 104° F., quickened respiration, dilated pupil, staggering gait, and greenish diarrhœa.

On **Post-Mortem** the blood was dark and tarry, the ventricles firmly contracted.

The bitter-sweet probably contains a second alkaloid, which has been called *dulcamarine*, to which is attributed the mydriatic effect which is not characteristic of solanin.

Cases of poisoning by the other species, *Solanum nigrum*, are very rare, and would probably present the same general features. *S. triflorum* has been reported as poisoning cattle in Nebraska.

Chemical Diagnosis.—In seeking for organic poisons solanidine passes from an alkaline solution into ether. Solanin behaves like morphine. Solanin colours orange, passing on warming to violet, and red-brown on warming with strong sulphuric acid, Erdmann's or Fröhde's reagents. A characteristic test consists in warming with a few drops of a solution of selenic (or selenious) acid in sulphuric acid (6 of acid to 8 of water by volume). A pale red colour is produced, and on cooling and standing a beautiful red is developed.

Nicotine.

Occurrence.—The tobacco plant, *Nicotiana Tabacum*, represents the third group of the poisonous *Solanaceæ*, very rarely cultivated in this country, but extensively used for smoking, and owing its activity to the very poisonous volatile alkaloid *nicotine*, which is contained to the extent of 5 to 7 per cent. of the dry leaf. Infusions of tobacco are sometimes used as constituents of parasiticides for external application, and also for spraying trees. Most of the accidents attributed to tobacco are due to the use of lotions for parasites, for nicotine is readily absorbed through wounds, though not through the whole skin, and the poison may also be licked off the skin. *N. glauca*, or wild tobacco, is responsible for poisoning in South Africa.

Toxic Doses.—According to Finlay Dun, poisoning is caused by tobacco-leaf in horses by 9 ounces; cattle, 1 pound; sheep, 1 ounce; dog, 1 to 2 drachms. Of nicotine 5 to 6 minims is poisonous to the horse and ox, 1 to 3 minims for the dog, and subcutaneously about $\frac{1}{10}$ part of these doses (Kaufmann).

Symptoms.—A warm solution of about 5 per cent. strong tobacco-juice content was applied to about one-third of the shaved body surface of mangy *horses*.* After a few minutes the horses which had scratches on their bodies showed profuse sweating, tremors, nausea, disturbed respiration, head extended, and dilated nostrils.

In another similar case of two colts,† one died within an hour. After five hours the second animal was stretched on the straw, ears cold, nose dry, pulse imperceptible, respiration very slow, and deep coma. Under strong coffee there was recovery.

G. H. Livesey ‡ describes the nicotine poisoning of a *fox terrier* to which tobacco had been given as a cure for worms. The vomit contained a lump of about ½ ounce of tobacco. There were clonic muscular spasms, eyes retracted within orbits, mouth moist, mucous membranes rather pale, with bluish tinge; quick full pulse, cold extremities, and deglutition impossible. Potassium bromide was given; the animal showed signs of collapse, the body was cold, respiration shallow, heart slow and feeble, legs limp and paralysed, occasionally convulsed, and the muscles of the right shoulder showed persistent tremor. Strychnine gave a good reaction, the convulsions ceased after five hours, and as soon as possible dilute alcohol was given. It vomited again. The vomit was stained brown, smelling of tobacco, and mixed with a small amount of blood; on the next day it was well.

Post-Mortem Appearances.—There will be observed inflammatory patches in the digestive tube; ecchymoses of the lung and left valves of the heart; injection of the

* Abstract, *Vet. Record*, 1906, p. 68. † *Ibid.*
‡ *Jl. Comp. Path.*, 1904, p. 359.

nervous centres; flesh pale, capillaries empty, the great vessels and right heart filled with black blood.

Chemical Diagnosis. — Nicotine, being volatile, is separated by distillation in a current of steam from the organic material made alkaline by caustic alkali. The distillate then contains the oily, pungent, and characteristic nicotine. In the absence of definite chemical reactions physiological tests should be made.

When the material is in sufficient quantity, it is very desirable to perform comparative tests to decide between nicotine and conine. An ether solution of nicotine, mixed with an ether solution of iodine, gives a precipitate, or, if there is but a trace of nicotine, a turbidity, and gradually long red crystals form, which reflect blue (Roussin's test). Conine does not give this reaction. Another distinction depends on the fact that a cold saturated (1 to 90 of water) solution of nicotine remains clear on warming, whereas a similar solution of conine becomes turbid, since conine is less soluble in warm than in cold water. Nicotine hydrochloride first separates as a resin, slowly becoming crystalline, whilst conine gives at once a crystalline hydrochloride.

SCROPHULARINEÆ.

This family contains many poisonous *genera*, of which those found in Britain and on the Continent are *Digitalis*, *Scrophularia*, *Pedicularis*, *Rhinanthus*, *Melampyrum*, *Verbascum*, and *Linaria*, whilst *Gratiola* is native to South-Eastern Europe. Those members which have been definitely studied have been found to contain glucosides of the digitalis and saponin classes.

Digitalis.

Botanical Characters.—The *Digitalis purpurea* (Fig. 36), common foxglove, finger flower, or dead men's bells, is a very common weed, often cultivated as a garden plant. It frequents dry, hilly wastes, roadsides, and banks. Being

Fig. 36.—Digitalis purpurea (Foxglove).

widely distributed and well known, a particular description is unnecessary in this place. In the United States the plant is cultivated, and is naturalized to some extent on Cape Breton Island.

Active Principles.—All parts of the fox-glove contain the digitalis glucosides, the second year's leaves being richer than the first. Neither boiling nor drying deprives the plant of its activity. At least four constituents have been characterised as components of digitalis. These are: (1) *Digitalin*, insoluble in water, soluble in alcohol, acting as an irritant and heart poison. It is prepared from the seeds. (2) *Digitoxin*, having similar solubilities, is the most active heart poison, and forms from 0·25 to 0·3 per cent. of the wild plant. (3) *Digitaleïn*, soluble in water and alcohol, has similar effects. (4) *Digitonin*, sparingly soluble in all solvents, is a colloidal glucoside resembling saponin, and is not a heart poison.

Tinctures and extracts of digitalis vary in their composition; thus, French digitalis made from the leaves contains chiefly digitoxin, and German digitalis, from the seeds, owes its activity chiefly to digitalin, but also contains about 50 per cent. of digitonin. Pure digitalin is known as *digitalinum verum*.

Toxic Doses.—Cornevin gives the toxic amounts of the green leaves as—

4–5 ounces for the horse.	1 ounce for the sheep.
6–7 ,, ,, ox.	$\frac{1}{2}$–$\frac{3}{4}$,, ,, pig.

Kaufmann gives 1 ounce of the powdered leaf as poisonous to the horse, 80 to 130 grains to the dog, and 30 grains to the cat. Of digitalin $2\frac{1}{2}$ grains for the horse, $\frac{1}{3}$ grain for the dog, and $\frac{1}{6}$ grain for the cat.

Effects.—Apart from the irritant effects, the chief action of digitalis is upon the heart, causing increased and prolonged systole, with diminished and shortened diastole. In poisoning the heart is arrested in systole. The drug is a powerful diuretic, and owed its introduction into therapeutics to this effect. Digitalis is a cumulative poison,

possibly on account of slow elimination, as with strychnine. It may thus produce chronic poisoning, and is contra-indicated in cases of kidney disease. With medicinal doses the pulse is slower, fuller, and more regular, but in poisoning it is rapid, weak, and irregular.

Symptoms.—Finlay Dun * gives a careful account of the experimental poisoning of a three-year old brown mare, which had received in all 9 drachms of powdered digitalis over a period of three days. Towards evening of the third day the mare showed dulness and loss of appetite. On the fourth day she was nauseated; nose, mouth, and ears cold; abdomen tympanitic, with colicky pain, and occasional pawing; pupil somewhat contracted; pulse firm at axilla and heart, but not very perceptible at jaw. At 4.30 p.m. she was down, much pained, and attempting to roll; pulse 82, but unequal. On the following day at 12 noon, pulse, imperceptible at jaw, about 120; respiration 25, and very much laboured; lips retracted, and saliva dripping from the mouth; enormous abdominal tympanites, and much pain; rapid sinking; died next day at 11 a.m.

Two other animals, which had received similar doses, recovered, not having displayed marked cardiac symptoms.

In a case mentioned by Graham Gillam [1] two *cows* and a *horse*, after eating hay containing dry foxglove, were observed to feed erratically, breathe hard, and lie down after feeding. Pulse almost imperceptible, contracted pupil, and excessive urination.

Horses [3] which had foxglove by mistake, showed sleepiness, swollen eyelids, dilated pupils, injected conjunctivæ, considerable swelling in submaxillary space, respiration normal, temperature 103·5° F., pulse full, between 65 and 75, most intermittent, being occasionally normal; the second heart sound was frequently obliterated. On the next day laboured breathing, head immensely swollen, tongue greatly enlarged and protruding, pulse 80, and most erratic, temperature very slightly up, great restlessness. The respiration became more difficult and ster-

* 'Veterinary Medicines,' 1910, p. 539.

torous, tongue and buccal membranes livid, jugular standing out. Tracheotomy failed to save.

Other horses showed less aggravated symptoms, and nearly all recovered under treatment.

In a case of the poisoning of *pigs* [4] the cause was the pouring by a servant of decoction of digitalis leaves into the pigs' bucket, whereby five animals were affected, and two died. They were languid and sleepy, refusing to eat or drink, attempting to vomit, and repeatedly passing small quantities of fæces. Urination was scanty and strained. In the case of those which recovered, the effects did not pass off for more than a week.

Post-Mortem Appearances.—The pigs above referred to showed acute inflammation of the mucosa of the stomach and intestines, with thin yellow contents. The kidneys were slightly congested, bladder empty, and other viscera healthy. These appearances seem to point to a predominating saponin-like effect, due to digitonin.

In Finlay Dun's case the stomach was ruptured, but there was no inflammation of the alimentary membranes.

In digitalis poisoning, as a rule, the abdominal viscera are healthy. The lungs are engorged with dark venous blood, and the heart shows great distension of the auricles, so that the transverse may be greater than the longitudinal section.

Treatment. — The treatment consists of purgatives, mucilaginous draughts, and stimulants. There is no specific antidote, but atropine may be given to counteract the irregular heart action.

Chemical Diagnosis.—Digitalis constituents are best sought for in ingesta, vomit, and the like. They are yielded to solvents in the extraction of the acid liquid in systematic work, on Dragendorff's principle. The best means of detection is given by the Kiliani-Keller test for digitoxin. A trace of the substance is dissolved in 3 or 4 c.c. of strong acetic acid, containing ferric iron (100 c.c. of acetic acid to 1 c.c. of 5 per cent. ferric sulphate), and a layer of strong sulphuric acid (also containing iron) is poured beneath the

acetic acid solution. A dark colour zone forms at the junction of the liquids, and in about two minutes a blue colour ring, whilst after about thirty minutes the whole of the upper (acetic) layer has a deep indigo blue colour, gradually passing to blue-green. Various tannins, especially from the quinine barks, give a similar reaction, as also does formaldehyde. A physiological test on a frog is therefore needful in a case of doubt, or for strict medico-legal purposes.

REFERENCES TO DIGITALIS.

[1] W. Graham Gillam, *Vet. Record*, 1906, p. 88.
[2] Damman and Behrens, *Vet. Jl.*, 1903, p. 78.*
[3] W. Pauer, *Vet. Record*, 1896, p. 598.
[4] H. Olver, *Veterinarian*, 1872, p. 173.

Verbascum, Scrophularia, Gratiola, and Linaria.

These genera do not contain plants which give rise to serious poisoning, and, moreover, have not received precise study. A brief mention will therefore suffice.

Verbascum.—*Verbascum Thapsus*, or great mullein, is a common roadside weed, extending as far north as Aberdeen. The leaves and flowers afford an emollient and expectorant extract, which is used as a medicine. The seeds are stated to be narcotic, but animals refuse to eat the plant (Cornevin).

Scrophularia.—*Scrophularia nodosa*, or figwort, and *S. aquatica*, or water scrophularia. The former occurs in woods, the latter in marsh or moist situations, and has a disagreeable odour. According to Walz, they contain bitter principles, *scrophularin* and *scrophulerin* respectively, which may cause fatal superpurgation. But the plants are not eaten by animals.

Gratiola.—*Gratiola officinalis*, or hedge hyssop, is found in Europe, and in the Southern United States. It owes its

* This is a remarkable case, in which three out of eight sheep died after eating garden clippings, including *Datura Stramonium*, *Hyoscyamus albus*, and *Digitalis purpurea*.

activity to *gratiolin*, a drastic purgative. Poisoning, which might possibly result from it, takes the form of super-purgation.

Linaria.—*Linaria vulgaris*, or toad-flax, *L. spuria*, or round-leaved linaria, *L. cymbalaria*, or ivy linaria, and *L. elatina*, or pointed linaria, are all acrid and poisonous, and have a repulsive smell and nauseous taste. Animals do not readily eat the plants, and poisoning, therefore, is very rare. Toad-flax is common in hedges and field borders, the others less frequent, and they ought to be included as noxious weeds.

Pedicularis, Rhinanthus, and Melampyrum.

Pedicularis.—This genus includes *Pedicularis palustris*, or red rattle, distributed over marshes, wet meadows and ditches, and *P. sylvatica*, or louse-wort, more widely distributed over moist pastures, meadows, and heaths. Many species are found in the United States, chiefly the Western.

Rhinanthus. — *Rhinanthus Crista-galli*, the rattle, or common rhinanthus, is a meadow and pasture weed, which often causes injury to the herbage, and whose grains are liable to become mixed with cereals.

Melampyrum.—*Melampyrum arvense*, or purple cow-wheat, occurs in cornfields of South-Eastern England and Norfolk, and is injurious to crops. The grains, like those of rhinanthus, may become mixed with cereals.

These plants contain a glucoside, *rhinanthin*, or closely allied compounds, in their seeds, and thus may be deleterious components of forage or flour. The doses for animals would be very large, for Cornevin quotes experiments in which 35 grammes of the grain and over 2 pounds of the fresh plant did not affect rabbits.

The general effect of rhinanthin is that of a saponin, and reference may be made to that substance for an account of its action and chemical recognition in flour or meal.

PHYTOLACCACEÆ.

This order is not found in England, but the *Phytolacca decandra* is acclimatised in Southern Europe and Africa, but is native to America, where it is known as the poke-weed, garget, or American nightshade.

It contains a purgative active principle in all parts. Formerly the berries were used to colour wine, a practice which is, however, now generally prohibited on account of possible danger. The effects are those of superpurgation, the extract being stated to be quickly fatal to the dog, whilst in the States cattle have occasionally been fatally poisoned by the leaves.

POLYGONACEÆ.

Some species of this order are held to be responsible for poisoning, although the records, and our knowledge of the active principles are scarcely such as to give the basis of a well-founded narrative. They include species of *Rumex*, or dock, of which *R. Acetosa*, sorrel dock, and *R. Acetosella*, sheep-sorrel, may be mentioned. These plants are well known to contain oxalates, which may possibly account for the alleged poisonings. They are widely distributed in the north temperate hemisphere. Whilst it is held by some that sheep-sorrel improves the condition of sheep that eat it, others assert that the mature and seeded plant causes poisoning in sheep and horses.

According to Cornevin, in the *horse* the chief *symptoms* include at first a condition recalling drunkenness, marked by vacillating gait, salivation, and cyanosis. Then there are muscular tremors, dilatation of the pupil, relaxation of sphincters, urination, and a feeble, slow, and intermittent pulse. There succeed to these, convulsive contraction of the lips, retraction of the eyeball, accelerated and stertorous breathing, extreme dilatation of the nostrils, tetanic contractions of the muscles of the neck, back, and limbs, abundant sweating, and falling. After a period of extreme exhaustion,

these symptoms are repeated, and death occurs in convulsions. The *lesions* are not characteristic, the right part of the stomach being inflamed.

The same authority describes remarkable effects caused by *Polygonum fagopyrum*, or buckwheat, the grains of which are used as food. The flowers appear to be the more dangerous part, and have caused fatal poisoning of the ox, sheep, pig, and rabbit. The effects appear to be due to a remarkable congestion, leading to redness and tumefaction, especially of the ears and head, but noticed also in other parts. This leads to great agitation, and sheep precipitate themselves against the wall. By affecting the nervous centres the congestion may prove fatal. The course of the poisoning, by its symptoms of hallucination and intoxication, recalls the action of *Cannabis indica* (Cornevin).

ARISTOLOCHIACEÆ.

The genus *Aristolochia* is mainly tropical, although some species are cultivated in gardens—*e.g.*, *A. Clematitis*, which is the chief poisonous example, although all the species should be regarded with suspicion; and Cornevin specially names of them *A. rotunda*, *A. longa*, *A. pistolochia*, and the American *A. Sipho*.

The poisonous effects are due to the alkaloid *aristolochine*, which has similar but more powerful effects than aloïn (of aloes). In rabbits it causes acute neurotic nephritis, with albuminuria and uræmic symptoms, and in dogs marked fall of blood-pressure, intestinal hæmorrhages, but no nephritis (Cushny).

Cornevin relates a case of the poisoning of *horses* by a ration containing rather more than 1 part of *A. Clematitis* to 7 of lucerne. There were noted immobility, torpor, and a drunken condition, with unsteady gait; pulse ample, quick and hard; periods of comatose somnolence, marked by slight spasms and convulsions; pupils dilated and vision obscured; loss of appetite, constipation, frequent urination, and genital spasm. Recovery was slow.

THYMELACEÆ.

This family is represented in Britain only by the genus *Daphne*, also found in the Cape and Australia, of which

FIG. 37.—DAPHNE MEZEREUM (COMMON MEZEREON).

Daphne mezereum, common mezereon or spurge-flax, and *D. laureola*, spurge-laurel or dwarf bay, grow wild. Of

the dangerous exotic species *D. cneorum* and *D. pontica* (from Asia Minor) are cultivated.

Botanical Characters.—Mezereon (Fig. 37) is a shrub

FIG. 38.—DAPHNE LAUREOLA.

attaining about 3 feet, cultivated in gardens, but found wild in woods, particularly in the South. The narrow lanceolate leaves form tufts at the ends of the branches, and the

small purple-scented flowers appear in February before the leaves are fully out. The berry is red.

The spurge-laurel (Fig. 38) attains about 4 feet, and has clusters of evergreen lanceolate leaves crowded at the ends of the branches. The flowers are greenish-white and scentless, and the berries bluish-black.

Active Principle.—The daphnes contain an acrid substance, *mezerinic acid*, in all parts, particularly the bark and berries. It is not destroyed by drying. Very little is known as to the chemistry and properties of mezerinic acid. The physiological effect is that of a powerful drastic, also exhibiting marked nervous effects.

The plants are very poisonous. Twelve berries of mezereon have been stated to kill the human subject, and about 1 ounce of the spurge-laurel is stated to prove fatal to the horse.

A case of poisoning of a horse by spurge-laurel (locally known as wood-laurel) was investigated by the author (1911) for H. D. Sparrow, of Rochford, Essex. From Sparrow's information it appears that the leaves are frequently dried, rubbed up in the hands, and given to horses for worms. In a case observed by him such administration preceded attacks of gripes. Several mysterious cases of colic encountered in the district would appear to have been caused by this practice.

Symptoms.—Daphne poisoning is marked by intense colic. As an immediate effect Sparrow noted constipation, lasting in one instance forty-eight hours, in spite of aloes, oil, and 1 grain eserine and 2 grains pilocarpine intravenously. There follow dysentery and copious evacuations of fæces streaked with mucus, blood, and intestinal epithelium. Between the spasms the animal is drowsy.

A similar case came under observation (1912) through C. F. Parsons, of Cheltenham, in the case of a seven-year-old cart-horse. The horse ate the shrub whilst waiting to be unloaded, and refused the evening meal of the same day. On the next day there was abdominal pain, staggering gait, anxious countenance, laboured breathing, pulse 80, tem-

perature 103·2° F., bowels normal. On the following day there was excessive purgation, pulse 120, temperature 104·2° F., and death occurred at midday.

On **Post-Mortem** the stomach and intestines are inflamed and all the ingesta fluid, and Sparrow also found the colon very much inflamed, the walls 1½ inches thick, and contents loose, blood-stained, and of a peculiar odour. There was no tympanites until after death.

The **Treatment** consists in elimination of the cause as with irritants in general, and treatment of the particular symptoms as indicated.

The detection of poisoning by analysis is very uncertain in the present state of our knowledge. The finding and identification of plant fragments offers the most certain means. When the bark of the plant is chewed there is produced after some minutes a very intense burning sensation, which lasts several hours. Extraction in the ordinary systematic routine yields an acid, which has no burning taste. It gives a smoky colour with ferric chloride, and a pink colour on prolonged warming with strong sulphuric acid. These observations are got both with the plant and with ingesta, but are scarcely characteristic. It is curious to note that after extraction of the acid, even in the cold, the toxicity, as tested on mice, so far as present observations go, disappears.

EUPHORBIACEÆ.

There are three genera of this family found in Britain, and from which poisoning may occur—namely, *Euphorbia*, or spurge, *Mercurialis*, and *Buxus*, or box.

Of exotic *Euphorbiaceæ*, *Ricinus communis*, or castor oil; *Croton tiglium*, or croton; and *Jatropha curcas*, or purging nut, are important species.

Euphorbia.

Botanical Characters.—The chief species which may give rise to poisoning is the *Euphorbia lathyris*, but no

doubt most, if not all, of the *euphorbiæ* have similar effects. The *E. hibernica*, or Irish spurge, is, for instance, used as a fish poison.

E. lathyris.—A tall, stout annual or biennial, often 3 feet high, or even more; very smooth and glaucous. Stem-leaves narrow-oblong, the upper ones broader, especially at the base, often 3 or 4 inches long, and all opposite, not alternate, as in other *euphorbiæ*. Umbels of three or four long rays, once or twice forked, with large ovate-lanceolate floral leaves. Glands of the involucre crescent-shaped, the points short and blunt; capsules large and smooth; seeds wrinkled. The plant grows wild in Sussex and Somerset, and is cultivated in cottage gardens.

Toxic Principle.—The *euphorbiæ*, of which *E. lathyris* is selected as typical, are distinguished by containing an acrid juice, and in the seeds a purgative oil. The resin *euphorbin* from the North African *E. resinifera* is a non-officinal purgative. Very little is known of the chemistry of the juices, which on the whole rather recall *ranunculus* in their action. Desiccation does not deprive the plant of its activity.

Symptoms.—Euphorbia is distinguished by its irritant action, with production of vomition, when possible, purgation, and in fatal doses superpurgation, together with nervous symptoms of vertigo, delirium, and muscular tremors.

The **Post-Mortem** appearances are those of acute gastro-enteritis.

Piss-grass.

The South African *Euphorbia genistoides*, known as piss-goed, or piss-grass, is a low shrub of about 8 inches, having close, many-branched stems, resembling a besom, and green apetalous flowers (Walsh). Like many others of this order, the stem contains an acrid juice, having powerful irritant properties.

Poisoning. — Poisoning by piss-grass mainly affects hamels, oxen, and geldings, probably by reason of the narrower urinary passage. It is marked by severe urethritis. The animal appears very uneasy, and attempts to

urinate are frequent and painful. The animal lies down, the bladder becoming more distended, and dies in coma, or in a violent effort at relief.

The *treatment* is difficult save in the early stages. Epsom salt and three-hourly doses of 10 grains of extract of belladonna have been recommended (Hutcheon). In the horse the catheter may be used, and for the ox amputation of the penis is a possible measure. Turpentine or any diuretic agent is to be avoided.

Mercurialis.

Both *M. perennis* and *M. annua* are poisonous, and the former is here described as typical.

Botanical Characters.—*M. perennis* (Fig. 39), or dog's mercury. Rootstock slender and creeping. Stems erect, simple, 6 or 8 inches, or rarely nearly 1 foot high. Leaves rather crowded in the upper half, oblong or ovate-lanceolate, 2 to 4 or 5 inches long, usually pointed, crenate or serrated, and rough or with short hairs. Flowers diœcious, on slender axillary peduncles, often nearly as long as the leaves; the males in little clusters, the females singly or two together. Ovaries larger than the perianth, with rather long, spreading styles. Capsules more or less covered with warts or soft prickles.

The plant is common in England and Scotland; less so in Ireland.

Toxic Principle.—*Mercurialis* has been found to contain a volatile basic oil having narcotic properties, which has been called *mercurialine*, and to which, in part at any rate, the toxicity of the plant is due.

Symptoms.—Mercurialis acts on the alimentary and urinary systems. In a case observed by C. Blackhurst* of the poisoning of cows, there was excessive bloody purgation, cessation of lactation, temperature 105° F., pulse 90, and increased respiration. The disease was protracted over several weeks, the animals being comatose after the first

* *Vet. Jl.*, 1896, p. 431.

symptoms, and eventually the neck assumed a curved position, like the half of the figure eight, and the animal was unable to raise its head from the ground.

Fig. 39.—Mercurialis perennis (Dog's Mercury).

Post-Mortem Appearances.—The thoracic visera were normal; on dissecting the neck the muscles of the right-hand side were rich in fibrous tissue, and the last three

joints partially anchylosed; the abdominal viscera showed inflammation with sloughing mucuses; the liver and kidneys showed fatty degeneration.

Cornevin notes, further, as a symptom of the poisoning, hæmaturia, with frequent, painful micturition, and passage of dark-coloured bloody urine.

Buxus.

Buxus sempervirens, the common and well-known evergreen shrub, the box, is found wild in parts of central and southern England, but is much used as an ornamental border in gardens. From this circumstance box may give rise to poisoning, but cases are rare.

The plant contains an alkaloid, called *buxine*, and a resin and essential oil. The action of the plant, probably due to the latter constituents, is emeto-purgative. In fatal doses there are intense abdominal pain, dysentery, convulsions, and death by asphyxia. The *lesions* shown are extended gastro-intestinal irritation, and pulmonary congestion.

Other Euphorbiaceæ.

Ricinus communis.—The castor-oil plant is an important exotic, yielding the well-known seeds from which castor oil is obtained by pressure. The *seeds* or *beans* of castor oil are oval, and about $\frac{2}{3}$ by $\frac{1}{3}$ inch in size. They have a brownish or buff colour, and are mottled or marbled with brownish specks and streaks.

The seed is rich in oil, of which it contains 50 per cent.

The residue left after pressure contains the active poison, which does not pass into the oil. Accidents may arise either from the giving of the beans in other food, or from the feeding of the residual press-cake, which is otherwise valuable as a manure.

Toxic Principle.—The effects of castor-oil bean are due to *ricine*, a typical example of the class formerly known as *toxalbumins*, now better designated as *toxines*, in the present instance *phyto-toxines*, being of vegetable origin.

In distinction to the majority of toxines—*cf.* snake venom and bacterial toxines—ricine, like *crotine*, from *Croton tiglium*, is absorbed from the alimentary tract, probably by reason of its resistance to decomposition by digestive enzymes. But the poisonous dose by the mouth is at least one hundred times as great as that by injection. The toxicity of ricine is unknown and enormous. Impure preparations may be purified by the action of trypsin, which destroys accompanying albumins, the weight of ricine decreasing without sensible diminution of toxicity.* Like other toxines and ferments, ricine and its allies, crotine and abrine (from *Abrus precatorius*), is destroyed by prolonged heating over 60° C. in the moist condition, but is more resistant to dry heat.

The formation of ricine immunity is characteristic. By increasing doses an animal may be made tolerant of enormous normal overdoses of such magnitude as from 400 to 800 times, the tolerance not being due to habituation of the tissues, but to the formation of a true *anti-ricine* in the serum. The anti-body is capable of conferring immunity on a second subject, and is specific against ricine, but not against other toxines.

The seeds of *Croton tiglium* resemble those of *ricinus* in size and shape, but are dull brown in colour, and not mottled.

Those of *Jatropha curcas*, curcas purgans, or American, Barbadoes, or purging nut, are not likely to cause poisoning in Great Britain.

Both *croton* and *jatropha* yield oils, and press residues, and both are very dangerous. The mechanism of croton poisoning is now known to be due to the toxine crotine, whilst jatropha, according to Stillmark, contains ricine. With croton is seems probable that in part the toxine passes into the expressed oil.

Symptoms.—Poisoning by ingestion of castor-oil beans, or residues, does not declare itself, as a rule, till after several days, and does not appear to be always marked by purga-

* See Oppenheimer, 'Toxines and Antitoxines,' 1904, p. 165.

tion, although this is more usual, the beans being considerably more active than the oil. In a case observed in the *horse*,* there was complete loss of appetite, shivering, coldness of extremities, dejection, abdominal pain, and constipation. Temperature 103° F., pulse 70, and death in about three days.

A case is noted by Chambers,† in which death occurred in *cattle* after external application of castor oil and lubricating oil against ticks. It probably does not illustrate ricine poisoning, but displays the possible ill-effect of large quantities of the oil.

The **Post-Mortem Appearances** after ingestion of the beans are those of intense gastro-enteritis.

Amongst the *Leguminosæ* the seed of *Abrus precatorius*, the crab's eye, or jequirity pea, contains the toxine *abrine*, and the leaves and bark of the American *Robinia pseudacacia*, or locust-tree, contain the toxine *robine*. The seed of *abrus* is about the size of a small pea, bright red in colour, and has a black spot. Abrine is less toxic than ricine, but has similar effects—indeed, Ehrlich only clearly differentiated the two toxines from the fact that anti-ricine is powerless against abrine, and similarly anti-abrine is of no avail against ricine.

It exercises a very powerful irritant action on the conjunctivæ, and has on this account found application in ophthalmic therapy.

The natives of India practice malicious poisoning by inserting splinters impregnated with abrus under the skins of beasts. This malpractice was detected by Calmette by means of the specific anti-abrine. The *Quarterly Journal of Veterinary Science in India*, 1883, p. 375, gives an account of such a poisoning, in which a spike 1½ inches long and ¼ inch at the thick end was inserted under the skin. It had a dark green colour, weighed 12 grains, and contained datura opium, abrus (or gunchi) seeds, onion, and spirit daru.

* Broad, *Vet. Record*, 1896, p. 226.
† *Vet. Jl.*, 1910, p. 717.

Chemical Diagnosis.—The vegetable toxines ricine, crotine, and abrine, all possess the power of agglutinating red blood corpuscles. In examining a cake or meal suspected to contain castor seeds the powdered material (about 2 grammes) is extracted by means of physiological saline (0·9 per cent. solution of common salt) in the incubator at 37° C. for a few hours, and the solution filtered. The clear solution is then added to a suspension of red corpuscles from fresh defibrinated, or citrated, blood suitably diluted (about ten times) with salt solution. More or less rapidly, according to the quantity of ricine present, the corpuscles clump together, or agglutinate, and fall to the bottom of the tube in the form of red flakes. With very small quantities of material the phenomenon may be observed in a hanging drop under the microscope.

An alternative test consists in making a layer of a small quantity of the suspected extract on the top of some serum from an animal immunised to ricine. With ricine such a serum forms a zone of amorphous precipitate (precipitin reaction).

PLANTS AND FOODS REPUTED TO BE POISONOUS.

Some plants and vegetable foods are popularly spoken of as poisonous, although no definite and specific toxic principle can be associated with their action. In this section mention will be made of the important substances which fall within this category, and it may be remarked at the outset that many of these so-called cases of poisoning prove, on scrutiny, to be cases of injury or death due to errors in diet, the bad preparation or condition of the ration, or the greed of the subject. As to the last point, it ought always to be remembered that animals, especially in periods of scarcity of green food, or after winter feeding, eat freely of any green plant they may encounter.

Cotton-Seed Cake, or Meal.—Undecorticated cotton cake is popularly described as poisonous. It has, in fact,

been long known that it is very harmful, and may produce dangerous illness and death. The husks of the cotton seed contain a large proportion of indigestible fibre (24 to 25 per cent., as against 5 to 6 per cent. in the decorticated).

The first observations on undecorticated cotton cake appear to be those made by A. Voelcker in 1859,* who examined a cake containing about 50 per cent. of husks. The case is typical as regards post-mortem observations, for the paunch was distended with food impacted like hard dough; lower stomachs empty, and the duodenum blocked by 72 pounds of comminuted and densely impacted husks. Stoppage due to balling or impaction of the rough fibrous husks is the clear cause of so-called cotton-cake poisoning.

That there is no specific poisonous principle is shown by the negative results attending chemical research for such matters, and by the numerous cases in which a judicious feeding test of a suspected cake has, in our experience, eliminated all question of poison.

In the *Veterinary Record* of 1909, p. 630, is an abstract from the German of the effects of cotton-seed meal, of which draught oxen had 2 pounds each. There were œdematous swellings at the extremities; unimpaired appetite; later, weakness of hind quarters, and, in a few cases, disturbances of equilibrium. Four out of fifteen became blind, the eyeball protruding, and the pupil abnormally enlarged. Laxatives and change of diet led to recovery.

It is interesting to record that Professor Macqueen and the writer investigated a somewhat similar case in which blindness was attributed to linseed cake, but no positive evidence in support of this view was got.

It should also be pointed out that cotton, as other cakes, may be contaminated—*e.g.*, with metals or castor beans—and in such instances would prove harmful by reason of those impurities.

The **Soya Bean** cake and meal, on their recent introduction, were often, in our experience, held to be poisonous. Feeding tests were invariably negative, the beans are not

* *Veterinarian*, 1859, p. 327.

cyanogenetic (like Java beans), and, so far as we at present know, no specific poison has ever been isolated therefrom.

Brewer's Grains and Distiller's Grains, the residues of the mashing in beer and spirit fabrication, are good feeds in themselves, but sometimes held responsible for 'poisoning.' But grains are liable to fermentation, and also to acidity, and consequently ought not to be too freely fed, and ought to be mixed with straw chaff; otherwise, digestive trouble and tympanites may result. The Belgian and French writers, further, describe drunkenness in stock by use of fermenting grains, which is alarming and sometimes dangerous. In the interests of hygiene it may be stated that grains should be free of alcohol, and of no, or very slight, acidity.

The addition of much common salt in order to preserve such foods as distillery sludge makes them improper, and possibly harmful, foods for pigs.

On the Continent somewhat similar considerations hold with regard to **beet pulp residues** from sugar fabrication. As regards the effects of sugary foods—molasses, molasses mixtures, and dry slices in which sugar is contained, sometimes to the extent of more than 30 per cent., attention may profitably be directed to their values as foods, regarding which reference should be made to the recent observations of Goodwin.*

The nuts of the **Beech**, *Fagus sylvatica*, are not likely to cause trouble in Great Britain. Cornevin states that the leaves are harmless, that the oil and decorticated press residue of the nuts are also harmless, but that cake of undecorticated press residue causes poisoning, recalling that of *lolium*. The cause is unknown; it may be due to a toxic principle like that of *lolium*, but the question is at present unsettled.

Acorn Disease.

The remarkable disorder known as acorn poisoning, or acorn disease, has attracted much attention in Great Britain, and offers some features of interest. It does not

* *Journal of Board of Agriculture*, 1911, p. 97.

affect sheep, deer, or swine, but only cattle. It may possibly be that the former animals reject the husks, but this does not seem an entirely adequate explanation.

The subject was first investigated in a satisfactory manner by Simmonds and Brown,* and the features of the disease are clearly defined in their articles of 1884, reprinted from the *Veterinarian* of 1871.

Symptoms.—Acorn disease sometimes does not declare itself until after all the ingested acorns have been digested and expelled. It is distinguished by constipation in the earlier stages, followed by persistent diarrhœa, with frequent small, dark, and bloody evacuations. There is loss of appetite, suspension of rumination, wasting, colic, and excessive urination, large quantities of pale urine being voided. There is no fever, and the temperature may be subnormal. Soreness of the mouth, pallor of membranes, and a discharge from the nose and eyes, which are sunken, are noticed.

The **Lesions** are generally those of an irritant poison, but the rumen and reticulum are usually healthy; no acorns may be found in the alimentary tract; the kidneys are pale, and bladder distended with colourless urine.

The **Treatment** of mild cases may be effected by oleaginous purgatives (ol. lini, O.i.) and opiates (opii aqua, ℥i.), and a pint each of linseed tea and oatmeal gruel, with ℥iv. opii aqua, and ℥iv. soda bicarbonate three times daily.†

Cornevin signalises poisoning by young **Oak-Leaves** as occurring on the Continent, but no records of such poisoning in this country are prominent.

The general features of **Oak-Leaf Poisoning** are not unlike those of acorn: Gradual wasting, loss of appetite, cessation of rumination, and lactation, and constipation, which may eventuate in fatal dysentery. But Cornevin observes fever and nervous symptoms of collapse, and, in particular, very dark urine, varying from red to deep wine-

* *Veterinarian*, 1884, pp. 25, 156, 385.
† Harrison, *Vet. Record*, 1893, p. 266.

red, a point which appears to sharply distinguish the otherwise nearly parallel cases.

Besides *gastro-enteritis*, there is *acute nephritis*, the kidneys being greatly enlarged, and the urine dark, albuminous, free of sugar, and bloody.

Causation.—The only known agent of poisoning by acorn, oak and beech is *tannin*, whose general action is that of an astringent. In overdoses it proves dangerous, probably by—(1) gastric disturbances, due to precipitation of proteins; (2) astringency, causing constipation; (3) dysentery, following on constipation. The immunity of certain animals to acorn, and the existence of a large number of different natural tannins, and the enormous doses required to induce dysentery, are obstacles in the way of this explanation. It has been suggested that specially active forms of tannin, or unrecognisable active principles, may be responsible, but positive evidence is lacking. It is noteworthy that the drinking of *tanning waste liquors* sometimes causes harm, probably from similar causes, and at present the commonly accepted explanation of the disorder, as one due to acute digestive derangement, is the best.

The *chemical recognition* of tannin in fluids is easy. The tannin may be separated from acid aqueous extracts, and recognised by the dark coloration with ferric chloride, and by the precipitation by tannin of gelatine and of alkaloids from solution.

The leaves, fruit, and twigs of **Hawthorn**, *Cratægus Oxyacantha*, are sometimes eaten by sheep, and may also cause death by gastric derangement. Usually there will be found very densely impacted masses of the plant on post-mortem examination.

Fern Poisoning.

The root of the **male fern** (*Aspidium filix-mas*) contains about 8 per cent. of *filicic acid* and also *filimarone*, a neutral substance. Filicic acid is a widely used and effective agent against tapeworm, and may act as a poison.

The powdered root is irritant and laxative, and in large doses causes hæmorrhagic gastro-enteritis, and nervous symptoms of drowsiness, occasionally convulsions, coma, and collapse (Finlay Dun). Blindness often follows large doses. The extract is prepared by means of ether, and Fröhner states that 5 drachms killed a dog of 40 pounds, 6 drachms a sheep of 88 pounds, and 3 ounces a cow of 660 pounds weight.

In the treatment of male-fern poisoning oils must be avoided. Castor oil greatly facilitates absorption, and according to Kobert in 57 per cent. of cases of poisoning this oil had been given. Evacuation of the stomach, mucilaginous medicines, and stimulants, are indicated as remedial measures.

Poisoning by **Bracken** (*Pteris aquilina*) is a so far obscure disorder, often observed in cattle in the early autumn (from August to November), after eating bracken. The plant is taken, although other green food may also be available.

The poisoning of *horses* after prolonged feeding on bracken along with other forage is mentioned by the German authorities,* and is stated to be marked by timidity, uncertain gait, loss of equilibrium, dilatation of the pupils, red, and later yellow, coloration of the conjunctivæ, and slowing of the pulse. Sometimes death occurs.

The poisoning is attributed by the Continental authorities to the effects of an acid (*pteritannic acid*), similar to, and possibly identical with, the filicic acid of male-fern. Both these acids are, indeed, derivatives of the polyhydric phenols (such as tannin, pyrogallol, and phloroglucinol). Such poisoning of the horse does not appear to have been observed in Britain, perhaps because the plant is rarely fed, though used freely as a litter.

The poisoning of *cattle* by bracken appears to have been first distinctly recognised in Britain in 1893, in which year D. M. Storrar† drew attention to the disorder, which he

* See Müller, *Landwirthschaftliche Giftlehre*, 1897, p. 29.
† *Jl. Comp. Path.*, 1893, p. 276.

held as probably due to a highly indigestible food, and not necessarily to a specific toxic effect of fern.

An editorial article * draws attention to important features of bracken disease, which further serve to distinguish it from anthrax. The main points relating to bracken disease are: The absence of bacilli in the fresh fluids and tissues; spleen quite normal; subpleural and subperitoneal hæmorrhage; considerable effusion of blood in the large intestine; a temperature of 106·8° to 108·4° F.; the disease lasts a few days; abundant bloody discharge from nose and rectum; occurs only in cattle.

Storrar † later noticed unusual symptoms of the disease in a bullock, consisting in effusion and hæmorrhage in the vicinity of the larynx, and the formation of numerous hæmorrhagic patches on the surface of the body, the cutaneous vessels being congested with black tarry-looking blood.

More recently bracken poisoning has been the subject of inquiry by the Board of Agriculture, and useful information is contained in the chief veterinary officer's reports of 1909 and 1910.

The **Symptoms** summarised in the 1909 report are: Temperature, 104° to 107° F.; loss of appetite; blood-tinged discharge from the mouth and nose; blood from the bowels; pallor of the membranes of the eye; great depression, coma, and death in from twelve to seventy-two hours after the onset of symptoms.

The **Lesions** include: Congestion of the pulmonary membranes and small hæmorrhages in the substance; congestion of the stomach and intestine, the walls of the latter being in certain parts deep red and thickened by infiltration of blood; blood may also be present in the lumen of the intestine; areas of diphtheritic inflammation and distinct ulceration may be present in the stomach and intestines; the serous membranes show hæmorrhages in their substance; and there are hæmorrhages in the heart and body muscle, and under the skin.

* *Jl. Comp. Path.*, 1894, p. 165. † *Ibid.*, 1899, p. 254.

Evidence is brought forward by the experiments conducted to show that the disease is not bacterial, although attention is drawn to the high temperature as incompatible with poisoning.

Feeding tests made with bracken as fresh as possible in 1909 and 1910 gave negative results. Thus a heifer took in all 60 pounds of bracken within a week. It was not eaten readily, and was therefore mixed with cut grass and sharps. After the first two meals, containing about 30 pounds of bracken, there was indigestion, but no toxic symptoms, and a normal temperature.

It came within the cognisance of the officers of the Board that the weed tormentil (*Potentilla tormentilla*) occurred along with bracken in many localities where bracken disease was reported. This plant is not commonly credited with being poisonous. In feeding tests made with tormentil a case of disease in a heifer having symptoms and lesions similar to those of bracken was produced.

In the present stage of the inquiry the chief officer refrains from designating tormentil as poisonous and as the cause of this disease, and points out that the possibility of its being a contagion carried by this weed is not excluded.

Moreover, it must be pointed out that S. B. Nelson,* of Washington, in 1898 fed 4 pounds of Potentilla to a sheep, which ate it within a day with no ill-effects.

* Fifteenth Annual Report of the Bureau of Animal Industry, 1898, p. 425.

CHEMICAL TOXICOLOGY

Introductory.—Under the respective headings in the text the chief tests for the poisons dealt with have been given. At first sight it may appear that few reactions have been quoted. Thus in the case of arsenic many well-known precipitation tests have been omitted. This has been done because it seemed advisable only to name those tests which are suitable for the recognition of traces, which are as characteristic as possible, and which can be applied to the substance in the form in which it is obtained from the material under research.

In the present section some details are given as to the general methods of separation of poisons from organic matter in the laboratory. For analytical purposes the poisons fall into four groups, viz.:

A. Volatile poisons.
B. Heavy metals and metalloids.
C. Non-volatile organic poisons.
D. Bases, acids, and alkali salts.

Preliminary Observations.—Before starting a systematic search for poisons, certain general observations ought to be made. These include *colour*. The existence of coloration, either local or diffused, is a valuable guide. A yellow colour suggests nitric and picric acids; greenish-blue points to copper; green to chromium compounds; black to iron compounds of tannin; blue may be due to indigo or Prussian blue, used as colouring agents for vermin powders, but the quantity is usually too small to be perceived; specks of red

may be vermilion or red lead; heavy black particles may be antimony sulphide. It is only rarely that colour is detected, for in most cases the dilution of a coloured poison in such a mass of green ingesta as is found in the stomach of the ox is too great for its recognition. With smaller animals the indication is more valuable.

Smell.—Compounds, easily recognisable by their smell, are phosphorus, hydrocyanic acid, alcohol, ether, chloroform, carbolic acid, savin, turpentine, essential oils, ammonia, and sulphuretted hydrogen. The two last are often products of decomposition, and caution is needed on this account in forming an opinion. In general the smell is difficult to observe on account both of the natural and putrefactive smell of the viscera.

The odorous substances fall into Group A of the volatile poisons, and their smells are best observed in the course of analysis for that group.

Suspicious particles and *vegetable fragments* ought to be carefully looked for and picked out. Valuable guidance is thus given, and in the case of many, or most, poisonous plants the finding and identification of vegetable detritus gives the best chance of correct diagnosis.

It is a good plan to stir up the semi-solid contents to a thin paste with water, and, after standing, carefully decant from heavy particles, which (if found) may be washed with a gentle stream of water. Particles of such substances as white arsenic, black antimony, vermilion, red lead, and white lead, may thus be separated.

In the case of dogs and foxes the nature of the stomach contents ought to be ascertained as far as possible. This gives very valuable information in tracing the source of poisoning. With these animals the presence in the stomach of scraps of fur, small bones, or feathers, usually points to a bait of rabbit or bird, which has been the vehicle of the poison.

Disposition of the material for the special analyses is a most important point, which is best left to discretion. The analyst must be guided entirely by considerations arising from the

quantity of available material, the nature of the poison suspected, and the degree of delicacy of its detection. It is always wise to reserve a portion, preferably about one quarter, for confirmatory analysis, or to replace accidental loss. When the analyst has at his disposal the whole stomachs of a horse, sheep, ox, or pig, he may consider that he has *carte blanche*, for the material suffices for more than one complete analysis. Quite otherwise when he has a small animal such as a bird, cat, or small dog to deal with. In such case he must use discretion in the taking of parts for the various operations.

Apparatus.—It is the common practice in medico-legal work on the human subject to advise perfectly new apparatus for each analysis. The apparatus used ought to be in good condition; thus porcelain dishes must have an unimpaired glaze; but the dogma of new vessels is somewhat extreme. To have used a set of apparatus in an analysis which has yielded negative results is the best proof of its cleanliness. It is an elementary maxim of the trained chemist to thoroughly clean all apparatus in such wise as to meet the object of the intended analysis. Thus a flask and condenser intended to be used in Reinsch's test will, as a matter of routine, be cleansed by boiling hydrochloric acid; a dish designed for the nitric acid solution of parts will be boiled out with that acid; flasks intended for the alcohol extraction of organic poisons will be cleaned with a mixture of strong sulphuric acid and potassium chromate after the laboratory attendant has 'cleaned' them. No chemist ever uses a piece of apparatus which he has not personally cleaned. Possibly if the Courts thoroughly appreciated the fact that it is a chemist's business to keep his apparatus clean, and not to get muddled, and mix the specimens or reagents, less weight would be attached to the general preliminary precautions. But, on the other hand, it is essential that the person who issues the report should himself either have performed, or personally supervised, all the operations.

Reagents.—All reagents designed for toxicological analysis must be of proved purity. The condition of reagents and

apparatus in this respect is best guaranteed by the performance of a blank test. If this gives a negative result—*e.g.* for arsenic—the reagents and apparatus are good. A blank ought to be carried through from time to time as a matter of routine precaution. Fortunately, to-day the manufacture of high grade chemicals has reached such a pitch of excellence that it is rarely necessary to specially purify one's reagents.

Qualitative Analysis.—This aims at the detection of the presence of a poison. It is the fundamental and really difficult task of toxicology. **Quantitative analysis** follows the qualitative, and its chief value is to guide the expert in the formation of an opinion as to the significance of the qualitative revelations. It has also a subsidiary value—that of providing a figure to be produced in evidence. The position of a witness who states that from 8 ounces of a material he separated $\frac{1}{40}$ grain of strychnine is stronger than that of the witness who states that he found a 'distinct trace' of that agent. No wise man would commit himself to the statement that the material contains a given weight of the poison; he will name the quantity he actually separated, and he will also be well advised to state that the weight given is his estimate, and be prepared to state how he determined it, and what is the probable error of his determination.

All quantitative data in toxicology are to be regarded as approximations. But even so they are indispensable and valuable approximations, for the reasons set forth above. The quantitative methods available for the estimation of traces of material are—

1. Direct weighing. This is the most satisfactory and sometimes the only possible method. It may be used for the measurement of quantities of lead and many other common metals. A residue of an alkaloid may also be weighed, but the weight ought not to be regarded as very exact, partly by reason of the normal error in weighing, but chiefly because the residue is rarely pure. Alkaloid extracts always contain traces of basic substance of animal

or vegetable origin (ptomaine bases) which when a small quantity of alkaloid is present (*e.g.*, 1 milligramme) may equal or exceed it in weight. Further purification leads to loss of material, which may be so great as to extend even to the vanishing point.

2. Volumetric methods depend on the performance of a reaction with a solution of a reagent of known strength, the end of the reaction being marked or indicated in various ways. As an illustration, hydrocyanic acid may be determined in the presence of sodium bicarbonate by adding measured quantities of a standard solution of iodine, which may be prepared of such strength that 1 cubic centimetre is equivalent to $\frac{1}{10}$ milligramme ($\frac{1}{2400}$ of a grain). If starch solution is also present the end of the reaction is marked by the production of the blue starch iodine coloration. From the number of cubic centimetres of iodine used the quantity of hydrocyanic acid may at once be calculated.

Free alkalis such as potash or ammonia, and free acids such as sulphuric, hydrochloric, or nitric, may also be determined by neutralisation with standard acid or alkali, respectively, in the presence of a suitable indicator, such as phenolphthaleïn, methyl orange, litmus, etc.

3. Colorimetric methods depend on the production of colour reactions. Having obtained a certain result, the colour may be matched by preparing similar tubes from measured quantities of the pure materials. In illustration: a certain tint of blue may be got by the Prussian blue test from an unknown quantity of a cyanide. This is matched by exactly similarly conducted preparations from known quantities of cyanide.

4. Allied to colorimetric methods are those dependent on the comparison of the relative intensity or bulk of a turbid precipitate, or a stain, such as an arsenical mirror in Marsh's test. Zinc is precipitated from ammonia solution by sulphuretted hydrogen, and when known amounts of zinc are tested similarly in the same bulk of fluid, it is easy to closely match the unknown by a known quantity. This method has received more study and

valuable application from German forensic chemists than it has received in this country. It is a very good method, capable of numerous applications—*e.g.*, to lead precipitated as iodide, to barium as sulphate or chromate, to silver as chloride or iodide, to tin as dioxide, etc.

In the determination of traces of arsenic the size and intensity of the mirror obtained in Marsh's test are compared with those given by known quantities of arsenic and formed under experimental conditions as nearly alike as possible.

GROUP A.

The separation of volatile poisons depends on the fact that they are more or less readily gasified and distilled along with steam from a boiling paste of the materials with water. In practice, distillation in steam is preferable to ordinary distillation, because the boiling is more regular, and the operation does not require so much attention. It is advisable to place the distilling flask in a boiling water bath, and to regulate the steam current, so that the quantity of boiling fluid remains about constant. In this way distillation may be allowed to proceed to any desired extent. For details of the process of distillation reference may be made to a textbook of practical chemistry.

The best method of procedure is to make a thin paste of the material, and ascertain its reaction (acid, alkaline, or neutral), if necessary acidify with either tartaric or dilute sulphuric acid, and distil in a current of steam. Volatile substances separated from an acid solution include—phosphorus, hydrocyanic acid, phenols, turpentine, savin, essential oils, alcohol, ether, chloroform, chloral, sulphuretted hydrogen, and sulphur dioxide.

The smell is characteristic (that of chloral in traces is faint), but, unless the substance is present in relatively large amount, it is masked more or less completely by the natural smell of the ingesta or organs. Distinct flakes (possibly of fatty acids) always pass over, and it is therefore

a good plan to allow the distillate to drop on to a small filter and collect the clear liquid in a flask or test-tube. Phenols (carbolic acid and creosote), turpentines (savin), essentials oils (camphor, etc.), and chloroform in fairly large proportions, form a distinct fluid turbidity or emulsion, and may actually separate into distinct oily drops, whilst camphor is solid. Phosphorus forms semi-solid globules, and the other substances named are soluble.

Phosphorus, creosotes, turpentine, and some essential oils, distil slowly, and a large bulk of liquid must be distilled in order to effect a complete separation. The other compounds—viz., hydrocyanic acid, alcohol, ether, chloroform, and the gases sulphuretted hydrogen and sulphur dioxide, being very volatile, concentrate in the first portions of distillate. If a large volume is collected, the dilution may be so great as to seriously interfere with the qualitative tests (especially with hydrocyanic acid), and it is therefore wise to stop the distillation after about 5 c.c. of clear distillate has been obtained. Small portions are then separately tested for the substances in question—*e.g.*, by the Prussian blue test for hydrocyanic acid, by the iodoform test for alcohol, by the neutral ferric chloride test for phenols (carbolic acid purple, creosote smoky colour), or bromine water for phenols (solid tribromphenol from carbolic acid, resinous bromcresols from creosote).

In order to effect a partial separation and purification of the volatile substances obtained at this stage the distillate is made alkaline with sodium hydroxide (not carbonate) and redistilled. Turpentines, essential oils, alcohol, ether, and chloroform (also given from chloral by alkali) distil over. If the alkaline liquid is now made acid with dilute sulphuric acid and again distilled, hydrocyanic acid, phenols, sulphuretted hydrogen, and sulphur dioxide pass over. In the course of these manipulations traces of phosphorus suffer oxidation, whilst with alkali on heating phosphuretted hydrogen and hypophosphorous acid are formed.

Having distilled from acid solution, the original residue in

the distillation flask is made alkaline with sodium hydroxide; or if none of the foregoing is present, a fresh portion of original substance is made into a paste with water and alkalised, and again steam distilled. In this case there distil over—volatile bases, ammonia, conine, nicotine, arecoline, and some bases of putrefaction—*e.g.*, trimethylamine (from brine), putrescine, and cadaverine (ptomaine). Chloral gives, with alkali, chloroform. These substances must be then detected and estimated by appropriate special methods.

The results above described may be summarised as follows:

Poisons distilled from dilute acid solution only	Hydrocyanic acid. Phenols. Sulphuretted hydrogen. Sulphur dioxide.
Poisons distilled from dilute caustic alkali solution only	Ammonia. Conine. Nicotine. Arecoline. Certain bases of putrefaction. Chloroform (from chloral).
Poisons distilled either from acid or alkaline solution	Phosphorus (not entirely without change). Turpentines. Essential oils. Alcohol. Ether. Chloral. Chloroform.

GROUP B.

Group B comprises heavy metals and metalloids—viz., arsenic, antimony, mercury, silver, lead, copper, zinc, chromium, and barium. Other metals which fall into the same category do not possess importance from the standpoint of veterinary toxicology, and are—tin, bismuth, cadmium, iron, manganese, gold, and platinum. Iron is always present, mainly from blood, and the others, if also present, offer no difficulties to the experienced worker.

It is unnecessary to make separate tests for each of these metals, although, having detected one or more of them, it may be needful to make special extractions of the original material for the purposes of estimation, and the obtaining of a specimen as a *corpus delicti*.

General schemes may be devised to cover a preliminary and comprehensive search, and several such schemes are available, each of which has its special claims to excellence, and its special advocates. The vital point is the destruction of organic matter, which is an extremely tedious process to carry out to completion. The English analysts rather appear to favour the complete destruction of organic matter either by ashing with previous addition of nitric and sulphuric acid, in which process all the organic matter is burnt away, or its destruction by prolonged heating with concentrated sulphuric acid in the presence of potassium sulphate. In the former process mercury is entirely lost, and in both there is danger of losing arsenic. Both are tedious, and, as our repeated experience has shown, do not possess advantage in point of accuracy.* The German experts rely on the process of Fresenius and Babo, which consists in the partial oxidation of albumins by warming with hydrochloric acid, and repeated small quantities of potassium chlorate (or chloric acid). The nascent chlorine is an effective oxidising agent, and the process yields a clear yellow solution, from which excess of chlorine must be driven by a current of air. From this acid liquid sulphuretted hydrogen, after prolonged standing, gives sulphides of arsenic, antimony (tin, cadmium, bismuth), mercury, silver, lead, and copper, along with sulphur, and organic matter. In practice this method is more tedious and no more accurate than the comparatively simple scheme outlined below, which has been adopted as the result of very extended practice and comparisons with other processes, but which merely systematises well-known analytical processes.

* See Lander and Winter, *Analyst*, 1908.

Two operations are performed:

(1) A Reinsch test direct on the material.
(2) Partial destruction of the organic matter with nitric acid alone, or along with sulphuric acid. This is the original method of Orfila, and is recommended by the German authorities in dealing with such materials as solid foodstuffs.

1. The material in suitable quantity, from ½ ounce to 6 or 8 ounces, according to circumstances, is boiled in a clean flask, with hydrochloric acid of about 16 per cent. strength, and pure copper foil, or gauze. Foil is better than gauze because the coating of a trace is more easily seen, on account of the smaller surface as compared with gauze. The flask is provided with an upright tube condenser, so that boiling may be prolonged without loss of water, or of arsenic by volatilisation as arsenious chloride. Before use the whole apparatus ought to be 'blanked' by boiling pure acid and copper together for half an hour, and showing that no deposit takes place on the metal. It is most important to remember that hydrochloric is the only suitable acid. In the event of free chlorine, potassium chlorate, nitric acid, or nitrates being present, solution of the copper will occur. In such case the acid mixture must be gently warmed until free of chlorine before putting in the copper.

In this test the following metals are deposited upon the copper: Arsenic, antimony (bismuth), silver, and mercury. If much of any of these is present, a deposit is seen on the copper in a few moments after heating, and unless the strip is taken out the whole of the copper will speedily pass into solution, and the deposit become disseminated throughout the mass and lost. Fresh strips are to be added and removed when well coated, until eventually no further deposition occurs.

If mere traces are present, boiling at a gentle heat ought to be continued for at least an hour before abandoning the test.

The delicacy of the test is extreme, especially for arsenic, of which $\frac{1}{1000}$ grain gives a distinct coating to a quarter square inch of copper, even when mixed with 4 ounces of organic matter. The test is less delicate with mercury, of which quantities much below the $\frac{1}{100}$ grain may escape deposition.

When the coating is at all considerable, that of arsenic has a steel-grey appearance, of antimony purplish-black, of bismuth black, and of mercury and silver the silvery lustre of those metals. When very small quantities are present, a definite stain of indistinct greyish-black tint may alone be given.

A good qualitative test and distinction is now got by thoroughly washing the coated copper with water and finally with pure alcohol, drying, and heating in a small perfectly dry ignition-tube. Arsenic forms a very characteristic sublimate of glistening octahedral crystals, easily observed under a low magnification. Antimony gives a white amorphous, or non-crystalline, sublimate. Mercury volatilises in globules of liquid metal. Bismuth is oxidised, but not volatilised, and silver remains unchanged by heat.

2. For the second process a suitable quantity of material, from 1 to 4 ounces, is mixed in a porcelain dish, with 50 per cent. nitric acid, to a thin paste, and about 1 to 2 c.c. of strong sulphuric acid added. The mixture is then warmed over a small flame with constant stirring. A violent reaction, accompanied by the evolution of brown oxides of nitrogen, sets in, and the operation must be conducted in a proper fume chamber. After from twenty minutes to half an hour's heating the liquid will be nearly all evaporated, and will have a brown colour and pasty consistency. No organic tissue remains, but fats are not completely destroyed. If heating is continued, the mass suddenly carbonises with some violence. There is a slight risk of loss if this occurs, but it is not a fatal objection. After carbonisation, which is unnecessary, the solutions are more deeply coloured, but the analysis is not hindered.

The heated mass is diluted with water, about 100 to

150 c.c., stirred well to disintegrate solid fatty matter, allowed to cool thoroughly, filtered, and the fatty residue washed with water. The pale yellow liquid now contains all the poisonous metals, except barium, whose sulphate is insoluble in dilute nitric acid. If barium is to be looked for, the sulphuric acid must be omitted, in which case the metal is in solution along with the others.

The acid solution is now made strongly alkaline with ammonia, whereby the colour changes to dark brown, and pure sulphuretted hydrogen bubbled into the warm liquid. A little yellow ammonium sulphide is added, and the liquid is filtered. The precipitate may contain sulphur, sulphides of lead, mercury, silver (bismuth), copper, zinc, manganese, and iron; phosphates of calcium and magnesium, and phosphate or hydroxide of chromium. Arsenic and antimony sulphides are soluble in ammonium sulphide, as also is organic matter. After washing the precipitate, it is therefore free from organic impurities, and may be dealt with according to the ordinary methods of qualitative analysis.

Iron in the form of black ferrous sulphide, sulphur, and phosphates (chiefly of calcium), are always present, and the other compounds named above may also be in the precipitate.

By passing hot dilute hydrochloric acid repeatedly through the filter, there are dissolved iron, phosphates, chromium, zinc, manganese, and lead. When, however, lead sulphide is present in considerable quantity, the sparingly soluble lead chloride forms needle-shaped colourless crystals on the paper and in the filtrate. Dilute hydrochloric acid also dissolves traces of copper and bismuth.

The dilute hydrochloric acid solution is concentrated until nearly all the free acid has been volatilised, the liquid diluted, and sulphuretted hydrogen solution added. Lead is precipitated as black sulphide, but with small quantities of the order of $\frac{1}{300}$ grain a yellow to brown coloration only is produced. In order to further characterise it, and to distinguish from copper and bismuth, the precipitate is

collected on a small filter, washed, dissolved in the minimum amount of dilute nitric acid, most of the acid evaporated off, and a small crystal of potassium iodide added. This gives with lead yellow lead iodide as an amorphous powder, dissolving on boiling to a colourless solution, which, on cooling, deposits yellow spangles of lead iodide, which are so characteristic that their formation is the best test for lead.

Having removed lead as the sulphide, the liquid is evaporated till free of sulphuretted hydrogen, oxidised by heating with a few drops of strong nitric acid, and ammonium hydrate added in excess. The precipitate always formed contains brown ferric hydroxide and phosphates. If the phosphate is in excess, the precipitate is pale yellow, and a few drops of ferric chloride must be added before the ammonia. In addition to these the precipitate may contain chromium hydroxide, known by its green colour. The filtrate is tested for zinc and manganese by adding ammonium sulphide, which gives colourless zinc sulphide and buff-coloured manganese sulphide as precipitates.

The residue left after treatment with hydrochloric acid may contain free sulphur, and the black sulphides of mercury, bismuth, copper, and silver. It is to be noted that silver, mercury, and bismuth will have been found in the Reinsch test.

Hot dilute nitric acid dissolves the silver, bismuth, and copper. If copper is present in significant amount, the solution will be of a pale, greenish-blue colour. Silver is recognised by adding dilute hydrochloric acid, which precipitates colourless silver chloride. Ammonium hydrate is now added, and precipitates bismuth hydroxide as a colourless flocculent precipitate. The solution is filtered, and the bismuth precipitate washed and dissolved in the minimum quantity of hydrochloric acid. On adding the chloride solution to water, an opalescent turbidity of bismuth oxychloride is produced. The ammoniacal filtrate containing the copper will have a more or less deep, pure blue colour, according to the quantity of copper. It is made acid with acetic acid and potassium ferrocyanide

added. This gives a reddish-brown coloration with traces of copper, and a reddish-brown precipitate with larger amounts, and is a more delicate test than the blue ammonia coloration.

The nitric acid leaves undissolved mercury. This is then dissolved in hydrochloric acid with the addition of a little potassium chlorate, and after boiling off the chlorine is tested by Reinsch's test with copper, or by adding stannous chloride solution, which precipitates insoluble mercurous chloride (calomel), and on warming converts this into mercury as a black deposit of finely divided metal.

The table (p. 289) summarises the operations set forth above.

The delicacy of the method is amply sufficient for toxicological purposes, and even for the recognition of traces which can hardly have medico-legal interest. Thus $\frac{1}{6000}$ grain of lead can be separated and recognised by sulphuretted hydrogen when originally contained in 2 ounces of organic matter.* With this amount of organic matter a positive reaction for lead may be anticipated with certainty. For mercury a proportion of $\frac{1}{3000}$ grain in 2 ounces can sometimes be recognised, at others not—that is, the figure quoted is an extreme lower limit. The limit for copper is $\frac{1}{1000}$ grain, and that for zinc $\frac{1}{60}$ grain, each in 2 ounces. The same figures apply to the detection of these metals when all the organic matter is burnt off by heating with strong sulphuric acid.

GROUP C.

This group comprises non-volatile organic poisons, of which the most important are the alkaloids and glucosides derived from poisonous plants. Of less importance are non-volatile phenols, such as tannin; organic acids, such as picric and oxalic; bitter principles, such as santonin and cantharidin; and synthetical drugs, such as antipyrin, sulphonal, and veronal, which do not play an important part in the toxicology of animals.

* See Lander and Winter, *loc. cit.*

ORIGINAL PRECIPITATE EXTRACTED WITH HOT DILUTE HYDROCHLORIC ACID.

<table>
<tr><td colspan="4">Solution (lead, zinc, manganese, chromium, iron, phosphates, and traces of copper and bismuth): concentrate, add sulphuretted hydrogen.</td><td colspan="4">Residue* (mercury, silver, bismuth, copper): boil with dilute nitric acid.</td></tr>
<tr><td rowspan="3">Precipitate: Lead and traces of bismuth and copper.</td><td colspan="3">Solution: boil off sulphuretted hydrogen, oxidise by nitric acid, add ammonia.</td><td rowspan="3">Residue: Mercury.</td><td colspan="3">Solution (silver, bismuth, copper): add hydrochloric acid.</td></tr>
<tr><td rowspan="2">Precipitate: Iron, Phosphates, Chromium.</td><td colspan="2">Solution (zinc and manganese): add ammonia sulphide.</td><td rowspan="2">Precipitate: Silver.</td><td colspan="2">Solution: add ammonia.</td></tr>
<tr><td>Precipitate: Zinc, Manganese.</td><td>Solution: Rejected.</td><td>Precipitate: Bismuth.</td><td>Solution: Copper.</td></tr>
</table>

* If cobalt and nickel are present, nickel would pass through with the original ammonium sulphide solution, and be lost; cobalt would be present as black sulphide in this residue.

The extraction of the poisons of this group depends upon the fact that they are all soluble in an excess of alcohol, to which a dilute acid (sulphuric or tartaric) is added in quantity sufficient to impart a distinct acidity. Acidification is necessary, because some alkaloids are very sparingly soluble in alcohol, but give salts which are more easily dissolved—*e.g.*, morphine. Since the organic acid salts of the alkaloids are more soluble in alcohol than the mineral acid salts, the use of tartaric acid is on the whole preferable to that of sulphuric. When the material is acid, no further addition is necessary.

A convenient amount of the material in as fine a state of division as possible is mixed with a large amount of alcohol. If 8 ounces of material is to be extracted, one uses about 400 c.c. of alcohol. The reaction to litmus is observed, and if not acid, sufficient tartaric acid is added to secure a distinctly acid reaction. In almost all cases the alcoholic mixture may be warmed nearly to boiling on a water-bath. Physostigmine, apomorphine, and taxine solutions should not be warmed, and in that case the mass should be allowed to stand twenty-four hours.

When cold, the material is filtered. The alcoholic filtrate contains any of the above specified poisons, and large quantities of such organic substances as fats, oils, carbohydrates, the simpler proteins, and bases derived from the decomposition of animal albumins. The residue may contain such toxines as ricine, and is rejected, since separate special search for these needs to be made.

The clear yellow to dark brown filtrate is now concentrated. This is best done by boiling in a vacuum, whereby the alcohol may be rapidly removed at a temperature of 30° to 40° C. A very thick, dark-coloured watery residue, amounting to about 10 to 20 c.c., and generally containing fat, remains.

The gradual addition of alcohol to this residue usually brings about a precipitation of impurities, such as dextrine and carbohydrates. This is removed by filtration, and the clear alcoholic solution again evaporated in a

vacuum to a small bulk. Water is added (20 to 50 c.c.) to the residue, and the mixture thoroughly shaken and filtered, the clear aqueous solution being collected in a separating funnel. The insoluble part is generally chiefly composed of fat, and ordinarily need not be further examined, although it may contain such drastic oils as croton.

The result of the processes is the obtaining of a watery extract of acid reaction, in which is concentrated the organic poison, and which has been moderately freed from organic products natural to flesh, tissues, and ingesta. It is always coloured, varying from pale yellow to dark brown.

Preliminary Observations on the Water Extract.—A tendency to foam and generally to simulate soap solution points to saponins.

Ferric chloride gives a red colour with meconic acid (from opium) in the acid solution. In a neutral solution ferric chloride gives violet colours with phenol, morphine, and salicylic acid, and a blue-black or black with tannins and gallic acid.

Extraction of the Constituents of the Water Extract.—The separation of the substances likely to be present in the water extract is effected through extraction of the water solution by means of organic solvents, which do not mix with or are only partially soluble in water, such as petroleum ether, ether, benzene, chloroform, ethyl acetate, and amyl alcohol. The nature of the substance extracted depends on the reaction of the water solution. If the solution is *acid*, solvents extract free acids, glucosides, and bitter principles; if it is *alkaline*, solvents extract alkaloids; if *neutral*, glucosides. Morphine is not extracted in the presence of strong alkali (sodium hydroxide), since being a phenol it forms salts of the alkali metals. It is, however, extracted from ammonia or sodium bicarbonate solution, since these are not strong enough alkalis to form salts with morphine. In practice it is sufficient to make alkaline with ammonia or bicarbonate as a usual procedure.

On the basis of these facts, an extraction of the acid liquid will yield glucosides, bitter principles, acid, and neutral compounds, and a subsequent extraction of the liquid made alkaline by ammonia will yield alkaloids. The two groups are thus partially separated, but many alkaloids are extracted from acid solution (being feeble bases)—*e.g.*, chelidonine, veratrine, colchicine, and solanidine.

The solvent to be used depends on the nature of the dissolved substance which it is desired to extract. Of those named petroleum ether has the most limited range, and amyl alcohol the widest. Attempts have been made to separate the possible components by systematic extraction. Thus Dragendorff developed a complete scheme dependent on—

(*a*) Successive extraction of the acid liquid with petroleum ether, benzene, chloroform, and amyl alcohol.

(*b*) Extraction by the same solvents in the same order of the liquid made alkaline by sodium hydroxide.

(*c*) Extraction by amyl alcohol after replacing sodium hydroxide by ammonia (by saturating with ammonium chloride) for morphine.

(*d*) The residual solution contains non-extractible compounds—curarine.

This scheme has little or no value in practice, partly because it rarely happens that more than one or two poisons are present, but chiefly because the separation is not sharp. Many compounds pass into each of the solvents in variable proportion, and are thus spread out over several fractions.

In actual experience, successive extractions with ether and chloroform (*a*) from acid and (*b*) from ammoniacal solution are sufficient. Instead of chloroform, it is very convenient to use ethyl acetate, which possesses nearly as wide a range of solvent power, which separates more easily from emulsion than chloroform, and which forms a layer lighter —and not like chloroform, heavier—than water.

The various extracts are evaporated either spontaneously or at a gentle heat in porcelain dishes, or on a porcelain

tile having a number of cup-shaped depressions. Before evaporation it is advisable to run the extract through a small dry filter-paper, in order to remove traces of watery solution.

The problem of detecting the organic poison now confronts the analyst, and is beset with difficulty, for many reasons, of which may be stated as prominent—

(*a*) The invariable presence of traces of impurity derived from the original organic material, such as fats, carbohydrates, and traces of protein resolution products of basic character. Whether a poison is present or not, there is always left after evaporation a more or less considerable smear of yellowish to brown colour, which reacts with such general alkaloidal reagents as phosphomolybdic acid, and has reducing properties. If, however, an organic poison is present in fair quantity—as, for instance, about $\frac{1}{30}$ or upwards of a grain, the difficulty on account of such normal impurity is diminished.

(*b*) In many cases characteristic or distinctive reactions are lacking. This holds particularly with bitter substances and glucosides.

Purification.—It is theoretically easy to devise methods for the purification of any of the special types of organic poison—in fact, it is hard to carry out, because the process is wasteful, and therefore in dealing with a trace there is risk of entirely losing it. This event befalls very often. A residue may show a certain reaction in an incomplete or masked fashion, leading to a strong suspicion of the presence of a particular poison. After appropriate purification the test may fail. This does not mean of necessity that the substance was absent, but, on the other hand, may point to it having been present in small quantities of such order as not to point to poisoning. In this sense such an observation is valuable.

Alkaloids which are strong bases may be purified by dissolving the residue in a dilute acid (acetic or sulphuric), and shaking the solution with solvents, which more or less fully remove adventitious impurities of a neutral, acid, or feebly alkaline nature. Thereafter the acid liquid is suitably

alkalised, and the organic base again extracted, now in a purer form.

Acids are purified by solution in an alkali (soda or ammonia), extraction of the alkaline solution with solvents, acidification and re-extraction of the acidic component.

Solution in water, alkali, or acid and re-extraction, often effect a partial purification of neutral substances.

Alkaloids may often be precipitated from solution by means of an alkaloid reagent, such as phosphomolybdic acid, and the precipitate filtered and washed. It is then decomposed—*e.g.*, with soda or baryta, and the alkaloid extracted with solvents.

Basic lead acetate precipitates proteins and many other impurities from organic solutions. After filtration, the excess of lead is removed by a current of sulphuretted hydrogen, and the organic substances are separated by solvents, or by evaporation, from the filtrate.

General Reactions of the Organic Poisons.—There are certain reactions by means of which it may be decided whether a substance is or is not an alkaloid or a glucoside.

Alkaloids.—There are numerous substances which give precipitates with alkaloids—*e.g.*, phosphomolybdic acid, bismuth potassium iodide, iodine in potassium iodide, tannin, the chlorides of platinum and gold, etc. The formation of a precipitate depends on the nature of the alkaloid, on the acid in which it is dissolved, on the concentration, and on the relative amounts of substance and reagent. As is to be expected, there is a wide range in point of delicacy. Phosphomolybdic acid, bismuth potassium iodide, and tannin are most to be recommended. To apply these tests a solution of a trace of the material should be prepared in a drop of dilute sulphuric acid (1 in 50), and placed on a glass on a black under-surface. A drop of the precipitant is then brought into contact with it on a glass rod, and the formation of an amorphous precipitate is looked for. The precipitates given by phosphomolybdic acid are pale to distinct yellow; by bismuth potassium iodide, brown to red; and by tannin, dirty white.

It is the habit of many writers on toxicology to carefully tabulate and compare the kind of such precipitates given by the alkaloids. This is entirely useless for the detection of traces recovered from organs, although possibly valuable when detecting a pure substance. Since the residues got in the laboratory are always, even when once purified, contaminated with basic animal products, they always give a positive reaction with all or some of the general reagents, and the use of these agents therefore misleads, wastes material, and is seldom resorted to.

Glucosides.—These compounds are all characterised by yielding sugars (generally glucose) on contact with mineral acids, and thereon is based the Brunner-Pettenkofer test, which is a modification of Pettenkofer's well-known bile test. A solution of the suspected substance is made in water along with a little purified ox-gall and placed in a test-tube. Strong sulphuric acid is poured down the side of the tube, forming a heavy layer below the water-bile solution. At the point of juncture there is developed a cherry-red colour zone, which gradually extends throughout the aqueous layer. Its formation depends on the splitting off of sugar from the glucosides, thus giving with sulphuric acid the bile reagent of Pettenkofer's test.

Unfortunately, residues often contain traces of carbohydrates, and thus the test fails, unless the glucoside has been obtained in sufficient quantity to permit of purification.

Sulphuric Acid as a Reagent for Alkaloids and Glucosides is of some value, although the indications are apt to be misleading when the material is not pure. A test performed either with pure sulphuric acid, or with sulphuric acid containing a small proportion of an oxidising agent, such as nitric acid (Erdmann's reagent), molybdic acid (Fröhde's reagent), vanadic acid (Mandelin's reagent), can only be taken as a guide, and must be confirmed by special characteristic chemical tests, or, failing these, by a physiological experiment.

The appended table sets forth the colorations given by some of the important alkaloids and glucosides, the order

of naming of the colours being the order in which they are developed.

REACTIONS OF CERTAIN ALKALOIDS AND GLUCOSIDES.

	Sulphuric Acid.	Erdmann's.	Fröhde's.
Aconitine	Yellow	Yellow	Yellow
Brucine	Colourless	Blood-red, passing to yellow	As Erdmann's
Bryonin	Orange-yellow, violet on warming	—	—
Chelidonine	Greenish-yellow, brown, cherry-red, violet	Green	Yellow-green, blue-green
Colchicine	Yellow	Violet, yellow	Violet, yellow
Cytisine	Colourless	Orange-yellow, yellow-brown	Colourless
Delphinine	Dark brown, deep red-brown	Red-brown	Brown, blood-red, cherry-red
Digitalin	Yellow, blood-red	—	—
Digitalein	Red	—	—
Digitoxin	Green, black-brown	—	—
Digitonin	Brown-red	—	—
Helleborein	Red	—	—
Helleborin	Violet	—	—
Lobeline	Yellow, red	Yellow-red	Violet, brown, yellow (with impure substance)
Morphine	Colourless, faint red	Faint red, darker on warming	Violet, blue, green, yellow, rose-red
Physostigmine	Colourless	Faint reddish-yellow	As Erdmann's
Solanine	Orange; on heating violet, red brown	As with sulphuric	As with sulphuric
Strychnine	Colourless	Colourless	Colourless
Taxine	Red	Yellow	—
Veratrine	Yellow, orange, red, carmine	As with sulphuric	As with sulphuric

GROUP D.

It is not possible to give a single comprehensive scheme for the detection of the various acids, alkalis, and soluble salts comprised under this heading. An excellent method of separation for soluble compounds of this class is afforded by dialysis, taking advantage of the fact that the soluble crystalloids pass through a membrane into surrounding pure water or dilute solution, whilst the colloid protein substances do not so diffuse, or if so, only extremely slowly. For the practice of dialysis the most convenient membrane is a diffusion shell or thimble of parchment paper. Failing this, an intact sausage-skin membrane may be used. The material under examination is made into a thin paste with water, and placed in the shell, which is then immersed in a narrow cylinder of distilled water. The dialysate is removed from time to time, fresh water being added.

Acids, bases and soluble salts are thus obtained in a fairly pure watery solution. Sometimes, however, it is sufficient to considerably dilute the material and filter; but the filtration is often very slow, and dissolved proteins are not removed.

The subsequent handling of the solution depends on the nature of the substances under research, and as a rule no difficulty exists in separating and identifying by means of evaporation to crystallising point, neutralisation, precipitation, and the like operations.

As regards *alkalis*, these are recognised by means of indicators, which are dye-stuffs having different colours according as to whether the solution is acid (contains hydrogen ions), or alkaline (containing OH ions). In this group may be found the strong alkalis—potash, soda, lime, strontia, and baryta—and weak alkalis, such as ammonia, ammonium carbonate, carbonates and bicarbonates of sodium and potassium, and borates and silicates of the alkalis. It ought to be carefully remembered that the rumen and intestinal contents are normally alkaline. Allowance must be

made for this, and a quantitative determination of alkali is imperative in order to establish the existence of an excess. The alkalis caustic soda and potash are soluble in alcohol, and may be thus separated and distinguished from the carbonates. A further discrimination is afforded by their different behaviour on neutralisation by acids with particular indicators. When phenolphthaleïn is used as the indicator, the neutral point reached on adding acid gives the whole of the alkali. Carbonates, on the other hand, show neutrality at the half point, and when a second indicator (methyl orange) is added, neutralisation may be pursued to completion.

As regards *acids*, it must be remembered that the digestive stomach is normally acid through the presence of free hydrochloric acid, and it must also be remembered that organic acids (*e.g.*, lactic and butyric) may be present. In order to distinguish mineral (strong) acids from organic (weak) acids, several methods may be adopted :

(*a*) A dilute solution of methyl violet is made by adding water to a few drops of the 1 per cent. alcoholic solution, until a clear violet colour is given. When a solution of mineral acid, such as nitric, hydrochloric, or sulphuric, is added to this, the colour passes through blue and green to yellow. Amongst organic acids, oxalic gives a blue, but not yellow, colour.

(*b*) Neutral ferric acetate does not give a red colour with potassium or ammonium sulphocyanide, and a dilute solution scarcely shows the faint red colour of the acetate. When a mineral acid is added, the non-ionised ferric acetate yields the ionised ferric salt of the mineral acid, which gives the blood-red coloration with the sulphocyanide.

(*c*) On similar grounds neutral ferric acetate, starch solution, and potassium iodide show no blue coloration. But the addition of mineral acid produces the ferric ion, which liberates iodine from the potassium iodide, and thus produces the blue coloration with the starch.

PTOMAINES.

Ptomaines (cadaveric or corpse alkaloids) may be defined as organic bases produced during the decomposition of animal substance, and the decomposition is chiefly due to bacterial action. Besides the ptomaines of putrefaction, definite animal bases are contained in normal and in pathological urine. Normal dog's urine, for example, contains, according to Kutscher, a poisonous base, cynosine.

By no means are ptomaines all poisonous, and the poisonous character depends on the duration and kind of the bacterial fermentation. Poisonous bases are formed in the earlier stages of decay. After advanced decay they pass into simpler, or more stable and harmless, compounds. Anaërobic decomposition is favourable to the production of poisonous ptomaines, although the total quantity of bases formed is smaller. Aërobic fermentation produces a larger quantity of bases, but of less poisonous nature.

The possible presence of ptomaines very naturally complicates the task of alkaloid detection. Corpse-conine, corpse-strychnine, corpse-delphinine, and other ptomaines resembling the corresponding vegetable alkaloids, have been obtained by various observers. In no case do these bases entirely coincide in properties with the alkaloids. Some one or more of the tests of the vegetable compounds may not be given by the ptomaine, or it may differ in its physiological action. Sometimes ptomaine is separated in relatively large quantities. In an investigation of the organs of a dog the writer in 1904 obtained considerable quantities of an alkaloid very closely simulating morphine in external properties, and in respect to those tests which depend upon the reducing power of morphine. It did not, however, give the characteristic Pellagri test.

For these reasons it is generally possible to avoid confusion between a ptomaine and an alkaloid, but it may be necessary to this end that the separated alkaloid be thoroughly purified. In practice the quantity of available

material does not always permit of this being done. In this event the history of the case is of great value in diagnosis.

Occurrence of Ptomaines.—In many cases the ptomaines are relatively simple compounds, and their genesis from proteins or other cell components may be readily traced. Thus lecithin yields the base choline, which is harmless, but passes into neurine and muscarine, which are both very poisonous, betaine (harmless), and eventually trimethylamine, the base characteristic of herring brine.

Muscarine is also found in the fungus fly-blown agaric.

The two-base putresceine (tetramethylene diamine) and cadaverine (pentamethylene diamine) are both resolution products of arginin and lysine, which are themselves cleavage products of proteins. These two ptomaines are non-toxic, but closely allied to cadaverine is the very poisonous base sepsine, formed at an earlier stage of decay, and passing very readily indeed into cadaverine.

Ptomaine Poisoning.—Ptomaine poisoning is attributable to ptomaine bases contained in spoilt food, or to the further activity of the bacteria of decay in the organism. The latter view appears to be the more probable, although instances of poisoning have been observed, in which bacteriological research failed to disclose the responsible organism.

Flesh poisoning may be due to the specific toxine of the *Bacillus botulinus*, as in sausage poisoning, or to poisoning by the very dangerous base sepsine produced in the early stages of decay. Sepsine is likewise produced in the putrefaction of brewer's yeast. It appears not unlikely that many of the cases of illness and death from time to time recorded, and ascribed to such foods as brewer's grains, distillery sludge, and the like, are, in fact, of this nature. Similarly cases of pig poisoning by the brine from salting meat or herrings are possibly attributable to the same cause.

Cadaverine is the eventual result of sepsine poisoning, and it is fairly stable and easily recognised. A good

method of identification may be based on—(*a*) its separation by distillation from alkaline solution; (*b*) its conversion into the dibenzoyl compound (by means of alkali and benzoyl chloride). The dibenzoyl compound when pure melts at 130° C. Its detection would thus probably form a valuable guide as to the nature of these poisonings.

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